DEVELOPER'S ASSESSMENT REPORT

April 2012

NICO COBALT-GOLD-BISMUTH-COPPER PROJECT

Human Health Risk Assessment

Submitted to:
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1.0 INTRODUCTION

1.1 Context

Fortune Minerals Limited (Fortune) proposes to develop a new underground and open pit cobalt, gold, copper, and bismuth mine and processing plant; hereinafter referred to as the NICO Cobalt-Gold-Copper-Bismuth Project (NICO Project). This report provides a detailed description of the Human Health Risk Assessment (HHRA) undertaken for the NICO Project. The HHRA provides an assessment of the potential health effects to humans that may occur as a result of changes to the environment due to predicted discharges from the NICO Project.

1.2 Purpose and Scope

The purpose of the HHRA is to address the following:

- satisfy the requirements of the Terms of Reference (TOR) issued by the Mackenzie Valley Review Board (MVRB 2009); and
- address the concerns raised by the Tłı̨chǫ Government and other citizens regarding the potential impacts to human health related to the development of the NICO Project.

The MVRB approach to the TOR included the identification of Key Lines of Inquiry (KLOI), which were defined as the “areas of greatest concern that require the most attention during the environmental assessment and the most rigorous analysis and detail in the Developer’s Assessment Report (DAR)” (MVRB 2009). Additional detail regarding the MVRB approach to environmental assessment was provided in the DAR (Fortune 2011).

Of the KLOI identified in the TOR for the NICO Project, 2 are relevant to human health: Water Quality and Closure and Reclamation (Table 1.2-1). Both KLOI, when addressed, intend to protect human use of surface water as a source of drinking water and fish, and the indirect potential effects to wildlife that may be consumed by humans as wild game.

Of the concerns raised by the Tłı̨chǫ Government with respect to the NICO Project, the following 4 concerns are relevant for human health and were addressed in this HHRA:

- Hislop Lake and Marian River are important traditional and culturally significant areas, and as such, both locations were assessed as potential receptor locations;
- Potential cumulative effects due to the old Rayrock mine and Colomac mine were qualitatively evaluated;
- Concerns regarding the presence of the waste rock piles, the safety of the tailings dam and the presence of the Co-Disposal Facility (CDF) were evaluated in the HHRA; and
- Concerns regarding the potential for wildlife contamination and exposure by humans through consumption of wild game were addressed in the HHRA.

Based on the above objectives, the following management goals, assessment and measurement endpoints, and decision criteria for the HHRA were determined (Table 1.2-2).

Table 1.2-1: The Key Line of Inquiry, Management Goals, Assessment, and Measurement Endpoints, and Decision Criteria for the Human Health Risk Assessment

Key Line of Inquiry	Management Goal	Assessment Endpoint	Measurement Endpoint	Decision Criteria
Water Quality	• Protection of surface water quality for human use • Continued opportunity for traditional and non-traditional use of fish	• Local community structure and function	• Comparison of concentrations of chemicals in surface water to drinking water guidelines	• Concentrations that exceed drinking water guidelines must be mitigated
Closure and Reclamation	• Protection of surface water quality for human use • Continued opportunity for traditional and non-traditional use of plants, fish, and wildlife	• Local community structure and function	• Comparison of concentrations of chemicals in surface water to drinking water guidelines	• Concentrations that exceed drinking water guidelines must be mitigated

Table 1.2-2: The Tłı̨chǫ Concerns, Management Goals, Assessment, and Measurement Endpoints, and Decision Criteria for the Human Health Risk Assessment

Tłı̨chǫ Concerns	Management Goal	Assessment Endpoint	Measurement Endpoint	Decision Criteria
Hislop Lake and Marian River	• Protection of air quality and surface water quality for human use and continued traditional and non-traditional use of the land	• Local community structure and function	• Comparison of concentrations of chemicals in surface water to drinking water guidelines • Comparison of concentrations of chemicals in ambient air to ambient air thresholds • Comparison of concentrations of chemicals in soil to soil guidelines	• Concentrations that exceed drinking water guidelines must be mitigated • Concentrations that exceed ambient air thresholds must be mitigated • Concentrations that exceed soil guidelines must be mitigated
Cumulative effects due to Colomac and Rayrock	• Protection of air quality and surface water quality for human use and continued traditional and non-traditional use of the land	• Local community structure and function	• Comparison of concentrations of chemicals in surface water to drinking water guidelines • Comparison of concentrations of chemicals in ambient air to ambient air thresholds • Comparison of concentrations of chemicals in soil to soil guidelines	• Concentrations that exceed drinking water guidelines must be mitigated • Concentrations that exceed ambient air thresholds must be mitigated • Concentrations that exceed soil guidelines must be mitigated
Presence of waste rock piles and tailings	• Protection of air quality and surface water quality for human use	• Local community structure and function	• Comparison of concentrations of chemicals in surface water to drinking water guidelines • Comparison of concentrations of chemicals in ambient air to ambient air thresholds • Comparison of concentrations of chemicals in soil to soil guidelines	• Concentrations that exceed drinking water guidelines must be mitigated • Concentrations that exceed ambient air thresholds must be mitigated • Concentrations that exceed soil guidelines must be mitigated
Potential for contamination of wildlife	• Protection of air quality and continued traditional and non-traditional use of the land	• Local community structure and function	• Comparison of concentrations of chemicals in ambient air to ambient air thresholds • Comparison of concentrations of chemicals in soil to soil guidelines	• Concentrations that exceed ambient air thresholds must be mitigated • Concentrations that exceed soil guidelines must be mitigated

The management goals identified for all KLOI and Tłı̨chǫ concerns are the protection of surface water quality for human use and continued traditional and non-traditional use of the land and its resources. Although the NICO Project water management plan includes meeting the site-specific water quality objectives (SSWQOs) at receiving waterbodies (Golder 2011), the SSWQOs were developed to be protective of aquatic life and not human health. Therefore, in addition to the SSWQOs, the HHRA has included the comparison of predicted surface water quality to drinking water quality guidelines.

To satisfy the requirements of the TOR and to address the concerns raised by the Tłı̨chǫ Government and other citizens, the HHRA focussed on the following:

- assessment of the potential health risks to humans due to emissions from the NICO Project, including those KLOI identified in the TOR as they pertain to human health; and
- addressing the concerns raised by the Tłı̨chǫ Government and other citizens as they pertain to human health. Specifically, Hislop Lake and the Marian River were assessed in the HHRA as potential receptor locations, the potential for cumulative effects due to neighbouring closed mines was assessed in the HHRA, the presence of the CDF was evaluated with respect to human health, and an assessment of the potential for chemicals, such as arsenic, to adversely affect human health was evaluated in the HHRA. Cyanidation was not included in the Project Description (Section 1.3) and as such was not considered further in the HHRA.

In mining projects, potential impacts can only occur where there is a direct link between project activity and the environment. Therefore, the HHRA focused on those aspects of the NICO Project that could result in Project-related discharges to the environment, thereby potentially impacting human health where there is a complete exposure pathway between a source and a receptor. To facilitate an understanding of the NICO Project activities that could result in potential impacts to human health, a brief description of the NICO Project, and the study areas used to analyze and assess effects to human health is provided in the next section.

1.3 Project Description

1.3.1 Project Location

The NICO Project is located approximately 160 kilometres (km) northwest of Yellowknife, Northwest Territories (NWT) within the Marian River drainage basin, approximately 10 km east of Hislop Lake at a latitude of 63°33’ North, and a longitude of 116°45’ West (Figure 1.2-1 of the DAR).

The NICO Project site has rugged topography. The site topography is illustrated in Figure 1.2-4 of the DAR. Absolute elevations at the NICO Project site range from 150 to 350 meters above sea level. The ore body is located on the northern slope of a bowl-shaped depression referred to as the “Bowl Zone”. The south end of the proposed mine is located on a ridge of exposed bedrock, which slopes down towards the north end of the proposed mine in the Grid Pond depression.

With the exception of Fortune’s leases, all of the land surrounding the mine is within the Tłı̨chǫ settlement lands owned and managed as fee-simple lands by the Tłı̨chǫ Dèts′ǫ Kàowo as per the Tłı̨chǫ Agreement (Figure 1.2-2 of the DAR). The Tłı̨chǫ lands are within the Wek′èezhìı co-management lands, jointly managed with the Northwest Territory and Federal Government. Fortune’s exploration leases were staked and brought to lease prior to settlement of the Tłı̨chǫ land claim and as Crown Land are administered by the Federal Government.

Subject to approvals, the plant site will be constructed approximately 500 metres (m) west of Nico Lake, between Nico and Lou lakes.

1.3.2 The Proposed NICO Project

The NICO Project includes development of an underground mine and open pit. The current proposed site development for the NICO Project is shown in Figure 1.2-3 of the DAR. Proposed on-site infrastructure includes the following:

- mine site with open pit and underground operations;
- tailings and mine rock management area (presented as a single CDF);
- mineral process plant (the Plant);
- Effluent Treatment Facility (ETF), with discharge into Peanut Lake through a diffuser;
- Sewage Treatment Plant (STP);
- drainage controls;
- camp;
- truck stop;
- fuel and chemical storage facilities;
- materials sorting facility;
- landfarm;
- explosives storage area;
- roads within the mine site and NICO Project Access Road with access to site via the proposed Tłı̨chǫ Road Route; and
- fresh water intake on Lou Lake and diffuser in Peanut Lake.

Primary processing of the ore will be conducted on-site in the Plant, including crushing, grinding, and floatation (consisting of primary and secondary stages) to produce bulk concentrate. The concentrate will then be shipped off-site for final processing. Cyanidation and a cyanide destruction circuit will not be incorporated into the final NICO Project design.

During operation of the mine, the NICO Project will generate mine rock and tailings. The mine rock includes soil and overburden from pre-stripping above the ore body and mine rock from development of the open pit. Processing of the ore will result in generation of tailings. Mine rock and tailings will be disposed of in the CDF. At closure, the CDF will be capped.

Several mine activities will generate excess water, including ore processing and pumping from the open pit and underground workings. All water that comes into contact with the mine facilities during construction, operations, and closure will be managed. During operations, the CDF will house the water management facilities, the major components of which will include the following:

- Reclaim Pond on the CDF will be relocated throughout the mine's operating life as the CDF develops;
- 5 Seepage Collection Ponds (SCPs) located downstream of the CDF;
- Surge pond near the Plant;
- Plant site runoff pond;
- STP;
- ETF; and
- related water management facilities, including drainage ditches, emergency spillways, pump stations, and the reclaim water pipeline system.

During operations, all water that has been in contact with ore or mine waste will be collected in one of the following: the SCPs, the open pit sump, or the Reclaim Pond. Collected water in these ponds/sump will be pumped to the Surge Pond. Water will then be pumped from the Surge Pond either to the Plant for reuse or to the ETF for treatment. Treated effluent from the ETF and STP will be pumped through a diffuser directly into Peanut Lake.

During closure, pumping water out of the open pit will cease and the open pit will slowly fill with water. The rate of filling will increase by directing CDF runoff (and seepage reporting to SCP No. 4) into the open pit by breaching the SCP No. 4 dam. The Project Description assumes that water that accumulates in SCP Nos. 1, 2, 3, and 5, as well as the Surge Pond will be passively treated in Wetland Treatment Systems and then released directly into Nico Lake. Overflow from the open pit will be passively treated in Wetland Treatment System No. 4 and released into Peanut Lake. This is subject to demonstrating the technical performance of the Wetland Treatment Systems.

Potential NICO Project activities that could result in emissions to the environment are listed below:

- emission of combustion chemicals to air from fuel sources such as mine equipment and vehicles;
- generation of road dust during transportation of concentrate to off-site processing facilities during operation;
- mining, crushing, and disposal of mine rock and tailings during operation;
- water discharges, including the following:
  - management and discharge of stormwater runoff;
  - discharge of water from the ETF and STP during operation;
  - seepage from the CDF during operation and post-closure; and
  - flooding of the open pit during post-closure.

1.4 Study Areas

This section contains a brief description of the study areas used to analyze and assess effects to human health with reference to sections and figures within the DAR (Fortune 2011).

1.4.1 General Setting

The NICO Project is located within the Marian River drainage basin, approximately 10 km east of Hislop Lake at a latitude of 63°33' North and a longitude of 116°45' West, and within the Taiga Shield and Taiga Plains Ecoregions (Ecosystem Classification Group 2007, 2008). The NICO Project spans 2 Level II Ecoregions: Taiga Shield and Taiga Plains.

The NICO Project site is located in the central part of the Tłı̨chǫ lands, NWT. The Tłı̨chǫ lands are described as part of the Tłı̨chǫ Land Claims and Self Government Agreement (the Agreement), negotiated by the Dogrib Treaty 11 Council, the Government of the Northwest Territories (GNWT), and the Government of Canada, and signed in August 2005 (http://www.ainc-inac.gc.ca/ai/mr/nr/j-a2005/2-02586-eng.asp). The current Tłı̨chǫ lands cover approximately 39 000 square kilometres, including the subsurface resources (http://www.ainc-inac.gc.ca/ai/mr/nr/j-a2005/2-02586-eng.asp).

There are 4 primary communities within the Tłı̨chǫ lands, including Behchokǫ̀, Whatì, Gamètì, and Wekweètì. The NICO Project is located approximately 80 km north of Behchokǫ̀, 50 km north of Whatì, and 70 km south of Gamètì. The fourth community, Wekweètì, is located the farthest from the NICO Project, approximately 145 km northeast. The NICO Project is within the traditional land use areas of the Tłı̨chǫ and the Métis.

1.4.2 Regional and Local Study Areas

A conventional terminology was used: regional study area (RSA) and local study area (LSA). The RSA is selected to capture the larger scale direct and indirect effects from the NICO Project on human health (i.e., contains the maximum zone of influence from the NICO Project). The LSA represents the area that may be directly affected by the mine footprint, and that may potentially experience small-scale indirect effects from activities associated with the NICO Project.

These study areas differ depending on the NICO Project disciplines. The study areas for the HHRA were aligned with the study areas identified by the NICO Project disciplines that will predict potential NICO Project-related changes to environmental quality (i.e., air quality, water quality, soil and vegetation chemistry), or that provided other information relevant to human receptors. The reader is referred to the relevant sections of the DAR for detailed descriptions of the study areas for the air quality assessment (Section 10.0, Figure 10.1-2 of the DAR), water quality assessment (Section 7.0, Figure 7.1-1 of the DAR), terrain and soils assessment (Section 13.0, Figure 13.1-2 of the DAR), vegetation assessment (Section 14.0, Figure 14.1-2 of the DAR), human environment (Section 16.0, Figure 16.1-2 of the DAR), and traditional knowledge/traditional land use (Section 5.0, Figure 5.1-2 of the DAR).

1.5 Content

This report is generally organized as follows:

- Section 2.0: Risk Assessment Framework and General Approach describes each component of the risk assessment (RA) framework (problem formulation, exposure assessment, toxicity assessment, risk characterization) and the general approach used in the HHRA.
- Section 3.0: Data Used in the Human Health Risk Assessment summarizes the data used in support of the HHRA.
- Section 4.0: Human Health Risk Assessment provides the assessment of the potential effects to human

health that may occur as a result of the changes to the environment due to predicted emissions from the NICO Project.

- Section 5.0: Summary of Human Health Results and Conclusions provides the overall assessment of NICO Project-related effects on human health (including an assessment of the cumulative effects due to foreseeable projects, developments, activities, and natural factors that influence the environment).
- Section 6.0: References provides the sources of information relied upon in the HHRA.

2.0 RISK ASSESSMENT FRAMEWORK AND GENERAL APPROACH

2.1 Risk Assessment Framework

Risk assessment is a scientific tool used to characterize the nature and magnitude of potential risks, if any, associated with the exposure of receptors (e.g., humans) to chemicals. For there to be a potential risk, the following 3 conditions must be met:

- a chemical must be present at levels that could be harmful;
- a receptor must be present; and
- there must be an exposure pathway by which the receptor can come into contact with the chemical.

These three conditions are illustrated in Figure 2.1-1, where risk is anticipated to occur when the three necessary conditions are met.

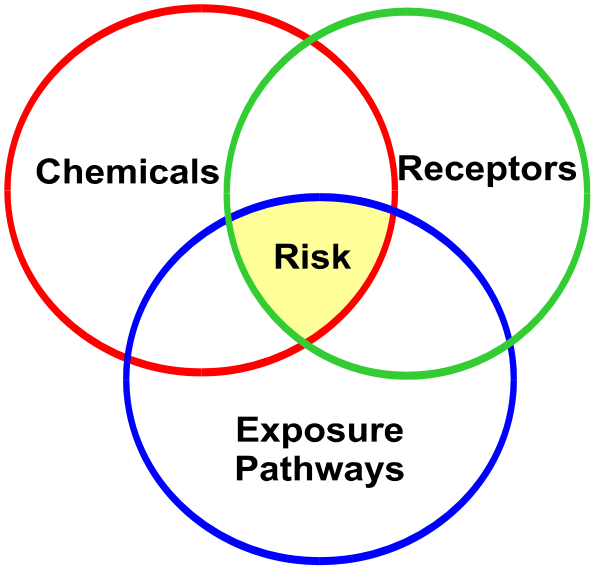


Figure 2.1-1: Venn Diagram showing the Three Conditions that must Exist for there to be a Potential Health Risk (modified from CCME 1996).

To determine whether these conditions are present, the RA framework used in Canada typically involves four components, as described below and depicted in Figure 2.1-2:

i) Problem Formulation: The Problem Formulation involves developing a focused understanding of how environmental quality might affect the health of receptors (i.e., humans) near the proposed project. The problem formulation identifies the following:

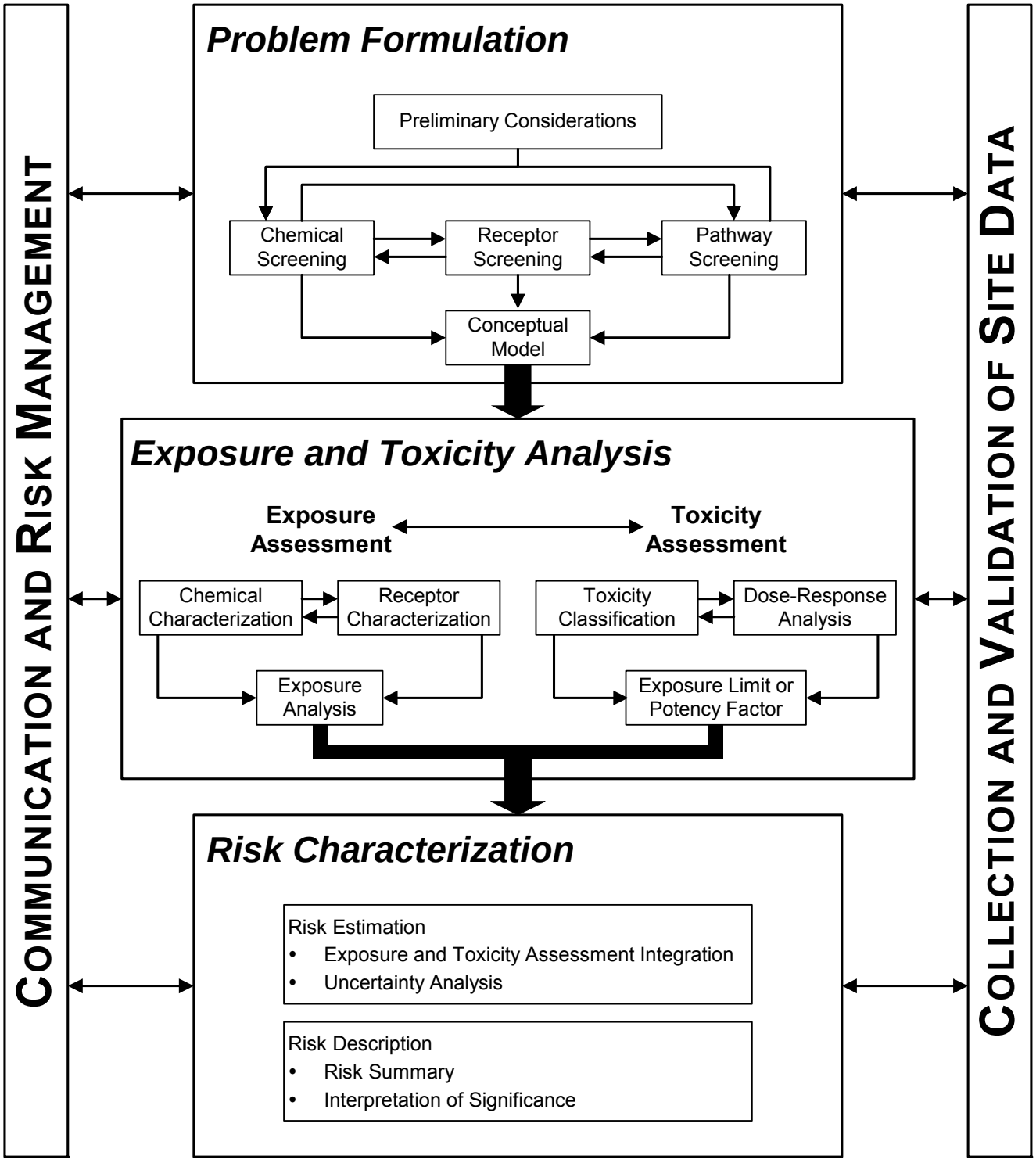
- a representative set of receptors (i.e., humans) that may be present in the vicinity of the project;
- chemicals that may be present at levels that may be harmful to receptors (termed Chemicals of Potential Concern [COPCs]); and
- pathways by which receptors may be exposed to COPCs (e.g., inhalation of COPCs in ambient air).

The information from the Problem Formulation is summarized in a Conceptual Site Model (CSM) which illustrates the pathways of the COPCs from their sources, through the relevant environmental media and to the receptors of interest. The approach to developing a CSM for an HHRA is outlined in Section 2.2.

ii) Exposure Assessment: The Exposure Assessment involves estimating the daily dose of a COPC received by the receptors for each relevant exposure pathway identified in the Problem Formulation. This value is called the Estimated Daily Intake (EDI) and is typically expressed as milligrams (mg) of a chemical per kilogram (kg) of body weight per day (mg/kg/day). The EDI is calculated from site-specific concentrations of COPCs in environmental media (e.g., water, sediment, fish, air, soil, or vegetation), the amount of time the receptor spends in the study area and receptor-specific parameters such as body weight, ingestion rate, and dietary preferences.

iii) Toxicity Assessment: The Toxicity Assessment provides the basis for assessing what is an acceptable dose and what dose may adversely affect the health of receptors. This involves identification of the potentially toxic effects of a COPC and determination of the dose to which a receptor can be exposed without experiencing adverse health effects. This value is called the Toxicity Reference Value (TRV). For human health, the TRV is expressed as mg of a COPC per kg of body weight per day (mg/kg/day).

iv) Risk characterization: The final component of an RA determines the potential for adverse health effects to occur. This is determined by comparing the dose received by the receptors (i.e., the EDI from the exposure assessment) with the dose that is determined to be acceptable (i.e., the TRV from the toxicity assessment). The characterization of risks includes consideration of the uncertainty and conservatism in the RA.



Source: Health Canada [unpublished] 1995.

Figure 2.1-2: Risk Assessment Framework. Modified from Health Canada (1995)

2.2 Conceptual Site Model

A CSM is developed in an HHRA to understand which substances (i.e., chemicals present at concentrations in excess of the applicable guidelines/standards or COPCs) are present in the LSA and RSA, how human receptors may use the affected areas, and the pathways of contact that are possible between these substances and the receptors. These substances, receptors, and pathways (the environmental risk components) are examined in detail to identify the "reasonably anticipated" combinations corresponding to potentially complete (i.e., significant) exposure pathways. Unimportant or incomplete pathways are eliminated from further consideration or are "screened out". The combinations of the environmental components that remain subsequent to the screening process, form the basis of the conceptual model, and are used to focus the HHRA.

The CSM addresses the following questions to characterize the effect of COPCs on human receptors:

- Which substances are present at elevated levels relative to applicable guidelines/standards?
- In which environmental media are they located (i.e., soil, groundwater, surface water, air, vegetation, etc.)?
- Is there a potential for the substances to migrate?
- In what concentrations are the substances present in the affected environmental media?
- Who are the human receptors (current and future users)?
- How is and/or will the NICO Project site be used?
- How can the human receptors come in contact with the substances?

Where exposure scenarios can be reasonably assumed to be complete, a more detailed examination or quantification of potential risks is required. The detailed assessment involves the remaining stages of the risk assessment including exposure assessment, toxicity assessment, and risk characterization.

2.3 General Approach

2.3.1 Pathway Analysis

In mining projects, potential impacts can only occur where there is a direct link between a project component or activity and the environment. Therefore, the HHRA focused on those components or activities of the NICO Project that could result in NICO Project-related emissions to the environment and corresponding potential effects to human health. Those components or activities of the NICO Project that could result in emissions to the environment were determined based upon the Project Description and the potential for releases of Project-related COPCs during the various phases of the NICO Project (i.e., construction, operations, closure, and post-closure; as summarized in Section 1.3), considering all proposed environmental design features and mitigation measures outlined in the DAR (Fortune 2011). This pathway analysis (the identification of the linkages between the NICO Project components or activities and corresponding potential effects to humans) are summarized in Table 2.3-1. Pathways were determined to be primary, secondary (minor), or as having no linkage, as described below:

- No linkage – pathway is removed by environmental design features and mitigation so that the NICO Project results in no measurable environmental change and effects to human health relative to baseline or guidelines values;

- Secondary – pathway could result in a minor environmental change, but would have a negligible effect on human health relative to baseline or guideline values; and
- Primary – pathway is likely to result in a measureable environmental change that could contribute to effects on human health relative to baseline or guidelines values.

Primary pathways require further analysis to determine the environmental significance from the NICO Project on human health. Pathways with no linkage to humans or that are considered minor (secondary) are not analyzed further because environmental design features and mitigation will remove the pathway (no linkage) or effects to humans can be determined to be negligible through a simple qualitative evaluation of the pathway. Pathways determined to have no linkage to humans or those that are considered to be secondary are not predicted to result in environmentally significant effects to human health. All primary pathways were assessed further in the HHRA.

The primary pathways assessed further in the HHRA are described below:

- Dust generated from the CDF during the operations phase, and potential impacts on human health via inhalation of dust and indirect impacts via deposition of dust on soil, vegetation, and waterbodies and subsequent ingestion; and
- Discharge of treated water from the ETF to Peanut Lake during operations, and potential impacts on downstream surface waters including Burke Lake and the Marian River.

The water management ponds were not evaluated directly in the HHRA because during operations, seepage collecting in the ponds will be collected and pumped to the treatment facility. In post-closure, seepage to the ponds will be routed to the open pit. Therefore, these ponds were not modelled in terms of water quality. However, given that water quality in the open pit was evaluated for the post-closure phase, seepage to the water management ponds has been indirectly assessed.

During closure and reclamation, water that accumulates in some of the SCPs, as well as the Surge Pond, will be passively treated in Wetland Treatment Systems and then released directly into Nico Lake (the detailed closure and reclamation plan is provided in Section 9.0 of the DAR; Fortune 2011). This is subject to the demonstration of the technical performance of the Wetland Treatment Systems. If the technical performance of the Wetland Treatment Systems is not demonstrated prior to closure, then the contingency will be to pump water from the SCPs, as well as from the Surge Pond, into the Open Pit. Initially, water will accumulate in the Open Pit. Just prior to pit overflow, the water quality at the top of the Flooded Open Pit will be evaluated, and a decision will be made about post-overflow treatment. The options include the following:

- providing the water quality is acceptable, overflow will be allowed to occur through wetland treatment system No. 4 into Peanut Lake with no further requirement for treatment;
- as a contingency, the Flooded Open Pit water can be treated in the pit by chemical or biological means, prior to the discharge of the overflow through wetland treatment system No. 4 into Peanut Lake; and
- as a contingency, a new ETF can be constructed and used to treat Flooded Open Pit water without spillover, with discharge through a diffuser into Peanut Lake.

Table 2.3-1: Contaminant Releases Associated with the NICO Project and Considered in the Human Health Risk Assessment

Project Component/ Activity	Effect Pathways	Valued Components	Environmental Design Features and Mitigation	Pathway Assessment
Construction & Operations: NICO Project Access Road	Dust generated from road traffic may deposit to soil, vegetation, and surface water. Receptors can be exposed to metals by direct pathways, such as inhalation, and indirect pathways via uptake through the food chain	Community receptors On-site workers	Access road will be as narrow as possible, while maintaining safe construction and operation practices. Watering of roads will suppress dust production. Enforcing speed limits will assist in reducing dust.	Secondary
Operations: Operation of Co-Disposal Facility	Dust generated from the Co-Disposal Facility may deposit to soil, vegetation, and surface water. Receptors can be exposed to metals by direct pathways, such as inhalation, and indirect pathways via uptake through the food chain	Community receptors On-site workers	Tailings will be deposited wet and exposed portions of the Co-Disposal Facility will be sprayed as necessary to control dust generation.	Primary
	Seepage may impact surface water quality around downstream waterbodies	Community receptors	Runoff from the tailings and co-disposal area will be captured and diverted to the Effluent Treatment Facility. Any potential acid-generating waste rock will be sequestered within the interior of the co-disposal area in a location that will freeze and remain frozen. Overburden directed to the co-disposal area will be used to cover any areas in the core of the pile where potentially acid-generating waste rock is to be sequestered to reduce any infiltration.	Secondary

Table 2.3-1: Contaminant Releases Associated with the NICO Project and Considered in the Human Health Risk Assessment (continued)

Project Component/ Activity	Effect Pathways	Valued Components	Environmental Design Features and Mitigation	Pathway Assessment
Construction & Operations: General construction and operation of mine and supporting infrastructure *Site water management*	Discharge of water (e.g., runoff, process water) to surface water could affect surface water quality	Community receptors	The NICO Project Water Management Plan will ensure that discharged water is contained on-site. Runoff from the mine site will be captured and diverted to the Effluent Treatment Facility. The site will have sufficient storage capacity to store both operating flows and storm events. Sewage will be treated and the effluent discharged to the tailings basin. Capture and reuse site water to reduce fresh water requirements. Water from tailings thickener and from the tailings basin will be recycled for grinding operations. Excess water from the collection pond (tailings basin) will be recycled in mill operations.	Secondary

Table 2.3-1: Contaminant Releases Associated with the NICO Project and Considered in the Human Health Risk Assessment (continued)

Project Component/ Activity	Effect Pathways	Valued Components	Environmental Design Features and Mitigation	Pathway Assessment
Construction & Operations: General construction and operation of mine and supporting infrastructure *Air emissions and dust deposition*	Air emissions, including dust, generated from onsite activities including, but not limited to, blasting, rock crushing, traffic, operation of equipment and trucks, are a source of direct and indirect exposure. Receptors can be exposed to metals by direct pathways, such as inhalation, and indirect pathways via uptake through the food chain	Community receptors On-site workers	Watering of roads will suppress dust production. Enforcing speed limits will assist in reducing dust. Regular maintenance of equipment to limit emissions. Processing equipment will use high efficiency scrubbers to limit emissions of particulate matter. Dust control systems on rock crushing and other dust generating equipment will limit dust emissions. Operating procedures will be developed that reduce dust generation (e.g. spraying). Tailings will be wet when placed in the Co-Disposal Facility.	Secondary
Closure and Reclamation: Co-disposal facility	Long-term seepage from the Co-Disposal Facility can change surface water quality	Community receptors	Develop a closure and reclamation plan (including water quality management post-closure such that water exiting the site meets site-specific quality criteria protective of human, wildlife, and aquatic health). Co-Disposal Facility will be capped during closure to isolate tailings and prevent direct exposure.	Secondary
Closure and Reclamation: Pit lake	Water quality in pit lake and outflow may be a source of exposure for all receptors	Community receptors	Flooded mine pit will be a sterile water body because of its physical dimensions with minimal primary production and habitat features capable of supporting aquatic life. As part of the closure plan, the flooded mine pit is not intended to be a functioning part of the ecosystem.	Primary

Table 2.3-1: Contaminant Releases Associated with the NICO Project and Considered in the Human Health Risk Assessment (continued)

Project Component/ Activity	Effect Pathways	Valued Components	Environmental Design Features and Mitigation	Pathway Assessment
Closure and Reclamation: Water treatment plant	Decommissioning of the water treatment plant may result in increased chemical concentrations in surface water	Community receptors	The decommissioning of the effluent treatment plant will occur once the effluent discharge to the wetlands (Seepage Collection Ponds) is at acceptable concentrations. The effluent treatment plant will be re-started and water will be treated, if necessary.	Secondary

2.3.2 Assessment Scenarios

To determine the potential effects of NICO Project-related emissions on human health, 2 scenarios were assessed in the HHRA:

- quantitative assessment of exposure to emissions from existing and approved sources (i.e., the Baseline Case); and
- quantitative assessment of exposure to cumulative emissions from existing and approved sources and from the NICO Project (i.e., Project Case).

The scenarios are described further below:

- The Baseline Case was assessed to gain an understanding of the environment as it currently exists without the NICO Project. This scenario used measured concentrations of chemicals in samples of environmental media (i.e., soil, water, sediment, fish, and vegetation) collected from the study area.
- The Project Case represents the change to the environment as a result of NICO Project components or activities for all phases of the NICO Project (construction, operations, closure, and post-closure), considering all proposed environmental design features and mitigation measures. This scenario was assessed quantitatively and used predicted concentrations of parameters in environmental media (i.e., air, soil, water, sediment, and vegetation) for the study area. The difference between the Baseline and Project Cases (i.e., Project Case concentration minus Baseline Case concentration) is the incremental change that is expected as a result of NICO Project-related emissions only. The Project Case scenario assessed exposure for the predicted worst-case phase (i.e., of the construction, operations, closure, and post-closure phases) of the NICO Project. It follows that if potential effects on human health are acceptable for the predicted worst-case phase of the NICO Project, than potential effects on human health for all other phases of the NICO Project will also be acceptable.

A qualitative assessment of exposure due to cumulative emissions was also considered in each component of the HHRA. The Cumulative Effects Case represents the cumulative change to the environment due to the NICO Project as described above and other foreseeable projects, developments, activities, and natural factors that influence the environment. Given that the potential changes to the environment as a result of other foreseeable projects, developments, activities, and natural factors could not be supported with numerical data, the Cumulative Effects Case was qualitatively evaluated.

The approach used in the HHRA was to use conservative assumptions to obtain worst-case estimates of exposure and risk and it is recognized that some of the assumptions could result in unrealistic predictions of exposure. However, this approach was used on the understanding that if there are no unacceptable risks to human health using conservative assumptions, there are no risks based on less conservative assumptions. If unacceptable risks to human health were identified using conservative assumptions, the assumptions were refined to obtain more realistic estimates of exposure and risk.

3.0 DATA USED IN THE HUMAN HEALTH RISK ASSESSMENT

To determine the incremental changes in the environment due to emissions from the NICO Project, the existing (or baseline) conditions of the environment must first be understood. Several studies were carried out in support of the NICO Project to characterize baseline environmental conditions. The baseline environmental data collected as part of these studies and used in support of the HHRA are summarized below:

- air quality data for the NICO Project and 2 stations near the site (Peanut Lake and Lion Lake) (Annex A of the DAR);
- water quality data for Nico, Peanut, and Burke lakes and the Marian River (Annex C of the DAR);
- sediment quality data for Nico, Peanut, and Burke lakes (Annex C of the DAR);
- fish tissue residue data for lake whitefish and northern pike from Nico, Peanut, and Burke lakes (Annex C of the DAR);
- tissue residue data for a variety of vegetation species (Annex I of the DAR); and
- soil quality data (Annex I of the DAR).

Baseline environmental conditions may change due to emissions from the NICO Project. Therefore, the HHRA also relied upon the following predicted environmental data:

- predicted air concentrations for the NICO Project and 5 off-site locations during the operations phase of the NICO Project, as determined through air quality modelling (Section 10.0 of the DAR);
- predicted water concentrations for Nico, Peanut, and Burke lakes and the Marian River for the construction, operation, closure, and post-closure phases of the NICO Project, as determined through water quality modelling (Section 7.0 of the DAR);
- predicted sediment concentrations for Nico, Peanut, and Burke lakes at closure, as determined through sediment quality modelling (Section 7.0 of the DAR);
- predicted soil concentrations for several locations in the study area, as determined using protocols provided in the Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA 2006b). The calculation of predicted soil concentrations is described further in Section 4.1.4.2 of this report; and
- predicted fish tissue and vegetation concentrations. The calculation of predicted fish tissue and vegetation concentrations is described further in Section 4.2.3.3 of this report.

4.0 HUMAN HEALTH RISK ASSESSMENT (HHRA)

4.1 General Approach

The HHRA was carried out consistent with the RA framework and methodology described in Section 2.0.

The HHRA was subdivided into 3 separate assessments to assess potential risks to human health: Air Quality Risk Assessment (further divided into acute and chronic), Particulate Matter Risk Assessment, and Multi-Media Risk Assessment. As such, the HHRA was organized such that each of these assessments was addressed separately. These assessments are described in more detail below.

- The Air Quality Risk Assessment focuses on exposure to substances that are emitted to air (i.e., criteria air contaminants [e.g., sulphur dioxide [SO_2], nitrogen dioxide (NO_2)], greenhouse gases [e.g., carbon monoxide (CO), ozone (O_3)], metals [e.g., arsenic, lead], polycyclic aromatic hydrocarbons [PAHs], volatile organic compounds [VOCs], and dioxins/furans). Project components and activities that may contribute to changes in air quality during the operations phase of the NICO Project were considered in the air quality predictions provided in Section 10.0 of the DAR.

  The human health risks associated with changes in air quality for short-term or acute exposures are assessed in Section 4.3, and those for long-term or chronic exposures are assessed in Section 4.4. Particulate matter is assessed separately in Section 4.5 as described below.

- The Particulate Matter Risk Assessment focuses on exposure to particulate matter that is emitted to air (i.e., particulate matter less than 10 microns in diameter [PM_{10}] and particulate matter less than 2.5 microns in diameter [$PM_{2.5}$]). Project components and activities that may contribute to changes in air quality during the operations phase of the NICO Project were considered in the air quality predictions provided in Section 10.0 of the DAR.

  The human health risks associated with changes to particulate matter is provided in Section 4.5.

- The Multi-Media Risk Assessment focuses on exposure to substances that are released to the environment, including soil, water, and other environmental media. Project components and activities that may contribute to changes in concentrations of substances in soil, water, wild game, berries, and fish during the operations phase of the NICO Project were considered in the air quality predictions provided in Section 10.0 of the DAR and in the water quality predictions provided in Section 7.0 of the DAR.

  The human health risks associated with changes to chemical concentrations in soil, water, wild game, berries, and fish are assessed in Section 4.6.

As introduced in Section 2.2.2, to determine the potential effects of NICO Project-related emissions on human health, 3 scenarios were assessed in each of these assessments within the HHRA, as follows:

- Quantitative assessment of exposure to emissions from existing and approved sources (i.e., the Baseline Case);
- Quantitative assessment of exposure to cumulative emissions from existing and approved sources from the NICO Project during the operations phase of the NICO Project (i.e., the Project Case); and

- Qualitative assessment of exposure to cumulative emissions from the Project Case and other nearby developments both existing and future (i.e., the Cumulative Effects Case).

To determine whether a chemical has the potential to affect human health (i.e., is identified as a COPC in the HHRA), the predicted peak concentrations for the Project Case were compared to relevant health-based screening standards and to baseline concentrations plus 10% (the Baseline Case). If a chemical only exceeds baseline concentrations and not its respective screening health-based criterion, it is not considered to be present at levels that would affect human health. If a chemical exceeds its respective screening health-based criterion but is present in the environment at levels within 10% of baseline concentrations, then it is not considered to be significantly different from baseline and no health effect is predicted to occur. When the chemical is present at levels measurably greater than baseline **and** greater than a health-based screening criterion, the NICO Project may contribute to increases in concentration that could affect human health where they did not before. Where both conditions are fulfilled, further assessment is carried out.

The addition of 10% to baseline concentrations is standard practice in Environmental Assessment's to represent the potential variability in baseline conditions. Comparison to a threshold of 10% above baseline concentrations was considered to represent a conservative evaluation of whether a measurable Project-related impact on environmental quality was likely to occur. Given spatial and temporal variability, field sampling variability, variability in laboratory methods, and the conservatism applied in the predictive models, a predicted increase in concentration of less than 10% above baseline concentrations was considered unlikely to reflect a "significant" change in environmental quality as a result of the NICO Project.

Note that the predicted emissions from the operations phase of the NICO Project was used to represent the Project Case as assessed in the HHRA. The operations phase was identified as the bounding case for the other phases of the NICO Project (i.e., construction, operations, closure, and post-closure) and the predictive air modelling (Section 10.0 of the DAR) and water quality modeling (Section 7.0 of the DAR) were carried out for the operations phase. The HHRA has used the predicted peak concentrations of COPCs in air and water as representative emissions from the NICO Project as input into the exposure calculations, which will result in a reasonable maximum exposure scenario. If the predicted risks are acceptable for the reasonable maximum scenario, then predicted risks can also be considered to be acceptable for other phases of the NICO Project when anticipated emission and discharge rates are lower.

4.2 Problem Formulation

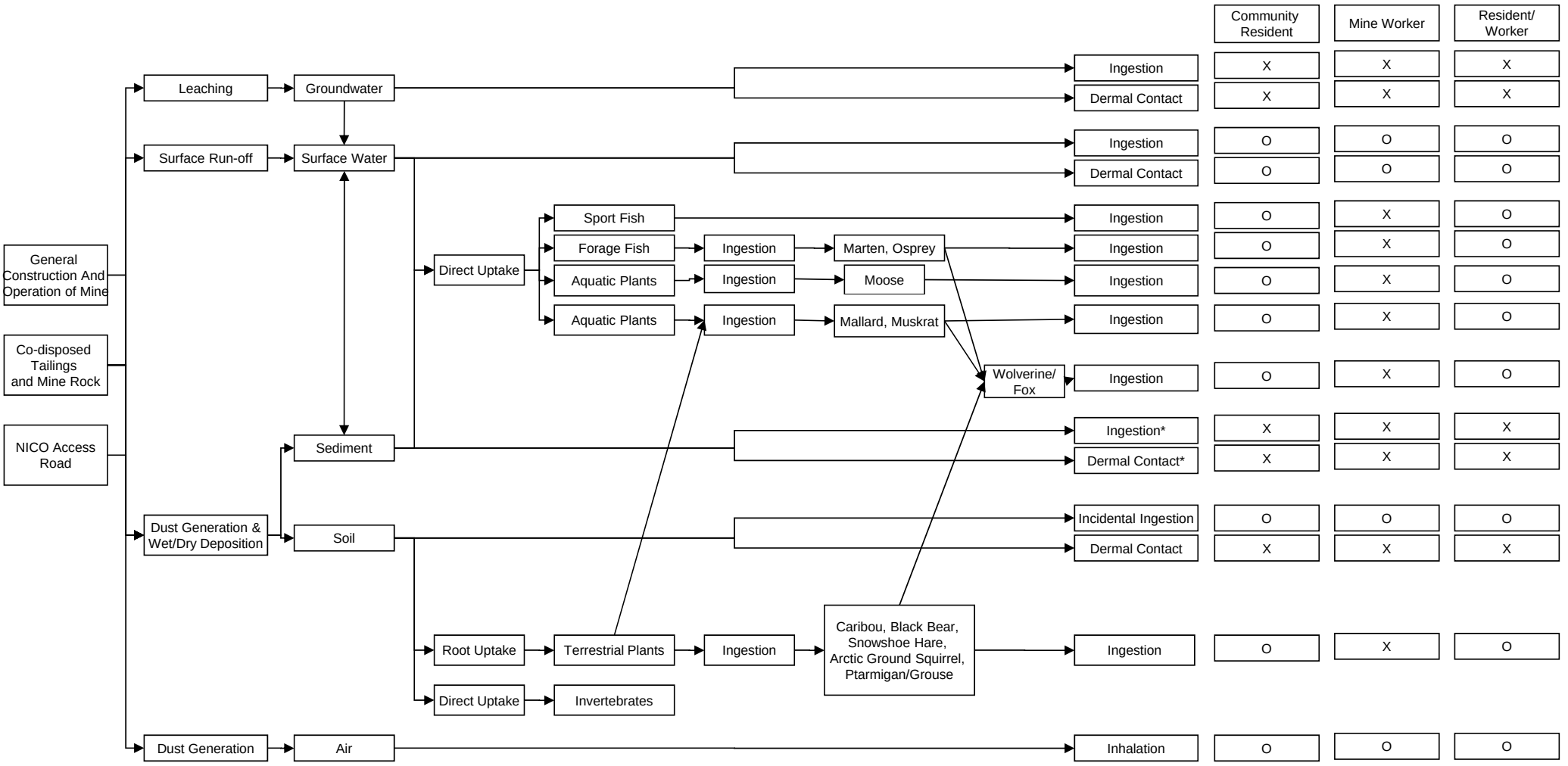
4.2.1 Conceptual Site Model

A CSM was developed for human health (Figure 4.2-1) using the approach outlined in Section 2.2. Three human receptors were identified in this HHRA, including a Community Resident, an On-Site Worker, and a Resident/Worker. The activity patterns and resulting complete exposure pathways are described further for each receptor below.

Conceptual Site Model for NICO Project – Human Receptors

Community Resident, Mine Worker, and Combined Resident/Worker

FIGURE 4.2-1



LEGEND

X Pathway incomplete and/or not evaluated

O Pathway complete and evaluated

NOTES

Please refer to the Wildlife CSM for the more detail on wildlife exposure pathways. Only the primary food type(s) for wildlife were included in the human CSM for simplicity.

Date: April 17, 2012	Golder Associates	CAD: AA
Project: 10-1373-0037 (2000)		CKD: RJ

The Community Resident was considered to be an individual that lives in one of the closest nearby communities (Gamètì and Whatì) and makes use of the culturally significant areas close to the NICO Project (Hislop Lake, Marian River, or Bea Lake) to hunt, fish, or trap. It was anticipated that Community Residents would spend up to one month at these culturally significant areas and collect enough wild game, fish, and berries to feed their families for the entire year. Therefore, this receptor was considered to be exposed to the soil, air, and surface water while at the culturally significant areas, and to be consuming wild game, fish, and berries throughout the year. As a worst-case assumption, it was also considered possible that this receptor could spend up to one month at the Maximum Point of Impingement (MPOI) location should that receptor spend time around Nico, Peanut, Lou, or Burke lakes; however, based upon the disturbances around the NICO Project site during operations, this is an unlikely scenario.

The On-Site Worker was considered to be an individual that both resides and works at the NICO Project site throughout the year. Workers would be expected to work 12-hour shifts on the mine site and sleep at the on-site worker camp. This receptor was considered to be exposed to soil and air at the Worker Camp and, as a worst-case, the MPOI location. This receptor was not considered to be consuming wild game, fish, and berries from the local area.

The Resident/Worker receptor was considered to be an individual that resides in a nearby community and works at the mine site. This receptor would be expected to be exposed to soil and air while at the mine site and also to be consuming wild game, fish, and berries.

4.3 Acute Air Quality Risk Assessment

4.3.1 Problem Formulation

The objective of the acute air quality RA is to evaluate substances potentially emitted from the NICO Project that may pose a health outcome following short-term or acute exposure duration (e.g., 1-hour) to human receptors.

4.3.1.1 Receptor Locations

Acute health effects associated with changes in air quality were assessed based on a regional and local basis. Locations where people are known or anticipated to spend their time were identified within the RSA. The RSA for human health is defined as a 94 km (east-west) by 124 km (north-south) area that encompasses the NICO Project and other existing and approved industrial emissions sources from Gamètì, Whatì, and Snare Rapids.

The LSA for human health is defined as a 30 km by 30 km area in the immediate vicinity of the NICO Project where the majority of air quality changes due to the NICO Project are expected to occur. Two receptor locations identified within the LSA are the worker camp and the MPOI, which is assumed to be the NICO Project site boundary. Five receptor locations were identified within the RSA and represent nearby communities and culturally significant locations.

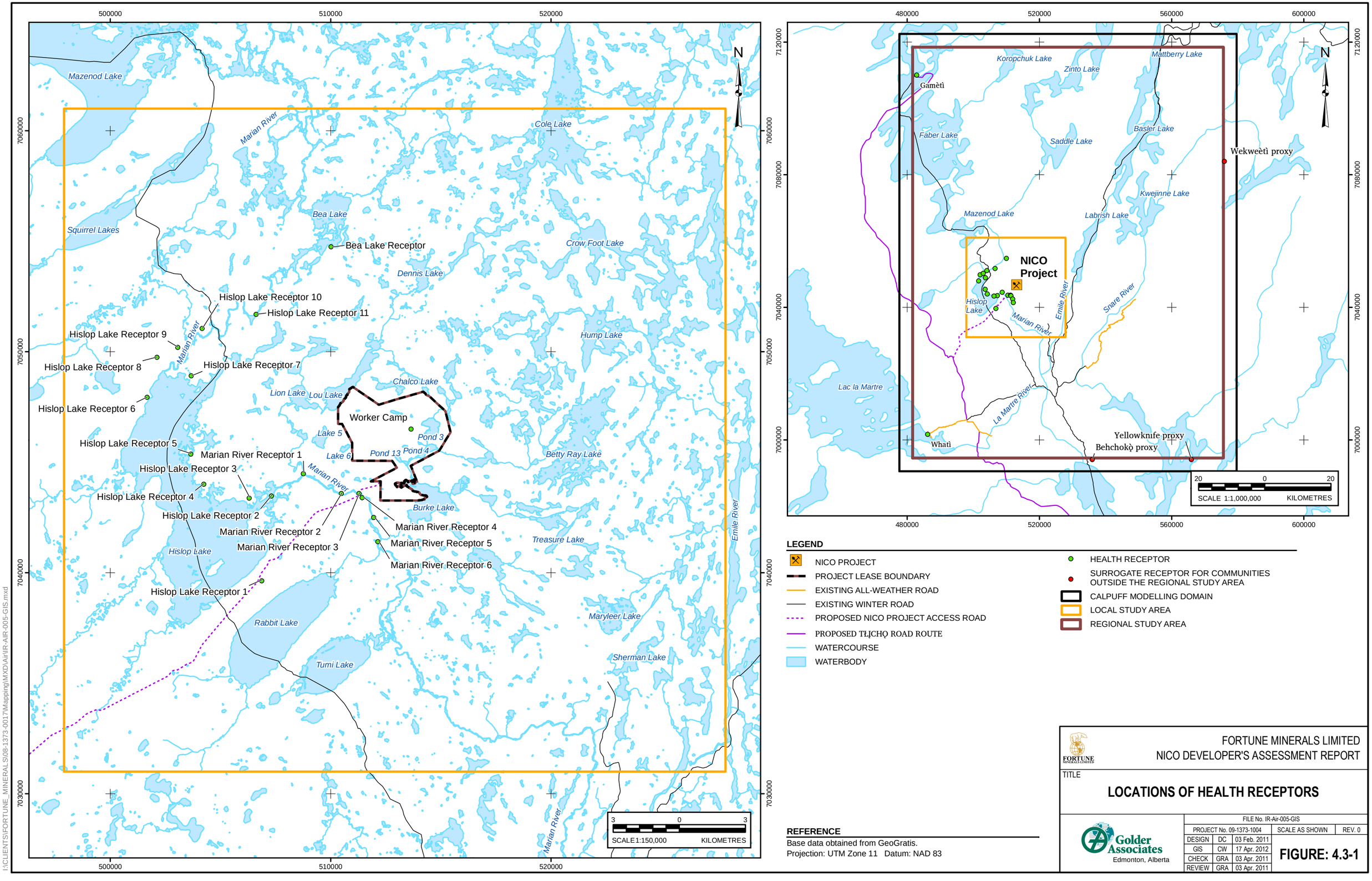
Potential human health effects related to short-term inhalation exposure for the NICO Project were evaluated for the following 7 receptor locations (Figure 4.3-1):

- MPOI;
- Worker Camp;
- community of Gamètì;

- community of Whatì;
- culturally significant location of Hislop Lake;
- culturally significant location of Marian River; and
- culturally significant location of Bea Lake.

To represent the off-site culturally significant locations of Hislop Lake and Marian River, several individual points were identified (Figure 4.3-1) based upon the locations of cabins, camping sites, other culturally significant areas, and locations of importance for the First Nations including burial sites. Out of the multiple individual points for each culturally significant location, the point with the highest predicted concentrations of substances in air was quantitatively assessed in the HHRA.

In addition, 2 communities were identified and assessed in the HHRA: Gamètì and Whatì. Proxy locations for the more distant communities of Wekweetì, Behchokǫ̀, and Yellowknife were also considered. If the predicted concentrations of substances in air at the closer communities of Gamètì and Whatì were acceptable, quantitative assessment of the 3 more distant communities was not carried out because smaller changes to air quality were predicted at these more distant locations.



LEGEND

- NICO PROJECT
- PROJECT LEASE BOUNDARY
- EXISTING ALL-WEATHER ROAD
- EXISTING WINTER ROAD
- PROPOSED NICO PROJECT ACCESS ROAD
- PROPOSED TŁĮCHǪ ROAD ROUTE
- WATERCOURSE
- WATERBODY
- HEALTH RECEPTOR
- SURROGATE RECEPTOR FOR COMMUNITIES OUTSIDE THE REGIONAL STUDY AREA
- CALPUFF MODELLING DOMAIN
- LOCAL STUDY AREA
- REGIONAL STUDY AREA

REFERENCE

Base data obtained from GeoGratis.
Projection: UTM Zone 11 Datum: NAD 83

FORTUNE MINERALS LIMITED
NICO DEVELOPER'S ASSESSMENT REPORT

TITLE

LOCATIONS OF HEALTH RECEPTORS

Golder Associates
Edmonton, Alberta

FILE No. IR-Air-005-GIS			
PROJECT No. 09-1373-1004		SCALE AS SHOWN	REV. 0
DESIGN	DC	03 Feb. 2011	
GIS	CW	17 Apr. 2012	
CHECK	GRA	03 Apr. 2011	
REVIEW	GRA	03 Apr. 2011	

FIGURE: 4.3-1

I:\CLIENTS\FORTUNE_MINERALS\08-1373-0017\Mapping\MXD\Air\IR-AIR-005-GIS.mxd

4.3.1.2 Selection of Acute Air Thresholds

Chemicals that may change in concentration as a result of the NICO Project were identified based upon the NICO Project components and activities that result in emissions to air as shown in Section 2.3 above. These chemicals include the following:

- criteria air contaminants (e.g., SO_2, NO_2);
- greenhouse gases (e.g., CO, O_3);
- metals (e.g., arsenic, lead);
- PAHs;
- VOCs; and
- dioxins/furans.

Peak 1-hour and 24-hour concentrations for criteria air contaminants, greenhouse gases, metals, PAHs, VOCs, and dioxins and furans were compared to the most conservative of the available acute health-based thresholds from the following agencies:

- Alberta Environment (AENV 2009);
- Agency of Toxic Substances and Disease Registry (ATSDR 2009, internet site);
- California Office of Environmental Health Hazard Assessment (OEHHA 2008, internet site);
- Government of the Northwest Territories (GNWT 2005, internet site);
- Ontario Ministry of Environment and Energy (OMEE 2005; OMEE 2008);
- Texas Commission on Environmental Quality (TCEQ 2009, internet site); and
- World Health Organization (WHO 2000; WHO 2005).

The standards provided by the GNWT were adopted, where available, given that these are territory-specific, they are generally health-based, and are largely adopted from the Canada Wide Standards. If GNWT did not provide a standard, then the most protective health-based threshold from another jurisdiction was used. If no health-based standards were available, then a standard based upon another endpoint, such as odour, was used. Additionally, the TCEQ was only used when thresholds from other jurisdictions were not available given that detailed supporting documentation is not always available from TCEQ.

Each of these agencies derived health-based air thresholds based upon a prescribed level of protection. Most often, these air thresholds are presented as air concentrations at and below which health effects are not expected to occur and may incorporate additional safety factors. Therefore, a predicted air concentration greater than the threshold indicates that a health effect is possible, but not certain. Further evaluation is required to determine the likelihood of that health effect occurring.

The available health-based 1-hour and 24-hour thresholds from the agencies listed above were considered for use in the Acute Air Quality Risk Assessment (Tables A.1 and A.2 in Appendix A). The threshold was selected

for comparison to the predicted 1-hour and 24-hour peak concentrations of substances if it was the most protective (i.e., the lowest) out of all of the thresholds and its supporting information was available for review.

In some cases, air thresholds were set for certain compounds (shown below) that are not based upon chemical toxicity of the compound to human health; instead, these air thresholds were based upon the toxicity of particulate matter itself. Given that particulate matter is assessed separately in Section 4.5, exceedances of the 1-hour or 24-hour air thresholds for these compounds were not retained in this assessment.

- Aluminum
- Bismuth
- Calcium
- Iron
- Lithium
- Molybdenum
- Nickel
- Palladium
- Potassium
- Silicon
- Sodium
- Strontium
- Titanium
- Tungsten
- Zinc

Many of the chemicals had sufficient toxicity information and screening standards available, which allowed them to be assessed as individual chemicals (specifically, metals and metallic combustion by-products, such as lead). However, other chemicals were assessed as groups because insufficient toxicity information was available for the individual chemicals. In these cases, the individual chemicals were grouped based upon their physical/chemical properties and mixture-specific toxicity data. These chemical groups were represented by a surrogate chemical for which toxicity information was available. This approach was applied to individual chemicals within PAHs, VOCs, and dioxins and furans. The groupings are shown below:

- Acrolein was evaluated as a group, which includes acrolein and methacrolein. Given that toxicity information was not available for methacrolein, the sum of both chemicals was assessed using acrolein as its surrogate in the HHRA.
- Aldehydes were evaluated as a group, which includes acetaldehyde, benzaldehyde, tridecanal, heptanal, decanal, undecanal, dodecanal, propanal, butanal, octanal, nonanal, crotonaldehyde, 2,5-dimethylbenzaldehyde, and hexanal. Within this group, some chemicals are classified as being possibly carcinogenic to humans and others are not classifiable as to their carcinogenicity. Therefore, the aldehyde group was evaluated both as a carcinogen and a non-carcinogen, using the surrogate acetaldehyde for which both threshold and non-threshold toxicity reference values are available.
- C2-C8 Aliphatics were evaluated as a group, which includes propylene, n-butane, 2-methylpentane, 2,4-dimethylpentane, methylcyclohexane, n-pentane, cyclohexane, n-octane, propene, isobutene, heptane, cyclopentane, trans-2-hexene, 2,2,4-trimethylpentane, 3-methyl-1-butene, 2-methyl-1-butene, 2,3-dimethylpentane, 2,3,4-trimethylpentane, 2,3-dimethylhexane, 3-methylhexane, 2,4-dimethylhexane, 2,4-dimethylhexane, cis-2-butene, 2-methylhexane, 2,5-dimethylhexane, 2-methylheptane, 3-ethylhexane, trans-2-butene, ethene, ethyne, 2,2-dimethylbutane, cis-2-hexene, isopentane, 2,3-dimethylbutane, e-methylpentane, and methylcyclopentane. These chemicals are all considered to be non-carcinogenic. Cyclohexane was used as a surrogate for this group.
- C9-16 Aliphatics were evaluated as a group, which includes nonylcyclohexane, n-nonane, n-dodecane, octylcyclohexane, decylcyclohexane, pentylcyclohexane, farnesane, 2,6,10-trimethyltridecane, hexylcyclohexane, n-hexadecane, heptylcyclohexane, n-tridecane, n-tetradecane, n-pentadecane, and

norfarnesane. These chemicals are all considered to be non-carcinogenic. Decane was used as a surrogate for this group.

- C9-16 Aromatics were evaluated as a group, which includes n-propylbenzene, fluorenone, m-ethyltoluene, p-ethyltoluene, indanone, 8b,13a-dimethyl-14b-[3'-methylbuthyl]-podocarpane, 8b,13a-dimethyl-14b-n-butylpodocarpane, xanthone, and acetophenone. Ethylbenzene was used as a surrogate for this group.
- C16+ Aliphatics were evaluated as a group, which includes n-eicosane, dodecylcyclohexane, tetradecylcyclohexane, pristine, norpristane, undecylcyclohexane, n-octadecane, tridecylcyclohexane, pentadecylcyclohexane, n-heptadecane, n-nonadecane, n-heneicosane, and phytane. These chemicals are all considered to be non-carcinogenic. Given that no air standards or TRVs were available for any of the C16+ aliphatics, decane was selected as the surrogate for this chemical group. Where air standards or TRVs were not available for decane, those of cyclohexane were used.
- Chlorobenzenes were evaluated as a group, which includes chlorobenzene, 1,2,3,5-tetrachlorobenzene, 1,2,4,5-tetrachlorobenzene, 1,2,3,4-tetrachlorobenzene, pentachlorobenzene, and hexachlorobenzene. Chlorobenzene was used as a surrogate for this group.
- Trimethylbenzenes were evaluated as a group, which includes 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene. 1,2,4-Trimethylbenzene was used as a surrogate for this group.

Additionally, one compound that was anticipated to be emitted from the NICO Project did not have toxicity information available (i.e., octachlorostyrene). Therefore, styrene was used to represent octachlorostyrene based upon similar physical/chemical properties.

Chemical concentrations (1-hour and 24-hour peak concentrations) were predicted for the NICO Project for individual compounds or chemicals groups as appropriate among criteria air contaminants, greenhouse gases, metals, PAHs, VOCs, and dioxins and furans for all of the receptor locations during the bounding operations phase.

4.3.1.3 Comparison of Predicted Peak Concentrations to Acute Thresholds

The predicted peak 1-hour and 24-hour concentrations of chemicals in air were compared to the most protective acute thresholds and to baseline concentrations to determine whether further assessment was required (Tables A.3 and A.4 in Appendix A). The maximum peak 1-hour and 24-hour concentrations out of all 7 receptor locations were selected for identification of COPCs. If the predicted peak concentrations were significantly higher than baseline (i.e., if peak concentrations were greater than baseline + 10%) and greater than the selected acute thresholds, then the chemical was retained as a COPC and considered further in the Acute Air Quality Risk Assessment. Chemicals that were retained as a COPC were assessed with respect to potential human health effects at all receptor locations.

Based upon the screening process outlined above, NO_2 and arsenic were retained as COPCs for the 1-hour averaging time, and acrolein (group containing acrolein and methacrolein), arsenic, and cobalt were retained for the 24-hour averaging time. These COPCs were assessed further in the Risk Characterizationsection below.

4.3.2 Exposure Assessment

The predicted 1-hour and 24-hour peak concentrations for identified COPCs were applied as the exposure point concentrations to which receptors, at each receptor location identified above, are exposed. It should be noted that the predicted peak concentration for "acrolein" is the sum of acrolein and methacrolein.

4.3.3 Toxicity Assessment

As discussed above, acute air thresholds provided by several agencies were reviewed and the most appropriate thresholds were selected for use in this assessment. These thresholds were used for comparison with the predicted peak 1-hour and 24-hour concentrations of each COPC. This comparison is shown in the Risk Characterization section below.

4.3.4 Risk Characterization

For each of the COPCs identified above, a Hazard Quotient (HQ) was calculated for each receptor location as follows:

$$HQ = \frac{Peak\ COPC\ concentration\ in\ air\ (\mu g/m^3)}{Acute\ threshold\ concentration\ (\mu g/m^3)}$$

An example calculation for 1-hour NO_2 concentrations at Gamètì for the Project Case is presented as follows:

$$\text{Gamètì } HQ = \frac{predicted\ peak\ 1\text{-}hour\ NO_2\ concentration\ in\ air\ (\mu g/m^3)}{Acute\ threshold\ concentration\ (\mu g/m^3)} = \frac{26.8\ \mu g/m^3}{400\ \mu g/m^3} = 0.07$$

An HQ value greater than 1 indicates that predicted exposure is greater than the threshold. For parameters and locations where HQ values were greater than 1, further impact analysis was conducted to determine the residual effects.

The HQ values were less than 1 at all community locations and culturally significant locations for the 1-hour and 24-hour predicted peak concentrations (Tables 4.3-1 and 4.3-2 below). One of the 6 Marian River receptor point locations (Marian River Receptor 1) exceeded the 1-hour threshold for arsenic. Several HQ values exceeded 1 at the on-site worker camp and MPOI for both the 1-hour and 24-hour periods.

Table 4.3-1: Hazard Quotients for 1-hour Averaging Time

Parameter	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Worker Camp	Maximum Point of Impingement
Baseline Case										
Nitrogen Dioxide	0.009	0.007	0.007	0.007	0.009	0.008	0.1	0.07	0.008	0.02
Arsenic	0.00004	0.00008	0.00004	0.00009	0.0001	0.00009	0.00002	0.00007	0.0002	0.0004
Application Case										
Nitrogen Dioxide	0.03	0.04	0.03	0.4	0.4	0.4	0.1	0.09	**2.3**	**1.0**
Arsenic	0.01	0.002	0.003	**1.1**	0.9	0.6	0.007	0.006	**25**	**36**

Notes: Units are in micrograms per cubic metre.
Bold and shaded values exceed the target HQ of 1.

Table 4.3-2: Hazard Quotients for 24-hour Averaging Time

Parameter	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Worker Camp	Maximum Point of Impingement
Baseline Case										
Acrolein	0.00001	0.000008	0.000005	0.000009	0.00001	0.000009	0.00008	0.00002	0.00001	0.0008
Arsenic	0.000002	0.000006	0.000005	0.00001	0.00002	0.000008	0.000003	0.000008	0.00002	0.00004
Cobalt	0.000004	0.000002	0.000002	0.000002	0.000004	0.000002	0.000048	0.00001	0.000005	0.00001
Application Case										
Acrolein	0.005	0.007	0.005	0.09	0.09	0.07	0.007	0.01	1	**3**
Arsenic	0.001	0.0001	0.0003	0.2	0.1	0.08	0.0006	0.0005	**4**	**7**
Cobalt	0.0005	0.00005	0.0002	0.06	0.05	0.03	0.0002	0.0003	**1**	**3**

Notes: Units are in micrograms per cubic metre.
Bold and shaded values exceed the target HQ of 1.
Acrolein = sum of acrolein + methacrolein.

To determine whether the COPCs with calculated HQs greater than 1 could have a significant impact on human health, a magnitude of effects assessment was carried out.

4.3.5 Magnitude of Effects Assessment

For COPCs and receptor locations where HQ values were greater than 1 (Table 4.3-3), additional analysis was carried out to determine the potential magnitude of the effect.

Table 4.3-3: Summary of Chemicals of Potential Concerns and Receptor Locations with HQ > 1

COPC	1-hour			24-hour	
	Marian River	MPOI	Worker Camp	MPOI	Worker Camp
Nitrogen Dioxide		✓	✓		
Acrolein				✓	
Arsenic	✓	✓	✓	✓	✓
Cobalt		✓	✓	✓	✓

Acrolein = sum of acrolein + methacrolein; MPOI = Maximum Point of Impingement

The Acute Air Quality Risk Assessment incorporated several conservative assumptions, which would be expected to overestimate potential exposure and risk for the assessed receptors. The following analysis was completed to determine whether the NICO Project has a negligible, low, moderate, or high potential for unacceptable acute health effects:

- comparison of the maximum, 95th and 75th percentile concentrations in air to the available acute thresholds;
- assessment of the number of hours or days that the predicted peak concentrations could be greater than the available acute thresholds;
- comparison of the magnitude of Project Case concentrations to Baseline Case concentrations;
- assessment of the conservatism in the air modelling assumptions used to predict future concentrations;
- assessment of the conservatism in the acute thresholds for each COPC; and
- assessment of the potential short-term health effects that may occur at the predicted concentrations.

The magnitude of effects assessment indicated that the NICO Project would likely have an overall negligible potential for acute health effects due to the COPCs identified (Tables 4.3-4 to 4.3-7).

Table 4.3-4: Magnitude of Effects Assessment for Nitrogen Dioxide (1-hour)

Analysis Criteria	Discussion
Comparison of the maximum, and 95th and 75th percentiles to acute thresholds	The maximum predicted 1-hour concentration of NO_2 of 465.4 µg/m^3 at the Worker Camp exceeded the 1-hour threshold of 200 µg/m^3. The 95th (75.1 µg/m^3) and 75th (5.8 µg/m^3) percentiles did not exceed the acute threshold. The maximum predicted 1-hour concentration of NO_2 of 204.1 µg/m^3 at the MPOI exceeded the 1-hour threshold of 200 µg/m^3. The 95th (105.8 µg/m^3) and 75th (31.2 µg/m^3) percentiles did not exceed the acute threshold.
Frequency of exceedance	There were 137 exceedances of the 1-hour threshold of NO_2 at the Worker Camp and there was 1 exceedance at the MPOI. The exceedances at the Worker Camp account for approximately 1.6% of the maximum operations year, or the 137 maximum hours during the highest emissions year out of the 18 years of the operations phase of the NICO Project. The exceedance at the MPOI accounts for approximately 0.01% of the maximum operations year, or the one maximum hour during the highest emissions year out of the 18 years of the operations phase of the NICO Project. Predicted emissions for the lower emission years would be expected to result in fewer exceedances.
Conservatism and uncertainty in air predictions	The predictive air modelling assumed that the NICO Project and all existing and approved developments operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time. Additionally, the predictive air modeling results were provided for the year with the highest predicted emissions out of the 18 years of the operations phase of the NICO Project.
Conservatism in the acute threshold	The WHO 1-hour threshold (200 µg/m^3) used in this assessment is based on threshold study in which no adverse respiratory responses were observed in asthmatics exposed to 190 µg/m^3; the supporting documentation indicated that at double the guideline value (i.e., 400 µg/m^3), small effects on asthmatics may occur. The guideline value was set at 200 µg/m^3 to be protective of simultaneous exposures to other airborne allergens that may exacerbate the asthmatic respiratory response. There are also health-based thresholds provided by OMOE (400 µg/m^3) and CalEPA (470 µg/m^3). There is no supporting documentation on the OMOE threshold, but the CalEPA threshold was developed from studies where no adverse health effects were seen in a sensitive subgroup of asthmatics at or below the threshold. Overall, the selected acute threshold is considered to be associated with a moderate to low level of conservatism given that it is not associated with an adverse response in asthmatics, and small responses have been observed at concentrations that are twice as high as the threshold.
Potential acute health effects	As described above, the WHO 1-hour threshold of 200 µg/m^3 is not associated with potential acute health effects at that level; at NO_2 concentrations twice the guideline (i.e., 400 µg/m^3), adverse respiratory effects in asthmatics have been reported. The CalEPA acute threshold of 470 µg/m^3 is based upon increased airway reactivity in asthmatics; no supporting documentation for the OMOE threshold of 400 µg/m^3 was provided.
Magnitude of effect	The predicted peak 1-hour exposure concentration for NO_2 exceeds the selected air threshold (i.e., WHO guideline). Due to the infrequency of exceedances at the Worker Camp and MPOI locations and the conservatism in the air modeling, the magnitude of effect for NO_2 is considered to be negligible.

MPOI = Maximum Point of Impingement; NO_2 = nitrogen dioxide; µg/m^3 = microgram per cubic metre

Table 4.3-5: Magnitude of Effects Assessment for Acrolein (24-hour)

Analysis Criteria	Discussion
Comparison of the maximum, and 95th and 75th percentiles to acute thresholds	The maximum predicted 24-hour concentration of acrolein (the sum of acrolein + methacrolein) of 0.237 µg/m^3 and the 95th percentile of 0.118 µg/m^3 exceeded the acute threshold of 0.08 µg/m^3 at the MPOI. The 75th percentile of 0.034 µg/m^3 did not exceed the acute threshold.
Frequency of exceedance	There were 35 exceedances of the 24-hour threshold of acrolein at the MPOI. The exceedances at the MPOI account for approximately 9.6% of the maximum year. These exceedances represent the 2 maximum hours during the highest emissions year out of the 18 years of the operations phase of the NICO Project. Predicted emissions for the lower emission years would be expected to result in fewer exceedances.
Conservatism and uncertainty in air predictions	The predictive air modelling assumed that the NICO Project and all existing and approved developments operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time. Additionally, the predictive air modeling results were provided for the year with the highest predicted emissions out of the 18 years of the operations phase of the NICO Project.
Conservatism in the acute threshold	The OMOE derived a health-based threshold using a LOAEL of 920 µg/m^3 (non-neoplastic lesions in nasal epithelium of rats) from 3 animal studies. The LOAEL was adjusted based on 6 hours daily exposure, 5 days per week and applying a regional gas dose ratio of 0.14 resulting in a LOAEL of 23 µg/m^3. A total uncertainty factor of 300 was applied (3 to extrapolate from a LOAEL to a NOAEL, 3 for interspecies extrapolation, 3 to extrapolate from subchronic to chronic exposure, and 10 for intraspecies variability, to account for sensitive individuals).
Potential acute health effects	The OMOE 24-hour threshold was set to be protective of the development of nasal lesions following chronic exposure to acrolein. The CalEPA 8-hour threshold of 0.7 µg/m^3 is based upon prevention of nasal lesions following a short-term (8-hour) exposure. Therefore, the use of a chronic threshold for a short-term exposure is a conservative approach.
Magnitude of effect	The predicted peak 24-hour exposure concentration for acrolein exceeds the selected air threshold (i.e., OMOE threshold). Due to the unlikelihood of a receptor being present at the MPOI, the conservatism in the air modelling, and the use of a chronic threshold for a short-term exposure duration, the magnitude of effect for acrolein is considered to be negligible.

LOAEL = lowest observed adverse effect level; MPOI = Maximum Point of Impingement; µg/m^3 = microgram per cubic metre

Table 4.3-6: Magnitude of Effects Assessment for Arsenic (1-hour and 24-hour)

Analysis Criteria	Discussion
Comparison of the maximum, and 95th and 75th percentiles to acute thresholds	1-hour: At the Marian River location, only the maximum predicted 1-hour concentration of 0.113 µg/m^3 exceeded the 1-hour threshold of 0.1 µg/m^3. The 95th (0.0095 µg/m^3) and 75th (0.000025 µg/m^3) percentiles did not exceed the 1-hour threshold. At the Worker Camp, the maximum predicted 1-hour concentration of arsenic of 2.50 µg/m^3 and the 95th percentile of 0.71 µg/m^3 exceeded the 1-hour threshold of 0.1 µg/m^3. The 75th percentile of 0.084 µg/m^3 did not exceed the 1-hour threshold. At the MPOI, the maximum, 95th percentile, and 75th percentile predicted concentrations of arsenic of 3.59, 1.54, and 0.16 µg/m^3, respectively, exceeded the 1-hour threshold of 0.1 µg/m^3. 24-hour: At the Worker Camp, the maximum and 95th percentile predicted 24-hour concentrations of arsenic of 1.12 and 0.51 µg/m^3, respectively, exceeded the 24-hour threshold of 0.3 µg/m^3. The 75th percentile of 0.14 µg/m^3 did not exceed the 24-hour threshold. At the MPOI, the maximum and 95th percentile predicted 24-hour concentrations of arsenic of 1.99 and 1.14 µg/m^3, respectively, exceeded the 24-hour threshold of 0.3 µg/m^3. The 75th percentile of 0.30 µg/m^3 did not exceed the 24-hour threshold.
Frequency of exceedance	1-hour: There were 3 exceedances of the 1-hour threshold for arsenic at Marian River (approximately 0.03% of the maximum year), 1985 exceedances at the Worker Camp (approximately 23% of the maximum year), and 2700 exceedances at the MPOI (approximately 31% of the maximum year). 24-hour: There were 52 exceedances at the Worker Camp (approximately 14% of the maximum year) and 92 exceedances at the MPOI (approximately 25% of the maximum year).
Conservatism and uncertainty in air predictions	The predictive air modelling assumed that the NICO Project and all existing and approved developments operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time. Additionally, the predictive air modeling results were provided for the year with the highest predicted emissions out of the 18 years of the operations phase of the NICO Project; predicted emissions for the other 17 years of the operations phase would be expected to result in fewer exceedances. An examination of the sources of dust emissions (i.e., TSP) was undertaken to ascertain the relative contribution of road dust to the air quality predictions. Most of the TSP generated during the operations phase was considered to be due predominantly to road dust rather than other process-related sources such as crushing. More importantly, the predictive air modelling considered that dust suppression measures, such as road watering, would only occur during the summer period (i.e., 5 months during May 1 to September 30) and that dust generation is possible during the

Table 4.3-6: Magnitude of Effects Assessment for Arsenic (1-hour and 24-hour) (continued)

Analysis Criteria	Discussion
	winter period (i.e., 7 months during October 1 to April 30) despite frozen ground and/or snow-covered roads that would preclude fugitive dust generation. Additionally, the predictive air modeling conservatively assumed that road surface material was equivalent to waste rock, which was estimated to contain arsenic at 0.112% by weight. A comparison of the peak/maximum concentration of TSP in the summer versus the winter periods indicated that arsenic concentrations were predicted to be 4 times higher at the MPOI and 3 times higher at the worker camp during the winter period compared to the summer period for the 1-hour averaging period, and approximately 8 times higher during the winter than the summer for both locations for the 24-hour averaging period. Additionally, approximately 5 times the number of exceedances during the summer period was expected for the winter period. It is anticipated that the predicted arsenic concentrations used in the HHRA is significantly overestimated during the winter period due to frozen ground and/or snow-covered roads that were not considered in the predictive air modeling.
Conservatism in the acute threshold	The Alberta Ambient Air Quality Objective 1-hour threshold of 0.1 µg/m^3 was adopted from the Texas Commission on Environmental Quality. It is based on health, but supporting documentation is not available. It is similar to the CalEPA value (0.2 µg/m^3) which is based on a LOAEL (0.26 mg/m^3) for decreased fetal weight in mice following maternal inhalation exposure. An uncertainty factor of 1000 was applied to the CalEPA threshold: 10 for extrapolation from animals to humans, 10 for sensitive individuals and 10 for use of a LOAEL. The OMOE 24-hour threshold of 0.3 µg/m^3 is based on a health endpoint, but supporting documentation is not available.
Potential acute health effects	Supporting documentation for the selected 1-hour threshold describing potential health effects was unavailable. However there is a similar acute threshold provided by CalEPA (0.2 µg/m^3) that is based on decreased fetal weight in mice. Supporting documentation for the selected 24-hour threshold describing potential health effects was unavailable.
Magnitude of effect	The predicted peak 1-hour and 24-hour exposure concentrations for arsenic exceeded the selected air thresholds (i.e., AAAQO and OMOE, respectively). Due to the infrequency of exceedance at Marian River, the magnitude of effect for arsenic at this location is considered to be negligible. The magnitude of effects at the MPOI and the Worker Camp were considered to be low based upon the high degree of conservatism in the predictive air modeling, which is largely driven by the assumption that there would be no dust suppression during the winter months. Although the exceedances were predicted to occur between 20 and 30% of the maximum year, the air modeling was conservative and the selected air threshold is half of that of the CalEPA value, which incorporates an uncertainty factor of 1000.

HHRA = human health risk assessment; LOAEL = lowest observed adverse effect level; MPOI = Maximum Point of Impingement; TSP = total suspended solids; µg/m^3 = microgram per cubic metre

Table 4.3-7: Magnitude of Effects Assessment for Cobalt (1-hour and 24-hour)

Analysis Criteria	Discussion
Comparison of the maximum, and 95th and 75th percentiles to acute thresholds	1-hour: At the Worker Camp, only the maximum predicted 1-hour concentration of cobalt of 0.30 µg/m^3 exceeded the 1-hour threshold of 0.2 µg/m^3. The 95th (0.09 µg/m^3) and 75th (0.01 µg/m^3) percentiles did not exceed the acute threshold. At the MPOI, only the maximum predicted 1-hour concentration of cobalt of 0.45 µg/m^3 exceeded the 1-hour threshold of 0.2 µg/m^3. The 95th (0.19 µg/m^3) and 75th (0.02 µg/m^3) percentiles did not exceed the acute threshold. 24-hour: At the Worker Camp, only the maximum predicted 24-hour concentration of cobalt of 0.14 µg/m^3 exceeded the 24-hour threshold of 0.1 µg/m^3. The 95th (0.064 µg/m^3) and 75th (0.018 µg/m^3) percentiles did not exceed the 24-hour threshold. At the MPOI, the maximum and 95th percentile predicted 24-hour concentrations of cobalt of 0.25 and 0.14 µg/m^3, respectively, exceeded the 24-hour threshold of 0.1 µg/m^3. The 75th percentile of 0.038 µg/m^3 did not exceed the 24-hour threshold.
Frequency of exceedance	1-hour: There were 34 exceedances of the 1-hour threshold for cobalt at the Worker Camp (approximately 0.4% of the maximum year), and 414 exceedances at the MPOI (approximately 4.7% of the maximum year). 24-hour: There were 8 exceedances at the Worker Camp (approximately 2.2% of the maximum year) and 31 exceedances at the MPOI (approximately 8.5% of the maximum year). These exceedances represent the maximum hours during the highest emissions year out of the 18 years of the operations phase of the NICO Project. Predicted emissions for the lower emission years would be expected to result in fewer exceedances.
Conservatism and uncertainty in air predictions	The predictive air modelling assumed that the NICO Project and all existing and approved developments operate continuously at their maximum design capacity at the same time. In reality, the operational life of each development will be staggered over time. Additionally, the predictive air modelling results were provided for the year with the highest predicted emissions out of the 18 years of the operations phase of the NICO Project; predicted emissions for the other 17 years of the operations phase would be expected to result in fewer exceedances. The predictive air modelling considered that cobalt is adhered to dust (or TSP), which is generated by the NICO Project through processing and road dust during the operations phase. Most of the TSP generated during the operations phase is due to road dust rather than processing. The predictive air modelling assumed that dust suppression would only occur during the summer period (i.e., May 1 to September 30) and that fugitive dust generation is possible during the winter period (i.e., October 1 to April 30) despite frozen ground conditions and/or snow-covered roads. As a result, much higher concentrations of cobalt were predicted for the winter period compared to the summer period. It is anticipated that road dust would be negligible during the winter period due to snow cover over the roads and the ground being frozen; thus, the predicted concentrations of cobalt used in the risk assessment are associated with a high degree of conservatism.

Table 4.3-7: Magnitude of Effects Assessment for Cobalt (1-hour and 24-hour) (continued)

Analysis Criteria	Discussion
Conservatism in the acute threshold	The Texas Commission on Environmental Quality 1-hour threshold of 0.2 µg/m^3 is based on a health endpoint, but supporting documentation is not available. The OMOE 24-hour threshold of 0.1 µg/m^3 is based on a health endpoint, but supporting documentation is not available.
Potential acute health effects	No supporting documentation on the basis of the 1-hour TCEQ or OMOE 24-hour thresholds was available and no other jurisdictions provided information on acute health effects. Potential acute health effects described by the Hazardous Substances Data Bank such as nausea, abdominal pains, loss of appetite, cough, and a deterioration of the sense of smell. Potential chronic effects included chronic bronchitis, early fibrotic changes, occupational asthma, and obstructive lung disease.
Magnitude of effect	The predicted peak 1-hour and 24-hour exposure concentrations for cobalt exceeded the selected air thresholds (i.e., AAAQO and OMOE, respectively). Due to the infrequency of exceedance at these locations, the conservatism in the air modelling, and that only the maximum concentrations and none of the percentiles exceeded the thresholds, the magnitude of effect for cobalt at these locations is considered to be negligible.

MPOI = Maximum Point of Impingement; TSP = total suspended solids; µg/m^3 = microgram per cubic metre

4.4 Chronic Air Quality Risk Assessment

The assessment of chemicals that may pose a long-term or chronic health risk was completed in 2 steps: Chronic Air Quality Risk Assessment (Section 4.4) and Multi-Media Risk Assessment (Section 4.6). Those chemicals that are emitted from the NICO Project that may persist in the environment (i.e., metals, semi-volatile compounds, such as PAHs and dioxins/furans) and are carcinogenic by all exposure pathways were assessed in the Multi-Media Risk Assessment. Because these chemicals may be present in soil, water, and vegetation in addition to air, receptors may be exposed to the same chemical from several sources and the exposures via all sources must be considered when interpreting the potential for human health effects. Chemicals that are volatile (i.e., not expected to persist in the environment in soil, water, and vegetation) and/or those that are only carcinogenic via the inhalation pathway are evaluated in the Chronic Air Quality Risk Assessment because exposure to these chemicals would only be expected to occur via inhalation of ambient air.

4.4.1 Problem Formulation

The objective of the Chronic Air Quality Risk Assessment is to evaluate substances potentially emitted from the NICO Project that may pose a health outcome following a long-term or chronic exposure duration (e.g., many years to lifetime) by identified receptors.

4.4.1.1 Receptor Locations

Chronic health effects associated with changes in air quality were assessed on a regional and local basis. Locations where people are known or anticipated to spend their time were identified within the LSA (worker camp and MPOI) and RSA (community/culturally significant locations).

Potential human health effects related to long-term inhalation exposure for the NICO Project were evaluated for the seven receptor locations identified for the Acute Air Quality Risk Assessment (Section 4.3).

4.4.1.2 Identification of Chemicals of Potential Concern

Chemicals that may change in concentration as a result of the NICO Project were identified based upon the NICO Project components and activities that result in emissions to air as described in the Acute Air Quality Risk Assessment (Section 4.3).

Chemical concentrations (annual averages for the year with the highest predicted emissions during the operations phase) were predicted for the NICO Project for criteria air contaminants, greenhouse gases, metals, PAHs, VOCs, and dioxins and furans for all of the receptor locations.

4.4.2 Chemical Screening Process

The chronic health-based thresholds for criteria air contaminants, greenhouse gases, metals, PAHs, VOCs, and dioxins and furans that were selected for chemical screening (Table A.5 in Appendix A) were the most protective of the available thresholds from the following agencies:

- Alberta Environment (AENV 2009);
- Agency of Toxic Substances and Disease Registry (ATSDR 2009, internet site);
- California Office of Environmental Health Hazard Assessment (OEHHA 2008, internet site);
- Canadian Council of Minsters of the Environment (CCME 2008);

- Government of the Northwest Territories (GNWT 2005, internet site);
- Texas Commission on Environmental Quality (TCEQ 2009, internet site);
- United States Environmental Protection Agency (US EPA 2010, internet site); and
- World Health Organization (WHO 2000; WHO 2005).

The chronic thresholds were based upon conservative levels of protection. For non-carcinogenic substances, a target hazard quotient of 1 was used, and for carcinogenic substances, a target cancer risk of one in one million (or $1x10^{-6}$) was used.

The maximum annual average predicted concentrations of substances in air at all receptor locations (i.e., all communities, culturally significant areas, on-site worker camp, and MPOI) for the Project Case (Table A.6 in Appendix A) were compared to the chronic air thresholds. For those chemicals for which individual thresholds are not available, the same groupings as those identified for the Acute Air Quality Risk Assessment were used.

If the predicted concentrations of Project Case substances were greater than the chronic air thresholds and greater than baseline concentrations plus 10%, they were defined as COPCs and were retained for further consideration. The following COPCs were identified for the chronic averaging period: acrolein, aldehydes, benzo(a)pyrene, arsenic, cadmium, cobalt, and manganese. Note that acrolein and aldehydes are chemical groups as identified in the Acute Air Quality Risk Assessment. The assessment of benzo(a)pyrene is described further below.

4.4.3 Toxicity Assessment

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.

4.4.3.1 Contaminant Classification

First, the carcinogenicity classification of the chemicals that are greater than the chronic air thresholds and greater than baseline concentrations plus 10% was determined.

Regulatory agencies, such as the MOE, classify contaminants based on their mode(s) of action. For substances exhibiting a threshold for toxicity (i.e., non-carcinogens), an acceptable level of exposure at or below which no adverse effects are anticipated is established. Typically, this threshold level is represented by a reference concentration (RfC) for the inhalation pathway. For non-threshold-acting chemicals (i.e., carcinogens), any level of exposure may theoretically pose a potential risk, and a unit risk is used to predict risks from estimated exposures.

Classification systems have been developed based on the carcinogenic properties of chemicals, including those from Health Canada, US EPA, and the International Agency for Cancer Research (IARC) (Table 4.4-1).

Table 4.4-1: Classification Systems for Carcinogenic Substances

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

[a] Health Canada Canadian Environmental Protection Act (Health Canada 1999).

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

[c] US EPA Risk Assessment Guidance for Superfund (RAGS) (US EPA 1989).

The carcinogenic classifications of the COPCs identified in the chronic chemical screening (Table 4.4-2) indicated that benzo(a)pyrene and arsenic are carcinogenic via the ingestion and inhalation exposure routes. Therefore, benzo(a)pyrene and arsenic were assessed within the Multi-Media Risk Assessment. The Chronic Air Quality Risk Assessment focussed on NO_2, acrolein, aldehydes, cadmium, cobalt, and manganese given that these chemicals are either non-carcinogenic or carcinogenic via the inhalation route only.

Table 4.4-2: Carcinogenic Classifications of the Chemicals of Potential Concerns in the Chronic Chemical Screening

Compound	Health Canada Classification	IARC Classification	US EPA Classification	Exposure Routes	Assessed in the Chronic Air RA?
Acrolein	ND	ND	Group C	N/A	Yes
Aldehydes	ND	Group 2B	Group B2	Inhalation	Yes
Benzo(a)pyrene	Group II	ND	Group B2	Ingestion, Inhalation	No[a]
Arsenic	Group I	Group 1	Group A	Ingestion, Inhalation	No[a]
Cadmium	Group II	Group 1	Group B1	Inhalation	Yes
Cobalt	ND	Group 2B	ND	Inhalation	Yes[b]
Manganese	ND	ND	Group D	N/A	Yes

[a] Those chemicals that were identified as carcinogenic via both ingestion and inhalation were deferred to the Multi-Media Risk Assessment (Section 4.6).

[b] Cobalt was not evaluated as a carcinogen because TRVs are not available.

ND = not documented; N/A = not applicable; RA = risk assessment

4.4.3.2 Dose-Response Assessment

The most protective of the available RfCs (Table 4.4-3) or unit risks (Table 4.4-4) were selected for use in the Chronic Air Quality Risk Assessment. The RfCs and unit risks were compiled only for the COPCs retained for further assessment in the Chronic Air Quality Risk Assessment: acrolein, aldehydes, cadmium, cobalt, and manganese. The TRVs for the COPCs retained for the Multi-Media Risk Assessment (i.e., benzo(a)pyrene and arsenic) are provided in Section 4.6.

Table 4.4-3: 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]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Volatile Organic Compounds						
Acrolein	n/a	**0.02**	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 per day and 5 days per week for 13 weeks. An uncertainty factor of 1000 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).
Aldehydes (surrogate: acetaldehyde)	n/a	**9**	n/a	n/a	n/a	The IRIS RfC is based on 2 subchronic inhalation studies in rats. The critical effect was degeneration of the olfactory epithelium. An uncertainty factor of 1000 was applied; 10 for sensitive individuals, 10 for extrapolation from animals to humans and for incompleteness of the database, and 10 for subchronic to chronic extrapolation.
Metals						
Cadmium	n/a	n/a	**0.01**	n/a	n/a	The ATSDR MRL is based on the 95% lower confidence limit of the urinary cadmium level estimated from meta-analysis of environmental exposure data that was associated with 10% excess risk of low molecular weight proteinuria. The associated air concentration was estimated using biokinetic models and assumed a dietary intake of 0.3 µg/kg/day. An uncertainty factor of 3 for human variability and a modifying factor of 3 were used to develop the final MRL.
Cobalt	n/a	n/a	**0.1**	0.5	n/a	The ATSDR MRL for stable cobalt is based on decreased respiratory function in exposed workers. The RIVM TCA is based on the LOAEC of 0.05 mg/m^3 for interstitial lung disease. An uncertainty factor of 100 was used (10 for the extrapolation from a LOAEC and 10 for human variability).

Table 4.4-3: Reference Concentrations for Chemicals of Potential Concern Evaluated in the Chronic Air Quality Risk Assessment – Non-Carcinogens (continued)

Parameter	Reference Concentration [µg/m^3]					Toxicological Endpoints and Derivations
	Health Canada[a]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Manganese	n/a	**0.05**	0.3	n/a	n/a	The IRIS RfC for manganese is derived from a LOAEL of 0.05 mg/m^3, which was based on effects on neurobehavioural function. The LOAEL was derived from an occupational-lifetime integrated respirable dust concentration of manganese dioxide dust for workers in a Belgian alkaline battery plant. It was based on an 8-hour TWA occupational exposure multiplied by individual work history in years. The geometric mean concentration was divided by the average duration of exposure to obtain a LOAEL TWA of 0.15 mg/m^3. The LOAEL was then adjusted to continuous exposure from 5 days per week. An uncertainty factor of 1000 was applied (10 to protect sensitive individuals, 10 for the use of a LOAEL, and 10 for database limitations for the less than chronic periods of exposure, the lack of developmental data, and the potential but unquantified differences in the toxicity of the different forms of manganese). The ATSDR MRL is based on the same effects and data as the US EPA IRIS RfC. The dichotomus models in the EPA Benchmark Dose software were fit for the incidence data. The MRL was obtained by adjusting the point of departure to a continuous exposure and applying an uncertainty factor of 100 (10 for human variability, 10 for limitations and uncertainties in the database including the lack of epidemiological for humans chronically exposed to soluble forms of manganese, and the concern that the general population could be exposed to more soluble forms of manganese compared to workers exposed in the principle/supporting studies).

[a] Health Canada (2009).

[b] United States Environmental Protection Agency Integrated Risk Information System (US EPA 2010, internet site).

[c] Agency for Toxic Substances and Disease Registry (ATSDR 2010, internet site).

[d] National Institute of Public Health and the Environment (RIVM 2001).

[e] Source of RfC is explained in toxicological endpoint section, as RfCs were available from other jurisdictions.

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).

LOAEC = Lowest Observed Adverse Effect Concentration; LOAEL =lowest observed adverse effect level; MRL = Minimal Risk Level; RfC = reference concentration; ppm = parts per million; n/a = Not available; mg/m^3 = milligram per cubic metre; µg/kg/day = microgram per kilogram per day; µg/m^3 = microgram per cubic metre

Table 4.4-4: Inhalation Unit Risks for Chemicals of Potential Concern Evaluated in the Chronic Air Quality Risk Assessment – Carcinogens

Parameter	Inhalation Unit Risks [µg/m^3]$^{-1}$					Toxicological Endpoints and Derivations
	Health Canada[a]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Volatile Organic Compounds						
Aldehydes (surrogate: acetaldehyde)	n/a	**0.0000022**	n/a	n/a	n/a	The IRIS UR is based on the incidence of nasal squamous cell carcinomas or adenocarcinomas in rats exposed to acetaldehyde by inhalation. IRIS used a multistage modelling approach with linearized low-dose extrapolation to derive the UR.
Metals						
Cadmium	**0.0098**	0.0018	n/a	n/a	n/a	The Health Canada UR is based on incidence of lung cancer in rats exposed for 23 hours per day and 7 days per week for a duration of 18 months (necropsied 13 months after exposure). The TC_{05} was 0.0029 mg/m³ (0.0051 mg/m³ adjusted for continuous exposure, standard lifetime, and difference in inhalation rate and body weight of rats and humans). The IRIS UR is based on the incidence of lung, trachea, and bronchus cancer deaths of occupationally exposed human males (cadmium smelter workers). The cohort consisted of 602 white males who had been employed in production work for a minimum of 6 months between the years 1940 to 1969. The cohort was followed to the end of 1978. This study is considered to supply limited evidence of human carcinogenicity due to the fact that the Standard Mortality Ratios observed were low and there is a lack of clear evidence of a causal relationship when examining correlation with cadmium exposure only.

[a] Health Canada (2009).

[b] United States Environmental Protection Agency Integrated Risk Information System (US EPA 2010, internet site).

[c] Agency for Toxic Substances and Disease Registry (ATSDR 2010, internet site).

[d] National Institute of Public Health and the Environment (RIVM 2001).

[e] Source of RfC is explained in toxicological endpoint section, as RfCs were available from other jurisdictions.

Note: Bolded Unit Risks were used in the risk assessment. Unless otherwise stated, the most conservative of the available URs was chosen (i.e., the highest).

UR = unit risks; n/a = Not available; mg/m^3 = milligram per cubic metre; µg/m^3 = microgram per cubic metre.

4.4.3.3 Chemical Mixtures

For chemicals with similar modes of action and target organs (Table 4.4-5), the HQs or incremental lifetime cancer risks (ILCRs) were summed.

Table 4.4-5: Potential Additive Interactions of the Chemicals of Potential Concern in the Chronic Air Quality Risk Assessment

Chemicals of Potential Concern		Target Organ[a]	Effects
Non-carcinogens	Acrolein and aldehydes	Nose	Nasal lesions
Carcinogens	Aldehydes	Respiratory tract	Respiratory tract tumours

[a] Based on information provided in US EPA (2010, internet site).

4.4.4 Exposure Assessment

For the Chronic Air Quality Risk Assessment, the annual average concentrations were predicted for each COPC and receptor location. These predicted concentrations are the exposure point concentrations used to estimate the exposure doses for each receptor using the equation below.

$$Exp_{inh} = \frac{C_{air} \times RAF_{inh} \times IR \times ET \times EF \times ED}{BW \times AT \times CF_1 \times CF_2}$$

Where:

Exp_{inh} = exposure dose due to inhalation of COPC in air (mg/kg-day)
C_{air} = annual average concentration of COPC in air (mg/m^3)
RAF_{inh} = inhalation relative absorption factor (unitless)
IR = inhalation rate (m^3/hr)
ET = exposure time (hr/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (yr)
CF_1 = unit correction factor (24 hr/d)
CF_2 = unit correction factor (365 d/yr)

The Community Resident was considered to spend 30 days per year at the MPOI location, and the Worker was considered to spend 250 days per year at the MPOI location or 250 days per year at the on-site Worker Camp. Therefore, the results for the MPOI location provide a maximum exposure scenario given that the air quality predictions are highest at the MPOI. The Resident/Worker was not evaluated separately given that exposure for this receptor would be bounded by that of the Worker at the Worker Camp location.

4.4.5 Risk Characterization

As a worst-case assessment, exposure doses and HQs or ILCRs were calculated for each receptor location that had predicted annual average concentrations exceeding the chronic air thresholds (i.e., MPOI, worker camp). All predicted HQs were less than the target HQ of 1 and all predicted ILCRs were less than the target ILCR of 1E-05 for community residents and workers.

Several conservative assumptions have been incorporated into the Chronic Air Quality Risk Assessment. First, the annual average COPC concentrations were calculated for the one year out of the 18 years of the operations phase that had the highest predicted emissions from the NICO Project. The annual average concentrations from this one worst year were then assumed to be present throughout the receptor's lifespan (i.e., 75 years for residents and 55 years for workers [i.e., adult lifestage only]). Both the NICO Project and other current projects were considered to be operating concurrently. Overall, chronic health effects due to emissions from the NICO Project are considered to be negligible for the human receptors assessed.

The HQs were calculated for each non-carcinogenic (Table 4.4-6) COPC and ILCRs were calculated for each carcinogenic (Table 4.4-7) COPC for the inhalation pathway for the Community Resident and Worker.

Table 4.4-6: Hazard Quotients for Non-Carcinogenic Chemicals of Potential Concern

COPC	Community Resident		Worker	
	MPOI	Worker Camp	MPOI	Worker Camp
Baseline Case				
Acrolein	0.000009	NC	0.00006	NC
Aldehydes	0.00000007	NC	0.0000004	NC
Cadmium	0.0001	NC	0.0001	NC
Cobalt	0.00000007	0.00000004	0.0000004	0.0000003
Manganese	0.0000005	NC	0.000003	NC
Project Case				
Acrolein	0.1	NC	0.7	NC
Aldehydes	0.004	NC	0.02	NC
Cadmium	0.008	NC	0.05	NC
Cobalt	0.02	0.01	0.1	0.07
Manganese	0.1	NC	0.8	NC

COPC = chemicals of potential concern; MPOI = Maximum Point of Impingement; NC = not calculated.

Table 4.4-7: Incremental Lifetime Cancer Risks for Carcinogenic Chemicals of Potential Concern

COPC	Community Resident	Worker
	MPOI	MPOI
Baseline Case		
Aldehydes	1E-12	8E-12
Project Case		
Aldehydes	7E-08	4E-07

COPC = chemicals of potential concern; MPOI = Maximum Point of Impingement

Given that all calculated HQs were less than 1 and ILCRs were less than 1E-05, estimated risks were acceptable for the Community Resident and Worker receptors. Given that the estimated risks for the Worker are also applicable to the Resident/Worker, estimated risks for this receptor are also acceptable.

4.4.5.1 Risk Characterization of Chemical Mixtures

The estimated risks for COPCs that have similar target organs, effects and mechanisms of action can be added together to determine a total risk for similar toxicological effects (Health Canada 2007). Likewise, estimated risks should be summed for carcinogenic COPCs with the same target organ and form of cancer. As shown in Section 4.4.3.3 above, acrolein and aldehydes both target nasal lesions as a non-carcinogenic effect.

The sum of the HQs for acrolein and aldehydes for all receptors were less than 1 (Table 4.4-8), indicating that chemical mixtures also have an acceptable risk on these receptors at all locations. As described above, the probability of chronic health effects is considered to be negligible considering the conservative assumptions incorporated into the air modelling.

Table 4.4-8: Hazard Quotients for Chemical Mixtures

Parameter	Maximum Point of Impingement
Community Resident (Project Case)	
Aldehydes + Acrolein	0.1
Worker (Project Case)	
Aldehydes + Acrolein	0.7

4.4.6 Magnitude of Effects Assessment

Given that none of the identified COPCs resulted in HQs greater than 1, a magnitude of effects assessment was not carried out in the Chronic Air Quality Risk Assessment. All HQs were less than 1 and no chronic health effects were estimated for the COPCs addressed in this section of the HHRA.

Please note that chronic health effects due to arsenic and carcinogenic PAHs have been addressed in the Multi-Media Risk Assessment (Section 4.6).

4.5 Particulate Matter Risk Assessment

This section of the HHRA describes the assessment of particulate matter (PM_{10} and $PM_{2.5}$) with respect to potential health effects.

4.5.1 Approaches to Particulate Matter Risk Assessment

Many epidemiological studies have been conducted over the past 20 years to identify the relationship between health effects and particulate matter. 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 substantial controversy regarding what specifically the relationship is. 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.

Two approaches to evaluating the potential health effects of exposure to particulate matter are described in this assessment. Each of these approaches was applied to the modeled data and the results are summarized herein.

4.5.1.1 Comparison of Predicted Concentrations with Regulatory Guidelines and Reference Levels

Predicted concentrations for the Baseline Case and Project Case were compared with available regulatory standards or objectives and with the reference levels for $PM_{2.5}$ and PM_{10} (Tables 4.5-1 and 4.5-2, respectively). Predicted 24-hour $PM_{2.5}$ and PM_{10} concentrations are less than their respective Canada-Wide Standards (i.e., 30 µg/m^3 and 50 µg/m^3, respectively) for all communities and assessment cases with the exception of the MPOI and the on-site worker camp during the Project Case (i.e., operations phase).

Predicted concentrations (peak and 98th percentile) for the MPOI and the on-site worker camp were greater than the $PM_{2.5}$ reference level of 15 µg/m^3 and the PM_{10} reference level of 25 µg/m^3. Therefore, further quantitative assessment of $PM_{2.5}$ and PM_{10} was carried out for these locations.

Table 4.5-1: Comparison of 24-hour Peak $PM_{2.5}$ Concentrations to the Canada-Wide Standard and Reference Levels

Receptor Location	24-hour Peak $PM_{2.5}$ Concentration [µg/m^3]	
	Baseline Case	Project Case
Gamètì	2.3	2.4
Whatì	2.3	2.6
Hislop Lake	2.2	6.2
Marian River	2.2	6.4
Bea Lake	2.2	6.9
Worker Camp	2.3	**52.6**
Worker Camp (98th percentile)	-	**32.5**
MPOI	2.3	**80.3**
MPOI (98th percentile)	-	**52.2**
Canada-Wide Standard[a]	30	
Reference Level[b]	15	

[a] CCME (2000).

[b] Health Canada and Environment Canada (1999).

Note: Bold text indicates locations at which $PM_{2.5}$ concentrations are predicted to be greater than the reference level.

MPOI = Maximum Point of Impingement; µg/m3 = microgram per cubic metre

Table 4.5-2: Comparison of 24-hour Peak PM_{10} Concentrations to the Canada-Wide Standard and Reference Levels

Receptor Location	24-hour Peak PM_{10} Concentration [µg/m^3]	
	Baseline Case	Project Case
Gamètì	2.3	2.6
Whatì	2.2	2.4
Hislop Lake	2.2	15.4
Marian River	2.2	18.6
Bea Lake	2.2	13.9
Worker Camp	2.2	**252.4**
Worker Camp (98th percentile)	-	**163.6**
MPOI	2.3	**500.1**
MPOI (98th percentile)	-	**317.4**
Canada-Wide Standard[a]	50	
Reference Level[b]	25	

[a] CCME (2000).

[b] Health Canada and Environment Canada (1999).

Note: Bold text indicates locations at which PM_{10} concentrations are predicted to be greater than the reference level.
MPOI = Maximum Point of Impingement; µg/m3 = microgram per cubic metre

4.5.1.2 SUM15 and SUM25 Approach

This method for evaluating exposure to $PM_{2.5}$ and PM_{10} is based on a statistical relationship reported by the Federal-Provincial Working Group on Air Quality Objectives and Guidelines (the Working Group) (Health Canada and Environment Canada 1999). The Working Group reviewed a number of epidemiological studies and determined a reference level of 15 µg/m^3 for $PM_{2.5}$, which was considered to be its Lowest Observed Adverse Effect Level (LOAEL). Concentrations greater than reference levels may result in statistically significant occurrences of adverse human health effects (Health Canada and Environment Canada 1999). The reference level for PM_{10} was determined to be 25 µg/m^3.

To quantify the potential health effects resulting from predicted concentrations that exceed the reference levels, the statistical relationship presented in the Working Group's report (Health Canada and Environment Canada 1999) was used to calculate the potential increase in mortality and hospital admissions due to respiratory and cardiac illnesses (Health Canada and Environment Canada 1999).

Health Canada and Environment Canada (Health Canada and Environment Canada 1999) indicate that each µg/m^3 increase in $PM_{2.5}$ greater than the reference level (i.e., greater than 15 µg/m^3) could result in the following:

- an increase of 0.026 deaths per million population per day;
- an increase of 0.0118 hospitalizations for respiratory related causes per million population per day; and
- an increase of 0.010 hospitalizations for cardiac related causes per million population per day.

These statistical relationships are based on epidemiological studies conducted in United States cities with populations much greater than communities within the RSA and with $PM_{2.5}$ concentrations much greater than

those predicted for the Application Case. Therefore, it is uncertain whether the statistical relationships determined from these epidemiological studies are applicable to the communities evaluated in the assessment. Nevertheless, this relationship was used to evaluate potential impacts to human health as a result of increases in particulate matter due to the NICO Project.

Additionally, Health Canada and Environment Canada (Health Canada and Environment Canada 1999) indicate that each µg/m^3 increase in PM_{10} greater than the reference level (i.e., greater than 25 µg/m^3) could result in the following:

- an increase of 0.014 deaths per million population per day.

The Working Group recommends calculating a parameter called SUM15 to associate potential health outcomes with ambient $PM_{2.5}$ concentrations and SUM25 for ambient PM_{10} concentrations. SUM15 is calculated as the sum of the daily $PM_{2.5}$ concentrations above 15 µg/m³ (i.e., $[PM_{2.5}]_{daily}$) throughout a calendar year, and has units of micrograms per cubic metre-days ([µg/m³]-days) (see equation below). For example, a daily concentration of 18 µg/m³ would be counted as 3 µg/m³ in the SUM15 calculation, whereas a daily average of 13 µg/m³ would not be taken into account in the SUM15 calculation. SUM15 includes all of the daily averages greater than 15 µg/m³ in a calendar year. The SUM25 is calculated similarly, except uses the daily averages of PM_{10} concentrations greater than 25 µg/m^3.

$$\text{SUM15 } [(\mu g/m^3)\text{-days}] = \sum ([PM_{2.5}]_{daily} - 15)$$

$$\text{SUM25 } [(\mu g/m^3)\text{-days}] = \sum ([PM_{10}]_{daily} - 25)$$

The annual SUM15 and SUM25 values were calculated for the locations that had exceedances of the reference levels.

4.5.2 Results of Particulate Matter Risk Assessment

4.5.2.1 Conventional Risk Assessment Approach

As shown in Section 4.5.1 above, the predicted 24-hour concentrations of $PM_{2.5}$ and PM_{10} at the MPOI and the Worker Camp exceeded the reference levels for the operations phase.

4.5.2.1.1 Exposure Assessment

The chronic exposure dose was calculated for the predicted annual average concentrations of PM_{10} (annual average concentrations were not calculated for $PM_{2.5}$) at the MPOI and the Worker Camp using the following equation (introduced in Section 4.4.5):

$$Exp_{inh} = \frac{C_{air} \times RAF_{inh} \times IR \times ET \times EF \times ED}{BW \times AT \times CF_1 \times CF_2}$$

Where:

Exp_{inh} = exposure dose due to inhalation of chemicals in air (mg/kg-day)
C_{air} = annual average concentration of COPC in air (mg/m^3)
RAF_{inh} = inhalation relative absorption factor (unitless)
IR = inhalation rate (m^3/hr)
ET = exposure time (hr/d)
EF = exposure frequency (d/yr)

ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (yr)
CF_1 = unit correction factor (24 hr/d)
CF_2 = unit correction factor (365 d/yr)

The Community Resident was considered to spend 24 hours per day for 30 days per year at the MPOI location, and the Worker was considered to spend 12 hours per day for 250 days per year at the MPOI location or 24 hours per day at the on-site Worker Camp. The exposure doses were calculated for both receptors at both locations (Table 4.5-3).

Table 4.5-3: Exposure Doses for the Community Resident and Worker Receptors at the Maximum Point of Impingement and Worker Camp Locations for PM_{10}

Receptor Location	Annual Average Concentrations ($\mu g/m^3$)		Exposure Doses (mg/kg/day)		
			Community Resident	Worker	
	MPOI	Worker Camp	MPOI	MPOI	Worker Camp
Exp_{inh} (PM_{10})	50.64	28.35	0.00235	0.0143	0.0799

MPOI = Maximum Point of Impingement; µg/m3 = microgram per cubic metre; mg/kg/day = milligram per kilogram per day

4.5.2.1.2 Toxicity Assessment

WHO (2005) provides an air quality guideline for PM_{10} of 20 $\mu g/m^3$, which was derived from the $PM_{2.5}$ value of 10 $\mu g/m^3$ and using a $PM_{2.5}/PM_{10}$ ratio of 0.5. The $PM_{2.5}$ value of 10 $\mu g/m^3$ is considered to be the mean annual average concentration below which health effects are not likely to occur for the general population, including sensitive subpopulations such as asthmatics and the very young and very old. Health effects include cardiopulmonary and lung cancer mortality. The air quality guideline of 20 $\mu g/m^3$ was converted to an inhalation reference dose of mg/kg-day to be directly comparable with the estimated daily intakes calculated in the exposure assessment using the receptor-specific inhalation rates and body weights.

4.5.2.1.3 Risk Characterization

Chronic or long-term health effects were evaluated by calculating HQs as described in Section 4.4.5.

The HQs were calculated for the Community Resident at the MPOI and for the Worker at the MPOI and Worker Camp (Table 4.5-4).

Table 4.5-4: Hazard Quotients for the Community Resident and Worker Receptors at the Maximum Point of Impingement and Worker Camp Locations for PM_{10}

Receptor Location	Community Resident	Worker	
	MPOI	MPOI	Worker Camp
HQ (PM_{10})	0.2	0.9	1.0

MPOI = Maximum Point of Impingement ; HQ = hazard quotient

The Worker receptor had a calculated HQ of approximately 1 at the Worker Camp, indicating that chronic exposure to the annual average PM_{10} concentration at the most probable location would not be expected to

result in unacceptable health risks. For the less likely scenario of the Worker spending 12 hours per day at the MPOI, an HQ of 0.9 was calculated. It should be noted that the TRV used to calculate the HQ was based upon the protection of sensitive individuals, and it is likely that worker receptors will be healthy adults and this HQ would likely be overestimated. Therefore, the health risk associated with chronic exposure to PM_{10} was considered to be negligible.

The predicted particulate matter concentrations were also assessed using the SUM15 and SUM25 approach, which is detailed below.

4.5.2.2 Results using the SUM15 and SUM25 Approach

The results using the SUM15 and SUM25 approaches are provided below. First, the results of the SUM15 ($PM_{2.5}$) assessment are provided, followed by those of the SUM25 (PM_{10}) assessment.

4.5.2.2.1 SUM15 Results

The SUM15 values for the predicted worst-case year for the Worker Camp and the MPOI for the Baseline Case and Project Case, as well as the current population of each community, are presented in Table 4.5-5. The SUM15 values could not be calculated for the remaining locations because concentrations were less than the reference levels at these locations for both assessment cases.

Further evaluation (i.e., estimation of mortality and hospital admissions) for $PM_{2.5}$ was done for the Worker Camp and the NICO Project Boundary as $PM_{2.5}$ concentrations exceeded the reference level at these locations.

Table 4.5-5: Community Populations and SUM15 Values for the Baseline and Project Cases

Receptor Location	Current Population	Baseline Case SUM15	Project Case SUM15
		($\mu g/m^3$)-days	
Camp	175[a]	116.3	491.7
MPOI	2957[b]	185.7	1451.2

[a] Maximum number of workers in any given year as provided in Project Description, Section 3.11.2 of the DAR.

[b] 2009 population data for nearby communities (Whatì = 497, Gamètì = 295, Wekweètì = 137, and Behchokǫ̀ = 2028).

MPOI = Maximum Point of Impingement; $\mu g/m^3$ = microgram per cubic metre

By applying the SUM15 values with the statistical relationship presented in the Working Group's report, as well as the number of people living within each community, possible health outcomes (i.e., hospitalizations and mortalities) can be estimated according to specific equations (Table 4.5-6). Current population estimates for communities within the study area were taken from Statistics Canada (Statistics Canada 2006) unless otherwise noted.

Table 4.5-6: Equations to Estimate Mortality and Hospital Admissions Due to $PM_{2.5}$ Exposure

Endpoint	Equation
$PM_{2.5}$	
Mortality (deaths per year)	$\text{SUM15} \times \frac{0.026}{10^6} \times \text{population}$
Respiratory hospital admissions (RHA per year)	$\text{SUM15} \times \frac{0.0118}{10^6} \times \text{population}$
Cardiac hospital admissions (CHA per year)	$\text{SUM15} \times \frac{0.010}{10^6} \times \text{population}$

Using these equations, the estimated mortality and hospital admissions were calculated and are presented in Table 4.5-7 below for each of the assessment cases. Given that all values were less than one, less than one case of mortality or hospital admissions are expected due to the NICO Project.

Table 4.5-7: Estimates of Mortality and Hospital Admissions due to $PM_{2.5}$ Exposure

Location	Deaths per Year		Hospitalizations for Respiratory Illness		Hospitalizations for Cardiac Illness per Year	
	Baseline Case	Project Case	Baseline Case	Project Case	Baseline Case	Project Case
Camp	0.0005	0.0022	0.0002	0.0010	0.0002	0.0009
MPOI	0.0143	0.1116	0.0065	0.0506	0.0055	0.0429

MPOI = Maximum Point of Impingement

4.5.2.2.2 SUM25 Results

The SUM25 values for the predicted worst-case year for the Worker Camp and the MPOI for the Baseline Case and Project Case, as well as the current population of each community, are presented in Table 4.5-8. The SUM25 values could not be calculated for the remaining locations because concentrations were less than the reference levels at these locations for both assessment cases.

Further evaluation (i.e., estimation of mortality and hospital admissions) for PM_{10} was done for the Worker Camp and the Project Boundary as PM_{10} concentrations exceeded the reference level at these locations.

Table 4.5-8: Community Populations and SUM25 Values for the Baseline and Project Cases

Receptor Location	Current Population	Baseline Case SUM25	Project Case SUM25
		($\mu g/m^3$)-days	
Camp	175[a]	262.8	6 017.4
MPOI	2957[b]	341.3	14 214.4

[a] Maximum number of workers in any given year as provided in Project Description, Section 3.11.2 of the DAR.

[b] 2009 population data for nearby communities (Whatì = 497, Gamètì = 295, Wekweètì = 137, and Behchokǫ̀ = 2028).

MPOI = Maximum Point of Impingement; $\mu g/m^3$ = microgram per cubic metre

By applying the SUM15 values with the statistical relationship presented in the Working Group's report, as well as the number of people living within each community, possible health outcomes (i.e., hospitalizations and mortalities) can be estimated according to specific equations (Table 4.5-9). Current population estimates for communities within the study area were taken from Statistics Canada (Statistics Canada 2006) unless otherwise noted.

Table 4.5-9: Equations to Estimate Mortality and Hospital Admissions Due to PM_{10} Exposure

Endpoint	Equation
PM_{10}	
Mortality (deaths per year)	$\text{SUM25} \times \frac{0.014}{10^6} \times \text{population}$

Using these equations, the estimated mortality was calculated and is presented in Table 4.5-10 below for each of the assessment cases. Given that all values were less than one, less than one case of mortality is expected due to the NICO Project.

Table 4.5-10: Estimates of Mortality due to PM_{10} Exposure

Location	Deaths per Year	
	Baseline Case	Project Case
Worker Camp	0.0006	0.0147
MPOI	0.0141	0.5884

MPOI = Maximum Point of Impingement

4.6 Multi-Media Risk Assessment

The objective of the Multi-Media Risk Assessment is to evaluate substances potentially emitted from the NICO Project that may pose a health outcome following a long-term or chronic exposure duration (e.g., many years to lifetime) by identified receptors from all potentially impacted environmental media (e.g. air, water, soil, vegetation, fish, and wild game).

4.6.1 Problem Formulation

4.6.1.1 Identification of Receptors

Effects to human health were assessed on a regional and local basis. Locations where people are known or anticipated to spend their time were identified within the RSA. The RSA for human health is defined as a 94 km (east-west) by 124 km (north-south) area that encompasses existing and approved industrial emissions sources from Gamètì, Whatì, and Snare Rapids, in addition to the NICO Project.

The LSA for human health is defined as a 30 km by 30 km area in the immediate vicinity of the NICO Project where the majority of air quality effects due to the NICO Project area expected to occur. All health receptors were located outside of the LSA except for the worker camp.

Potential human health effects related to long-term multi-media exposure for the NICO Project were evaluated for the following 7 receptor locations (Figure 4.1-1):

- MPOI Location;
- Worker Camp;
- community of Gamètì;
- community of Whatì;
- culturally significant location of Hislop Lake;
- culturally significant location of Marian River; and
- culturally significant location of Bea Lake.

To represent the off-site culturally significant locations of Hislop Lake and Marian River, several individual points were identified (Figure 4.3-1) based upon the locations of cabins, camping sites, other culturally significant

areas, and locations of importance for the Tłı̨chǫ including burial sites. Out of the multiple individual points for each culturally significant location, the point with the highest predicted concentrations of substances in air was quantitatively assessed in the HHRA.

In addition, 2 communities were identified and assessed in the HHRA: Gamètì and Whatì. Proxy locations for the more distant communities of Wekweetì, Behchokǫ̀, and Yellowknife were also considered. If the predicted concentrations of substances in air at the closer communities of Gamètì and Whatì were acceptable, quantitative assessment of the 3 more distant communities was not carried out because smaller changes to air quality were predicted at these more distant locations.

4.6.1.2 Identification of Potential Exposure Pathways

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. A chemical represents a potential health risk only if it can reach receptors through an exposure pathway at a concentration that could potentially lead to adverse effects. If there is no pathway for a chemical to reach a receptor, then there cannot be a risk, regardless of the chemical concentration. All potential pathways between chemicals and people were considered.

Based upon the receptors that are likely to be on the mine site (i.e., workers) and in the LSA/RSA (i.e., community residents that may also use areas around the NICO Project site that are culturally significant), several exposure pathways were identified. The rationale for selection of exposure pathways for the multi-media risk assessment is provided in Table 4.6-1.

Table 4.6-1: Exposure Pathways Identified in the Multi-Media Risk Assessment

Exposure Pathway	Evaluated	Not Evaluated	Rationale
Inhalation of air	✓		People may be exposed to airborne chemicals released to air from the NICO Project.
Inhalation of soil dust	✓		Airborne chemicals may deposit to soil and people may inhale dry soil dust particulates.
Ingestion of surface water	✓		Community residents were assumed to obtain their drinking water source from local surface waters while using the areas around the NICO Project that are culturally significant; consequently this pathway was evaluated. Workers were assumed to obtain their drinking water from Lou Lake, which was anticipated to be unaffected by the NICO Project.
Ingestion of groundwater		✓	Impacts to aquifers used for potable water sources (i.e., drinking water wells) are considered to be unlikely. Additionally, given that receptors were not considered to obtain their drinking water from groundwater sources while using the areas around the NICO Project or while working on the NICO Project site, ingestion of groundwater was not evaluated.
Ingestion of tap water		✓	Impacts to aquifers used for potable water sources (i.e., drinking water wells) are considered to be unlikely. Additionally, given that receptors were not considered to obtain their drinking water from tap water sources while using the areas around the NICO Project or while working on the NICO Project site, ingestion of tap water was not evaluated.

Table 4.6-1: Exposure Pathways Identified in the Multi-Media Risk Assessment (continued)

Exposure Pathway	Evaluated	Not Evaluated	Rationale
Dermal contact with surface water		✓	If people swim or bathe in potentially affected waterbodies or watercourses, they would not receive significant exposures through this route relative to water ingestion.
Ingestion of fish	✓		Given that impacts to watercourses and waterbodies were predicted, ingestion of fish was considered for community residents.
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.
Ingestion of plants	✓		People may consume plants that have received airborne deposition or that have taken up chemicals from the soil. Plants include wild traditional plants (berries).
Ingestion of animals	✓		People may consume animals harvested from areas near the NICO Project. Caribou health is a significant concern raised by the Tłı̨chǫ government; therefore consumption of caribou meat was evaluated.

✓ = Pathway was evaluated in the multi-media exposure model.

Therefore, the following exposure pathways were evaluated in the Multi-Media Risk Assessment for community residents:

- inhalation of suspended dusts;
- ingestion of surface water as drinking water;
- ingestion of fish;
- ingestion of berries/plants;
- ingestion of wild game;
- incidental soil ingestion;
- inhalation of soil dust; and
- dermal contact with soil.

The following exposure pathways were evaluated in the Multi-Media Risk Assessment for on-site workers:

- inhalation of suspended dusts;
- incidental ingestion of soil;
- inhalation of soil dust; and
- dermal contact with soil.

4.6.1.3 Identification of Chemicals of Potential Concern

The chemical types that were considered in the Multi-Media Risk Assessment include only those that can persist and not degrade in all environmental media (i.e., air, water, soil, vegetation, wild game). These include metals, PAHs, and dioxins and furans.

Volatile organic compounds and acid gases were not evaluated in the Multi-Media Risk Assessment because they are only considered to be present in air. This is mainly due to the physical-chemical properties of VOCs. Since VOCs have high vapour pressures, the proportion of VOCs that exist under normal conditions in the vapour phase is far greater than that which deposits onto, or absorbs into, soil and vegetation. Consequently, VOCs will tend to remain in the vapour phase (i.e., airborne) with only a small percentage partitioning into soil and vegetation (US EPA 2006b). The primary property that determines this is Henry's Law constant, which is directly influenced by the water solubility of the compound (MacKay et al. 1992). The small percentage that does deposit typically does not persist, rapidly biodegrades and volatilizes to the atmosphere. On the basis of these physical-chemical properties, the primary pathway of exposure to VOCs is considered to be via air. Consequently, the gaseous constituents (VOCs and acid gases) were assessed through the inhalation pathway only.

Particulate matter was not explicitly considered in the Multi-Media Risk Assessment because it was previously evaluated within the Particulate Matter Risk Assessment. However, soil deposition was determined using the predicted particulate matter emissions from the Project.

A comprehensive chemical screening process was used to determine the COPC in each media (i.e., air, water, soil, and food), as discussed in the following subsections.

4.6.1.3.1 Chemical Screening Process for Chemicals in Air

The chemical screening process for airborne chemicals was described previously in the Chronic Air Quality Risk Assessment (Section 4.4) and was the same process carried out in support of the Multi-Media Risk Assessment. More specifically, as described in Section 4.4.3.1, the annual average concentrations of chemicals in air were compared to chronic air thresholds and to baseline concentrations + 10%; where a predicted concentration exceeded both of these values, the chemical was retained as a COPC for the Chronic Air Quality Risk Assessment.

The annual average concentrations of arsenic and benzo(a)pyrene in air were greater than their respective air thresholds and were retained for the Multi-Media Risk Assessment. Arsenic was identified at the MPOI, the worker camp, Marian River, Hislop Lake, and Bea Lake, and benzo(a)pyrene was identified at the MPOI only. No COPCs were identified at the communities of Gamètì and Whatì. Therefore, potential multi-media impacts to these communities as well as the more distant communities of Wekweetì, Behchokǫ̀, and Yellowknife were considered to be negligible as a result of the NICO Project.

4.6.1.3.2 Chemical Screening Process for Chemicals in Water

The screening of the chemicals potentially released into regional surface water as a result of the NICO Project was carried out for metals only. The other chemical groups considered in the air quality assessments (i.e., particulate matter and criteria air contaminants, VOCs, PAHs, and dioxins/furans) were not expected to affect surface water quality for the following reasons:

- Particulate matter: When deposited onto a surface waterbody, particulate matter eventually settles in the basin as sediment and does not affect surface water quality.
- Criteria air contaminants: Criteria air pollutants such as NO_2 and SO_2 are volatile chemicals and would be expected to remain in the vapour phase when emitted. These chemicals would not affect surface water quality.
- VOCs: Similar to criteria air contaminants, these chemicals are volatile and are expected to remain in the vapour phase when emitted and not affect surface water quality.
- PAHs: This group of chemicals are either volatile or semi-volatile compounds. The volatile compounds would be expected to remain in the vapour phase when emitted. The semi-volatile compounds have a high affinity for soil or sediment and would be expected to adhere to these media and not dissolve in the water column. Therefore, PAHs were not expected to affect surface water quality.
- Dioxins/furans: Similar to PAHs, these chemicals have a high affinity for soil or sediment and would be expected to adhere to these media and not dissolve in the water column. Therefore, dioxins/furans were not expected to affect surface water quality.

As described in the Aquatic Health Risk Assessment, SSWQOs were derived for the protection of aquatic life in the receiving waterbodies (i.e., Nico Lake and Peanut Lake) (Table 4.6-2).

Table 4.6-2: Site-Specific Water Quality Objectives for the NICO Project

Chemical of Potential Concern	CWQG for the Protection of Aquatic Life (µg/L)	Site-Specific Water Quality Objective (µg/L)		Guidelines for Canadian Drinking Water Quality, December 2010 (µg/L)
		Nico Lake	Peanut Lake	
Aluminum	100[a]	420 (dissolved aluminum)	410 (dissolved aluminum)	NV
Ammonia	Guideline based on temperature and pH	4160 (total ammonia-N/L)		NV
Antimony	NV	30 (dissolved antimony)		6.0
Arsenic	5.0	50		10
Cadmium	0.017	0.15		5.0
Chloride	NV	353,000		≤250,000 (AO)
Cobalt	NV	10		NV
Copper	2[b]	25 (dissolved copper)	22 (dissolved copper)	≤1000 (AO)
Iron	300	1500		≤300 (AO)
Lead	1[c]	7.6		10
Nitrate	13,000	133,000		45,000
Selenium	1.0	5.0 (total selenium)		10
Sulphate	NV	500,000		≤500,000 (AO)
Uranium	NV	27		20
Zinc	30	110		≤5000 (AO)

[a] Based on the guideline for a pH of ≥6.5.

[b] Based on the guideline for water hardness of 0-120 mg/L as $CaCO_3$.

[c] Based on the guideline for water hardness of 0-60 mg/L as $CaCO_3$.

NV = No guideline value; µg/L = milligram per litre

However, these SSWQOs did not take into consideration the protection of human health. Therefore, the predicted concentrations of metals in surface water were compared to the Guidelines for Canadian Drinking Water Quality (Health Canada 2010). Exceedances of arsenic and iron were predicted (Table B.1 in Appendix B). Given that the guideline for iron is an aesthetic objective and not health-based, iron was not retained as a COPC on the basis of water quality. Therefore, arsenic was retained as a COPC in surface water in the Multi-Media Risk Assessment.

Arsenic was identified at Nico, Peanut, and Burke lakes at concentrations greater than the Canadian Drinking Water Quality (CDWQ) guideline. These waterbodies may be used as a drinking water source by human receptors that may spend time around the MPOI (i.e., NICO Project Boundary). To provide a conservative assessment, the peak 95th percentile concentration of arsenic during the operations phase at Nico Lake, which is the location and time period when arsenic is predicted to peak at 44 µg/L, was selected as the exposure point concentration in the Multi-Media risk Assessment for the MPOI location.

Workers at the on-site worker camp are proposed to obtain their potable water from Lou Lake, which was considered to remain unaffected by the NICO Project and continue to meet the CDWQ guidelines. To provide a conservative assessment, the maximum measured baseline concentration of arsenic in Lou Lake of 0.8 µg/L was used in the assessment.

The water quality at the Marian River was predicted to be less than the CDWQ guideline throughout the NICO Project. Given that the water quality at Marian River bounds that of Hislop Lake and other waterbodies further downstream, water quality at Marian River was used to represent water quality at the recreational locations of Marian River, Hislop Lake, and Bea Lake. Again, the predicted peak concentration at Marian River was the peak 95th percentile during the operations phase of 1.7 µg/L and was selected as the exposure point concentration in the Multi-Media Risk Assessment for the 3 recreational locations.

The measured average baseline concentrations at each of these locations were used in the assessment to represent the Baseline Case.

The physical/chemical properties of PAHs (e.g. benzo[a]pyrene) indicate that these chemicals tend not to remain in the water column and preferentially become adsorbed to sediment; additionally, baseline measured concentrations of PAHs in surface water were less than MDLs. Therefore, predictive modeling for PAHs in water was not carried out.

4.6.1.3.3 Chemical Screening Process for Chemicals in Soil

Annual deposition rates for airborne metals and PAHs were predicted for the Baseline and Project cases. There are no regulatory guidelines or risk-based concentrations that can be directly compared to deposition rates. Thus, the deposition rates were used to predict mixed surface soil concentrations which were compared to the Canadian Soil Quality Guidelines for the protection of human health (CCME 2008). Where standards from CCME were not available, the US EPA Regional Screening Levels were used because these values are also risk-based and protective of the same direct contact pathways.

Incremental soil concentrations were calculated using protocols provided in the Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA 2006b). Specifically, the equations below were used to calculate the incremental soil concentrations of inorganic and organic chemicals, respectively.

$$ISC\ (Inorganic\ Chemicals) = (100^* (Dyd+Dyw)^*tD) / (Zs^*BD)$$

Where:
ISC = Incremental Soil Concentration (mg/kg dw)
Dyd = Dry Deposition Rate ($g/m^2/yr$; Project and chemical specific)
Dyw = Wet Deposition Rate ($g/m^2/yr$; Project and chemical specific)
tD = Deposition Time (21 yr; Project specific)
Zs = Soil Mixing Depth (0.02 m untilled land; US EPA 2005b)
BD = Bulk Density (1.5 g/cm^3; US EPA 2005b)

$$ISC\ (Organic\ Chemicals) = [(100^*(Dyd+Dyw)^*[1-exp(-Ks * tD)]/(Zs^*BD^*Ks)]$$

Where:
ISC = Incremental Soil Concentration (mg/kg dw)
Dyd = Dry Deposition Rate ($g/m^2/yr$; Project and chemical specific)
Dyw = Wet Deposition Rate ($g/m^2/yr$; Project and chemical specific)
tD = Deposition Time (21 yr; Project specific)
Zs = Soil Mixing Depth (0.02 m untilled land; US EPA 2005b)
BD = Bulk Density (1.5 g/cm^3; US EPA 2005b)
Ks = Soil Loss Constant (yr^{-1}; chemical specific [US EPA 2005b])

A detailed description of the deposition modelling is provided in Section 10.0 of the DAR. The modelled deposition rates represent a worst case scenario from any phase of the NICO Project (i.e., construction, operations, active closure, and post-closure). As the deposition rates used to predict incremental soil concentrations are a worst case scenario from any phase of the NICO Project, a deposition time of 21 years was used to be consistent with the Wildlife Health Risk Assessment. This included 1 year of construction, 18 years of operation, and 2 years of active closure. All chemicals deposited onto soil were assumed to mix within the top 0.02 m, as recommended for untilled soils (US EPA 2005b). Soil was assumed to have a bulk density of 1500 kilograms per cubic metre (US EPA 2005b). Loss due to weathering and degradation was only assumed for organic chemicals because metals are not degraded by processes such as microbial degradation and photolysis (US EPA 2005b).

The incremental soil concentrations for inorganic chemicals were added to the average baseline concentrations and the incremental soil concentrations for organic chemicals were added to the maximum baseline concentrations. The average baseline concentrations were used to predict the inorganic chemical concentrations because of the large variability in the inorganic chemical concentrations in the baseline sampling.

Predicted soil concentrations were compared first to baseline concentrations plus 10% (average baseline concentrations for metals and maximum baseline concentrations for PAHs). Comparison to a threshold of 10% above baseline concentrations was considered to represent a conservative evaluation of whether a measurable NICO Project-related impact to soil was likely to occur. Given spatial and temporal variability, field sampling variability in laboratory methods and the conservatism applied in the predictive deposition modelling, any predicted increased of less than 10% above baseline concentrations was considered unlikely to reflect a considerable change in environmental quality as a result of the NICO Project. Next, predicted soil concentrations were compared to the Canadian Soil Quality Guidelines for the protection of human health (CCME 2008). Where standards from CCME were not available, the US EPA Regional Screening Levels were used because these

values are also risk-based and protective of the same direct contact pathways. The chemicals in soil were retained as COPCs if the predicted soil concentrations exceeded baseline concentrations plus 10% and exceeded a soil guideline/screening level.

The comparison of predicted soil concentrations to baseline concentrations plus 10% and to the selected soil guideline/screening levels are provided in Table B.2 in Appendix B. The maximum predicted concentration of arsenic in soil using the deposition rates at the MPOI location was both greater than the CCME guidelines and baseline plus 10%. Additionally, cobalt and iron were identified as COPCs in soil at the MPOI and Worker Camp receptor locations.

4.6.1.3.4 Chemicals of Potential Concern in the Multi-Media Risk Assessment

There were 4 COPCs identified for the Multi-Media Risk Assessment: arsenic, cobalt, iron, and benzo(a)pyrene.

Arsenic was identified as a COPC in the following media:

- Air: predicted concentrations of arsenic in ambient air at the MPOI, worker camp, Marian River, Hislop Lake, and Bea Lake exceeded both baseline and the chronic air quality threshold.
- Water: predicted concentrations of arsenic in water at the Nico, Peanut, and Burke lakes exceeded both baseline and the chronic water quality guideline.
- Soil: the predicted concentration of arsenic in soil at the MPOI location exceeded both baseline and the soil quality guidelines.

Cobalt and iron were identified as COPCs in the following media:

- Soil: the predicted concentrations of cobalt and iron in soil at the MPOI and worker camp locations exceeded both baseline and the soil quality guidelines.

Benzo(a)pyrene was identified as a COPC in the following media:

- Air: predicted concentrations of benzo(a)pyrene in ambient air at the MPOI location exceeded both baseline and the chronic air quality threshold.

These COPCs were retained in the Multi-Media Risk Assessment considering exposure to all environmental media (i.e., air, water, soil). Also, given that benzo(a)pyrene is one chemical in a group of carcinogenic PAHs that all have the same toxicological endpoint, all of the carcinogenic PAHs were retained for assessment.

4.6.2 Exposure Assessment

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 COPC identified in the problem formulation. For the Multi-Media Risk Assessment, exposure was estimated as a daily dose of each COPC. This value is termed the EDI and is typically expressed as milligram of a chemical per kilogram of body weight per day (mg/kg-day).

The following inputs are required to calculate EDIs and are defined in the following subsections:

- Baseline Case and Project Case COPC concentrations in soil, surface water, and air;

- Baseline Case and Project Case COPC concentrations estimated using uptake factors for plants and fish;
- Exposure factors for the assessed receptors; and
- Bioavailability of the COPCs.

4.6.2.1 Estimating Tissue Concentrations in Traditional Food Sources

The EDI was calculated from site-specific concentrations of substances in each environmental medium (e.g., air, water, and soil), the amount of time a receptor spends at a location, and receptor-specific parameters, such as body weight, ingestion rates and dietary preferences. It should be noted that while COPCs were identified based upon screening chemical concentrations in air, water and soil, chemicals in these environmental media can be transferred to potential food sources including wild game, fish, and plants. Therefore, the Multi-Media Risk Assessment considered the unique diets and lifestyles of the residents of nearby communities such as Gamètì and Whatì, including their reliance on wild game, fish, and plants as food sources and the use of local traplines and hunting areas in the vicinity of the NICO Project.

With respect to wild game tissue concentrations, site-specific data were not available. Therefore, values from the literature were used. Biotransfer factors for inorganics are provided in Baes et al. (1984) and those for organics are calculated based upon an equation provided by Travis and Arms (1988) which uses the chemical-specific log octanol-water coefficient (log Kow):

$$\log BTF = - 7.6 + \log Kow$$

Uptake of COPCs into plants and fish was estimated using site-specific uptake factors and predicted concentrations in soil and surface water. The site-specific uptake factors were calculated from measured baseline data; specifically, the average measured baseline concentrations in plants, fish, soil, and surface water were used in the calculation.

$$UF = \frac{C_T}{C_M}$$

Where:
UF = Uptake Factor (unitless)
C_T = Baseline COPC concentration in tissue (plant or fish) (mg/kg dw for plants; mg/kg ww for fish)
C_M = Baseline COPC concentration in medium (soil or water) (mg/kg for soil or mg/L for surface water)

Paired soil and plant data for metals were used to derive the plant uptake factors (there were insufficient site-specific data available for PAHs; a literature value was used). Paired data was available for berries and species-specific uptake factors were calculated. Similarly, the average baseline fish data for metals from Nico Lake (northern pike muscle and liver), Peanut Lake (northern pike muscle and liver, and lake whitefish muscle and liver), and Burke Lake (northern pike muscle and liver, and lake whitefish muscle and liver) were used to calculate the site-specific fish uptake factors. These measured baseline data was obtained from the soil and vegetation chemistry (Annex I of the DAR) and aquatic baseline reports (Annex C of the DAR).

In turn, the calculated uptake factors were used to estimate predicted chemical concentrations in plants and fish by rearranging the equation above.

$$C_T = UF \times C_M$$

Where:
UF = Uptake Factor (unitless)
C_T = Predicted COPC concentration in tissue (plant or fish) (mg/kg dw for plants; mg/kg ww for fish)
C_M = Predicted COPC concentration in medium (soil or water) (mg/kg for soil or mg/L for surface water)

Using the simple uptake factor, above, to estimate plant and fish tissue concentrations based on the average predicted soil and surface water concentrations is a conservative approach. Equations 3 and 4 assume a linear relationship between media concentrations (soil or surface water) and tissue concentrations (plants or fish) and likely overestimates tissue concentrations. For example, for fish, the equation does not consider increased excretion from the fish at higher exposure concentrations. Nevertheless, this conservative approach was used in the HHRA. It should be noted that PAH data for surface water were all reported as less than MDLs; therefore, predictions of fish tissue concentrations is unreliable and was not carried out.

A summary of the meat biotransfer factors and plant and fish uptake factors is provided in Table 4.6-4 below for each chemical retained as a COPC.

Table 4.6-4: Site-Specific Uptake Factors[a] for Plants and Fish and Biotransfer Factors for Meat

Chemicals of Potential Concern	Plants[b] (ww)	Fish (ww)	Meat (ww)
Arsenic	0.0011	59.7	0.002
Cobalt	0.0046	53.9	0.02
Iron	0.0003	315	0.02
Benzo(a)anthracene	0.0039	NA	0.00832
Benzo(a)pyrene	0.0026	NA	0.0324
Benzo(b,k)fluoranthene	0.0023	NA	0.0324
Benzo(ghi)perylene	0.0012	NA	0.126
Chrysene	0.0039	NA	0.0832
Dibenz(a,h)anthracene	0.0014	NA	0.126
Indeno(1,2,3-cd)pyrene	0.0012	NA	0.126

[a] Plant UFs for PAHs were obtained from SRS (1999).

[b] Plant UFs were adjusted to wet weight using site-specific average moisture content of berries (83%).

ww = UFs in terms of wet weight; NA = not applicable given that surface water PAH data were less than MDLs and prediction of fish tissue using MDLs are unreliable

4.6.2.2 Exposure Point Concentrations

Exposure point concentrations represent the concentrations of each COPC that are present in each environmental medium at each receptor location that were considered in estimating the EDIs (Table 4.6-5 for Baseline Case and Table 4.6-6 for Project Case). Arsenic was identified as a COPC at the MPOI, Worker Camp, Marian River, Hislop Lake, and Bea Lake due to exceedances of the chronic air thresholds at these locations; arsenic was also predicted to exceed its water quality guideline at Nico, Peanut, and Burke lakes. Carcinogenic PAHs were identified as COPCs at the MPOI location only due to exceedances of the chronic air thresholds at this location. None of the nearby communities (Gamètì and Whatì) had any measurable changes predicted to air, soil, or water quality.

Table 4.6-5: Baseline Case Exposure Point Concentrations for Environmental Media

Chemicals of Potential Concern	Air (µg/m^3)	Water (mg/L)	Soil (mg/kg)	Plant (mg/kg)	Fish (mg/kg)	Meat (mg/kg)
Arsenic						
MPOI	7.02E-07	0.0066	152.13	0.09	0.392	0.30
Worker Camp	2.74E-07	0.0008	152.13	0.09	0.048	0.30
Marian River	1.34E-07	0.0006	152.13	0.09	0.036	0.30
Hislop Lake	2.06E-07	0.0006	152.13	0.09	0.036	0.30
Bea Lake	1.49E-07	0.0006	152.13	0.09	0.036	0.30
Cobalt						
MPOI	8.93E-08	0.0006	37.32	0.36	0.032	0.75
Worker Camp	5.30E-08	0.0005	37.32	0.36	0.027	0.75
Iron						
MPOI	3.23E-07	0.177	6776.40	13.37	56	136
Worker Camp	2.17E-07	0.989	6776.40	13.37	311	136
Total PAHs at MPOI						
Benzo(a)anthracene	1.12E-08	<0.000010	0.007	0.001	NC	5.90E-05
Benzo(a)pyrene	2.32E-09	<0.000010	0.191	0.001	NC	6.18E-03
Benzo(b,k)fluoranthene	2.20E-08	<0.000010	0.012	0.001	NC	3.98E-04
Benzo(ghi)perylene	5.02E-09	<0.000010	0.012	0.021	NC	1.56E-03
Chrysene	2.76E-08	<0.000010	0.005	0.001	NC	4.07E-05
Dibenz(a,h)anthracene	3.13E-09	<0.000010	0.004	0.001	NC	4.63E-04
Indeno(1,2,3-cd)pyrene	3.74E-09	<0.000010	0.010	0.001	NC	1.26E-03

MPOI = Maximum Point of Impingement; µg/m^3 = microgram per cubic metre; mg/L = milligram per litre; mg/kg = milligram per kilogram

Table 4.6-6: Project Case Exposure Point Concentrations for Environmental Media

Chemicals of Potential Concern	Air (µg/m^3)	Water (mg/L)	Soil (mg/kg)	Plant (mg/kg)	Fish (mg/kg)	Meat (mg/kg)
Arsenic						
MPOI	1.99E-01	0.0441	280.73	16.18	2.63	0.56
Worker Camp	1.13E-01	0.0008	225.05	9.20	0.048	0.45
Marian River	1.72E-03	0.0017	153.39	0.24	0.103	0.31
Hislop Lake	1.72E-03	0.0017	153.00	0.19	0.103	0.31
Bea Lake	9.99E-04	0.0017	152.79	0.17	0.103	0.31
Cobalt						
MPOI	2.51E-02	0.0061	53.44	2.38	0.329	1.07
Worker Camp	1.41E-02	0.0005	46.40	1.50	0.027	0.93
Iron						
MPOI	1.60E+01	3.297	17104.27	1267.80	1038	342
Worker Camp	8.29E+00	0.989	12143.00	664.27	311	243
Total PAHs at MPOI						
Benzo(a)anthracene	1.48E-05	<0.000010	0.008	1.08E-03	NC	6.43E-05
Benzo(a)pyrene	1.49E-05	<0.000010	0.192	1.08E-03	NC	6.20E-03
Benzo(b,k)fluoranthene	7.40E-05	<0.000010	0.015	1.38E-03	NC	4.99E-04
Benzo(ghi)perylene	2.40E-05	<0.000010	0.013	2.09E-02	NC	1.69E-03
Chrysene	2.87E-05	<0.000010	0.006	1.16E-03	NC	5.12E-05
Dibenz(a,h)anthracene	1.15E-05	<0.000010	0.004	1.06E-03	NC	5.24E-04
Indeno(1,2,3-cd)pyrene	8.63E-06	<0.000010	0.010	1.04E-03	NC	1.30E-03

MPOI = Maximum Point of Impingement; µg/m^3 = microgram per cubic metre; mg/L = milligram per litre; mg/kg = milligram per kilogram

It should be noted that PAHs in surface water were not predicted for the Project Case as discussed in Section 4.6.1.3.2. The values provided in the table for the MPOI location are the maximum baseline concentrations of carcinogenic PAHs measured in Nico Lake, Peanut Lake, Burke Lake, Marian River, and Reference Lake, which were all lower than the laboratory method detection limits.

4.6.2.3 Receptor Scenarios

As shown above, COPCs were identified at the MPOI, Worker Camp, Marian River, Hislop Lake, and Bea Lake receptor locations. Given that no COPCs were identified at any of the local communities (i.e., Gamètì and Whatì), community residents were not assessed herein. The following receptors were identified:

- Recreational User: where individuals were assumed to use the culturally significant areas for 30 days per year for a lifetime;
- Worker: where individuals were assumed to live at the on-site worker camp and work on the mine site for 250 days per year during the duration of the NICO Project; and
- Resident/Worker: where individuals were assumed to live in selected communities and work on the mine site. It was assumed for this scenario that workers would be on a work schedule with two weeks on, two weeks off. Therefore, the Resident/Worker would be expected to be at the Worker Camp for half of the time

that the Worker receptor spends on-site (i.e., 125 days per year); the remainder of the year was considered to be spent at a nearby community. It was also assumed that the Resident/Worker could also use the culturally significant areas around the NICO Project for 30 days per year.

The Recreational User was considered to spend the 30 days at one of the 3 culturally significant areas of Hislop Lake, Marian River or Bea Lake, or at the MPOI location as a worst-case scenario. The Worker was considered to be present at the on-site Worker Camp for 250 days per year. The Resident/Worker was considered to spend half of the year (125 days/year) at the on-site Worker Camp and 30 days per year at one of the three culturally significant areas of Hislop Lake, Marian River or Bea Lake, or at the MPOI location as a worst-case scenario.

4.6.2.4 Exposure Pathways Assessed for each Receptor

Exposure pathways differed depending on the receptor scenario assessed (Table 4.6-7). For example, Workers were not assessed for ingestion of traditional foods (i.e., fish, wild game, and plants) because these receptors were not considered to be First Nations residents of the nearby communities and they were considered to be consuming store-bought foods only. However, the Resident/Worker, which was considered to be a member of a nearby community that could work at the mine, was assumed to consume traditional foods.

Table 4.6-7: Exposure Pathways Assessed for Each Receptor Type

Exposure Pathway	Recreational User	Worker	Resident/Worker
Inhalation of air	✓	✓	✓
Inhalation of soil dust	✓	✓	✓
Ingestion of surface water	✓	✓	✓
Ingestion of fish	✓	x	✓
Ingestion of soil	✓	✓	✓
Dermal contact with soil	✓	✓	✓
Ingestion of plants	✓ (berries)	x	✓ (berries)
Ingestion of animals	✓ (caribou)	x	✓(caribou)

✓ = Evaluated; x = not evaluated.

4.6.2.5 Exposure Factors

Exposure factors are used to define the receptor characteristics (e.g., body weight), contact rates (e.g., water ingestion rate) and exposure frequencies (i.e., number of days exposed) of each receptor assessed in the HHRA (Table 4.6-8). These exposure factors are used in the EDI calculations.

For non-carcinogenic COPCs, the adult and toddler lifestages were evaluated for recreational users. Toddlers are considered to be more susceptible to the effects of chemicals than adults because they typically have a greater intake rate to body weight ratio and certain behavioral activities that 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 adults. Consistent with risk assessment guidance (Health Canada 2007), the toddler life stage (i.e., 7months to 4 years) was chosen as the most sensitive child lifestage.

For carcinogenic COPCs, a composite receptor was employed to amortize exposure over the average lifetime expectancy (75 years), consistent with Health Canada guidance (Health Canada 2007). A composite receptor is used to assess risk across all lifestages combined over a lifetime. The life stages include infants (i.e., birth 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).

Worker receptors were considered to be adults throughout the time they spend working at the NICO Project. As such, assessment of non-carcinogenic and carcinogenic COPCs included evaluating exposure for adulthood exposure only (i.e., did not include exposure during childhood).

Table 4.6-8: Exposure Factors for the Human Receptors Assessed in the Multi-Media Risk Assessment

Exposure Factor	Units	Worker	Reference	Infant	Toddler	Child	Adolescent	Adult	Reference
Age	years	> 20	Health Canada (2004)	birth to 6 months	6 months to 4 years	5 to 11	12 to 19	>20	Health Canada (2004)
ET (exposure time)	hr/d	12	TK Report	24	24	24	24	24	Assumed
EF (exposure frequency)	d/yr	250 (Worker camp)/30 (Rec. Area)	TK Report	30	30	30	30	30	Assumed
ED (exposure duration)	yr	18	Life of Mine	0.5	4.5	7	8	55	Health Canada (2007)
AT (averaging time - non-carcinogens)	yr	18	Life of Mine	0.5	4.5	7	8	55	Health Canada (2007)
AT (averaging time - carcinogens)	yr	75	Health Canada (2007)	75	75	75	75	75	Health Canada (2007)
BW (body weight)	kg	70.7	Health Canada (2004)	8.2	16.5	32.9	59.7	70.7	Health Canada (2004)
IR_s (soil ingestion rate)	g/d	0.02	Health Canada (2004)	0.02	0.08	0.02	0.02	0.02	Health Canada (2004)
IR_{dw} (drinking water ingestion rate)	L/d	1.5	Health Canada (2004)	0.3	0.6	0.8	1.0	1.5	Health Canada (2004)
IR_{fish} (fish ingestion rate)	kg/d	0	Assumed	0	0.095	0.17	0.20	0.22	Health Canada (2004)
IR_{meat} (wild game ingestion rate)	kg/d	0	Assumed	0	0.085	0.125	0.175	0.270	Health Canada (2004)
IR_{veg} (berry ingestion rate)	kg/d	0	Assumed	0.010	0.024	0.019	0.018	0.019	US EPA (2011)

Table 4.6-8: Exposure Factors for the Human Receptors Assessed in the Multi-Media Risk Assessment (continued)

Exposure Factor	Units	Worker	Reference	Infant	Toddler	Child	Adolescent	Adult	Reference
SA (exposed surface area) hands	cm^2	890	Health Canada (2004)	320	430	590	800	890	Health Canada (2004)
R_{adher} (rate of adherence) hands	g/cm^2	0.001	Health Canada (2004)	1.0E-07	1.0E-07	1.0E-07	1.0E-07	1.0E-07	Health Canada (2004)
R_{inh} (rate of inhalation)	m^3/d	15.8	Health Canada (2004)	2.1	9.3	14.5	15.8	15.8	Health Canada (2004)
PM_{10} (inhalable dust concentration in air)	$\mu g/m^3$	28.3	Predicted annual average at the Worker Camp	50.6	50.6	50.6	50.6	50.6	Predicted annual average at the MPOI
F_{soil} (fraction of dust from site soil)	-	1.0	Assumed	1.0	1.0	1.0	1.0	1.0	Assumed

yr = year; kg = kilogram; g/d = gram per day; L/d = litre per day; kg/d = kilogram per day; cm^2 = square centimetre; g/cm^2 = gram per square centimetre;m^3/d = cubic metre per day; $\mu g/m^3$ = microgram per cubic metre

4.6.2.6 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 (Health Canada 1995; US 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.

The following bioavailability adjustments were made for arsenic and PAHs.

4.6.2.6.1 Arsenic in Soil

Inorganic arsenic in soils impacted by mining activities and smelters have been shown in in vitro bioaccessibility tests to have estimated absorption efficiencies of 5 to 50%. It was considered conservative to adopt the highest absorption efficiency of 50% for this HHRA.

4.6.2.6.2 Arsenic in Food

An oral RAF of 50% was used for arsenic in food (berries).

4.6.2.6.3 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 2005b).

4.6.2.6.4 Polycyclic Aromatic Hydrocarbons in Fish

Biomagnification of PAHs up aquatic food chains is unlikely to occur since the elimination of PAHs and their metabolites occurs relatively rapidly in fish (ATSDR 1995a; Eisler 1987; Health Canada 1994b). The breakdown products from PAH metabolism (polyhydroxy compounds) are eliminated in feces and urine (ATSDR 1995b). As a precautionary measure, the non-detect values for the PAHs identified in the problem formulation were used to assess the dietary risks for PAH compounds from fish consumption.

4.6.2.7 Equations used for the Estimated Daily Intakes

Exposure estimate equations used to calculate EDIs for the Multi-Media Rsk Assessment were adapted from those provided by Health Canada (2004) and are provided below. Exposure parameters used in the assessment are presented in Table 4.6-8.

4.6.2.7.1 Incidental Ingestion of Soil

$$EDI\ \ (mg/kg \cdot day) = \frac{C_S \times IR_S \times RAF_{GIT} \times EF \times ED}{BW \times AT \times CF}$$

Where:

C_S = concentration of COPC in soil (mg/kg)
IR_S = receptor soil ingestion rate (kg/d)
RAF_{GIT} = relative absorption factor from the gastrointestinal tract (unitless)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF = unit conversion factor (365 d/yr)

4.6.2.7.2 Inhalation of Soil Particles

$$EDI\ (mg/kg \cdot day) = \frac{C_S \times R_{Inh} \times PM_{10} \times F_{soil} \times RAF_{Inh} \times ET \times EF \times ED}{BW \times AT \times CF}$$

Where:

C_S = concentration of COPC in soil (mg/kg)
R_{Inh} = receptor inhalation rate (m^3/hr)
PM_{10} = concentration of particulate matter less than 10 microns (kg/m^3)
F_{soil} = fraction of inhaled dust generated from soil (unitless)
RAF_{Inh} = inhalation absorption factor (unitless)
ET = exposure time (hr/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)
AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF = unit conversion factor (365 d/yr)

4.6.2.7.3 Dermal Contact with Soil

$$EDI\ (mg/kg \cdot day) = \frac{C_S \times RAF_{DER} \times R_{adher} \times \sum(SA) \times EV \times EF \times ED}{BW \times AT \times CF}$$

Where:

C_S = concentration of COPC in soil (mg/kg)
RAF_{DER} = relative absorption factor for skin (unitless)
R_{adher} = rate of soil adherence to skin (kg/cm^2-event)
SA = exposed skin surface area (cm^2)
EV = events per day (event/d)
EF = exposure frequency (d/yr)
ED = exposure duration (yr)
BW = body weight (kg)

AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF = unit conversion factor (365 d/yr)

4.6.2.7.4 Ingestion of Contaminated Drinking Water

$$EDI\ \ (mg/kg \cdot day) = \frac{C_W \times IR_W \times RAF_{GIT} \times EF \times ED}{BW \times AT \times CF}$$

Where:

C_W = concentration of COPC in water (mg/L)
IR_W = ingestion rate of water (L/day)
RAF_{GIT} = relative absorption factor from the gastrointestinal tract (unitless)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF = unit conversion factor (365 d/yr)

4.6.2.7.5 Dermal Contact with Contaminated Water

$$EDI\ \ (mg/kg \cdot day) = \frac{C_W \times RAF_{DER} \times K_p \times \sum(SA) \times CF_1 \times ET \times EF \times ED}{BW \times AT \times CF_2}$$

Where:

C_W = concentration of COPC in drinking water (mg/L)
RAF_{DER} = relative absorption factor for skin (unitless)
K_p = dermal permeability coefficient (cm/hour)
SA = exposed skin surface area (cm^2)
CF_1 = unit correction factor (10^{-3} L/cm^3)
ET = exposure time (hours/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF_2 = unit conversion factor (365 d/yr)

4.6.2.7.6 Ingestion of Contaminated Produce, Fish, Game or Other Food

$$EDI\ \ (mg/kg \cdot day) = \frac{\left[\sum\left(C_{Foodi} \times IR_{Foodi} \times RAF_{GIT} \times EF\right)\right] \times ED}{BW \times AT \times CF}$$

Where:

C_{Foodi} = concentration of COPC in food "i" (mg/kg)
IR_{Foodi} = receptor ingestion rate for food "i" (kg/d)
RAF_{GIT} = relative absorption factor from the gastrointestinal tract for food "i" (unitless)
EF = exposure frequency for food "i" (d/yr)
ED = exposure duration (d/yr)
BW = body weight (kg)
AT = averaging time (equal to ED for non-carcinogens; equal to 75 years for carcinogens)
CF = unit conversion factor (365 d/yr)

4.6.2.8 Predicted Estimated Daily Intakes

The predicted EDIs for the Baseline Case and Project Case for COPCs that are non-carcinogenic (i.e., arsenic, cobalt, and iron) are provided in the tables below for the recreational user (Table 4.6-9), worker (Table 4.6-10), and resident/worker (Table 4.6-11).

Table 4.6-9: Total Estimated Daily Intakes for Non-Carcinogens for the Recreational User

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
MPOI	1.4E-04	1.4E-03
Marian River Receptor	1.1E-04	7.0E-04
Hislop Lake Receptor	1.1E-04	1.2E-04
Bea Lake Receptor	1.1E-04	1.2E-04
Cobalt		
Maximum Point of Impingement	3.9E-04	9.3E-04
Iron		
Maximum Point of Impingement	8.9E-02	8.1E-01

Table 4.6-10: Total Estimated Daily Intakes for Non-Carcinogens for the Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp	3.6E-05	6.5E-05
Cobalt		
Worker Camp	1.6E-05	2.0E-05
Iron		
Worker Camp	1.6E-02	1.8E-02

Table 4.6-11: Total Estimated Daily Intakes for Non-Carcinogens for the Resident/Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp/ Maximum Point of Impingement	9.1E-05	4.5E-04
Worker Camp/Marian River Receptor	7.2E-05	9.2E-05
Worker Camp/Hislop Lake Receptor	7.2E-05	9.1E-05
Worker Camp/Bea Lake Receptor	7.2E-05	9.1E-05
Cobalt		
Worker Camp/Maximum Point of Impingement	2.6E-04	5.0E-04
Worker Camp/Marian River Receptor	2.5E-04	2.5E-04
Worker Camp/Hislop Lake Receptor	2.5E-04	2.5E-04
Worker Camp/Bea Lake Receptor	2.5E-04	2.5E-04
Iron		
Worker Camp/Maximum Point of Impingement	6.6E-02	4.2E-01
Worker Camp/Marian River Receptor	5.1E-02	5.3E-02
Worker Camp/Hislop Lake Receptor	5.1E-02	5.3E-02
Worker Camp/Bea Lake Receptor	5.1E-02	5.3E-02

Similarly, the predicted EDIs for the Baseline Case and Project Case for COPCs that are carcinogenic (i.e., arsenic and carcinogenic PAHs) are provided in the tables below for the recreational user (Table 4.6-12) and resident/worker (Table 4.6-13). Note that carcinogenic PAHs were retained only for the MPOI location; therefore, the only receptors for which EDIs (and ILCRs) were calculated are the recreational user and the resident/worker given that the worker was only considered to be at the Worker Camp location.

Table 4.6-12: Total Estimated Daily Intakes for Carcinogens for the Recreational User

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Maximum Point of Impingement	3.0E-05	4.2E-04
Marian River Receptor	9.4E-06	1.6E-04
Hislop Lake Receptor	8.7E-06	1.5E-05
Bea Lake Receptor	8.7E-06	1.4E-05
Carcinogenic PAHs		
Maximum Point of Impingement	3.5E-06	3.7E-06

Table 4.6-13: Total Estimated Daily Intakes for Carcinogens for the Resident/Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp/ Maximum Point of Impingement	8.9E-05	2.3E-03
Worker Camp/Marian River Receptor	8.1E-05	2.0E-03
Worker Camp/Hislop Lake Receptor	8.1E-05	1.6E-03
Worker Camp/Bea Lake Receptor	8.1E-05	1.6E-03
Carcinogenic PAHs		
Maximum Point of Impingement	4.6E-06	4.8E-06

4.6.3 Toxicity Assessment

The toxicity assessment provides the basis for evaluating what is an acceptable exposure and what level of exposure may adversely affect people's health. The toxicity assessment for the Multi-Media Risk Assessment is based on long-term (chronic) toxicity studies. Toxicity assessment involves determining the amount of a chemical a person may take into his or her body through all applicable exposure pathways without it affecting their health. This parameter is called 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). An RfD is an estimate of daily oral exposure to the human population (including susceptible subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. An SF is an upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to a chemical.

Available TRVs (RfDs and SFs) were compiled from the following agencies:

- Health Canada (Health Canada 2008);
- US EPA's IRIS (US EPA 2010, internet site);
- World Health Organization (WHO 2000);
- Agency for Toxic Substances and Disease Registry (ATSDR 2009, internet site); and
- Netherlands National Institute of Public Health and the Environment (RIVM 2001).

If RfDs or SFs were not available from any of these agencies, relevant RfDs and SFs were compiled from other sources including provisional sources. The most conservative of the TRVs was selected for use in the Multi-Media Risk Assessment, unless the most conservative TRV was not based on health endpoints or otherwise was not applicable to the assessment.

The available RfDs and RfCs (described in Section 4.5), selected RfDs and RfCs and their toxicological bases are presented in Table 4.6-14. The available SFs and unit risks, selected SFs and unit risks and their toxicological bases are presented in Table 4.6-15.

With the exception of arsenic and the carcinogenic PAHs, none of the COPCs for the Multi-Media Risk Assessment are carcinogenic via the oral route.

Table 4.7-14: Reference Dose and Reference Concentrations for Chemicals of Potential Concern Evaluated in the Multi-Media Risk Assessment

Parameter	Toxicity Reference Value					Toxicological Endpoints and Derivations
	Health Canada[a]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Arsenic						
Reference Dose (mg/kg/day)	n/a	**0.0003**	0.0003	0.001	n/a	The IRIS oral RfD is based upon a chronic epidemiological study of the Taiwanese population exposed to arsenic in drinking water. No evidence of skin lesions or blackfoot disease were observed where the average arsenic concentrations were 0.009 mg/L (range of 0.001 to 0.017 mg/L). Adjustments based upon water intake rate, arsenic ingested in food, and body weight yielded a NOAEL of 0.0008 mg/kg-day; an uncertainty factor of 3 to account for lack of reproductive data and uncertainty whether sensitive individuals were accounted for was applied. The ATSDR chronic duration oral MRL is based upon the same dataset and derivation as the IRIS oral RfD. Using the same dataset as above, RIVM derived a tolerable daily intake (TDI) of 0.001 mg/kg-day based upon the WHO PTWI (permissible tolerable weekly intake) of 15 µg/kg bw/week translated into a daily intake of 2.1 µg/kg bw/day. An uncertainty factor of 2 was applied to compensate for observational errors that are inevitable in epidemiological studies.
Reference Concentration (µg/m^3)	n/a	n/a	n/a	**1.0**	n/a	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 intrahuman variability.
Cobalt						
Reference Dose (mg/kg-day)	n/a	n/a	n/a	**0.0014**	n/a	RIVM (2001) provides a TDI of 0.0014 mg/kg-day; this value was derived in a previous evaluation of Vermeire et al. (1991, as cited in RIVM, 2001), based on a migration limit for packaging materials. For humans, the lowest LOAEL reported is 0.04 mg/kg bw/day, for cardiomyopathy following intermediate oral exposure. An uncertainty factor of 3 for intra-human variation, and a factor of 10 for extrapolation to a NOAEL were applied, resulting in a TDI of 0.0014 mg/kg-day.

Table 4.7-14: Reference Dose and Reference Concentrations for Chemicals of Potential Concern Evaluated in the Multi-Media Risk Assessment (continued)

Parameter	Toxicity Reference Value					Toxicological Endpoints and Derivations
	Health Canada[a]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Reference Concentration (µg/m^3)	n/a	n/a	1.0	**0.5**	n/a	The ATSDR chronic duration inhalation MRL is based on a NOAEL of 5.3 µg/m^3 based upon decreased respiratory function in exposed workers. The NOAEL was adjusted for continuous exposure (1.9 µg/m^3) and an uncertainty factor of 300 was applied (3 for extrapolation from an assumed subchronic to chronic study, 10 for database insufficiencies including lack of developmental inhalation studies and multigenerational studies, and 10 to account for lack of data on human variability and sensitive populations). A TCA (tolerable concentration in air, analogous to an inhalation RfC) of 0.5 µg/m^3 was available from RIVM. The TCA is based upon a LOAEL of 50 µg/m^3 for interstitial lung disease, from which the TCA of 0.5 µg/m^3 was derived using an uncertainty factor of 100 (10 for the extrapolation from a LOAEL to a NOAEL and 10 for intrahuman variability).
Iron						
Reference Dose (mg/kg/day)	n/a	n/a	n/a	n/a	0.7	The PPRTV for iron is based upon daily treatment with 60 mg elemental iron for one month that was associated with a statistically significant increase in gastrointestinal effects compared to a placebo. The LOAEL of 71 mg/day was determined by adding the daily treatment dose to the estimated dietary intake of iron of 11 mg elemental iron per day. Using a reference body weight of 70 kg and an uncertainty factor of 1.5 (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), the provisional RfD of iron of 0.7 mg/kg-day was derived.
Reference Concentration (µg/m^3)	n/a	n/a	n/a	n/a	n/a	No inhalation RfCs were available for iron.
Benzo(a)pyrene						
Reference Dose (mg/kg/day)	n/a	n/a	n/a	n/a	n/a	No oral RfDs were available for benzo(a)pyrene.
Reference Concentration (µg/m^3)	n/a	n/a	n/a	n/a	n/a	No inhalation RfCs were available for benzo(a)pyrene.

[a] Health Canada (2009).

[b] United States Environmental Protection Agency Integrated Risk Information System (US EPA 2010, internet site).

[c] Agency for Toxic Substances and Disease Registry (ATSDR 2010, internet site).

[d] National Institute of Public Health and the Environment (RIVM 2001).

[e] Source of TRV is explained in toxicological endpoint section, as TRVs were available from other jurisdictions.

Note: Bolded Toxicity Reference Values (TRVs) were used in the risk assessment. Unless otherwise stated, the most conservative of the available TRVs was chosen (i.e., the lowest).

RfC = reference concentration; RfD = reference doses LOAEL = lowest observed adverse effect level; n/a = Not available; µg/m^3 = microgram per cubic metre; mg/kg/day = milligram per kilogram per day

Table 4.7-15: Inhalation Unit Risks for Chemicals of Potential Concern Evaluated in the Multi-Media Risk Assessment

Parameter	Toxicity Reference Value					Toxicological Endpoints and Derivations
	Health Canada[a]	US EPA IRIS[b]	ATSDR[c]	RIVM[d]	Other[e]	
Arsenic						
Oral Slope Factor ([mg/kg/day]$^{-1}$)	**1.8**	1.5	n/a	n/a	n/a	The Health Canada oral slope factor is based on an epidemiological study in humans exposed to drinking water containing elevated concentrations of arsenic (Health Canada 2008).
Inhalation Unit Risk ([µg/m^3]$^{-1}$)	**0.0064**	0.0043	n/a	0.01	n/a	The Health Canada Inhalation Unit Risk is based on an epidemiological study of lung cancer in occupationally exposed workers in which the TC_{05} was 7.83 µg/m³. The IRIS Inhalation Unit Risk is based on the geometric mean of several studies for the incidence of lung cancer in human males with occupational exposure. The RIVM TCA is based on the trivalent form of arsenic, and is derived from the LOAEC of 10 µg/m^3 for lung cancer in humans. An uncertainty factor of 10 was applied for the variation in susceptibility.
Benzo(a)pyrene						
Oral Slope Factor ([mg/kg/day]$^{-1}$)	**2.3**	7.3	n/a	n/a	n/a	The Health Canada oral SF is based upon a subchronic study in mice exposed to benzo(a)pyrene in the diet for 110 days. Gastric tumours (squamous cell papillomas, some carcinomas) were observed. Using linear extrapolation and surface area correction, a unit lifetime risk of 5E-05 was derived, which was used to derive the oral SF. The IRIS oral SF was based upon the geometric mean of four SFs ranging from 4.5 to 11.7 per mg/kg-day based oral dietary studies in rats and mice where the critical effects were forestomach and squamous cell papillomas and carcinomas; squamous cell carcinoma of the forestomach; and forestomach, larynx, and esophagus papillomas and carcinomas. The Health Canada SF was derived more recently and was used preferentially over the IRIS SF.
Inhalation Unit Risk ([µg/m^3]$^{-1}$)	**0.000031**	n/a	n/a	n/a	0.011 (Cal EPA)	The Health Canada Inhalation Unit Risk is based on the TC_{05} for respiratory tract tumours in hamsters exposed for 4.5 hours per day and 7 days per week for up to 96 weeks. The Cal EPA Unit Risk is based on respiratory tract tumours in hamsters from an inhalation bioassay and intratracheal administration of benzo(a)pyrene.

[a] Health Canada (2009).

[b] United States Environmental Protection Agency Integrated Risk Information System (US EPA 2010, internet site).

[c] Agency for Toxic Substances and Disease Registry (ATSDR 2010, internet site).

[d] National Institute of Public Health and the Environment (RIVM 2001).

[e] Source of TRV is explained in toxicological endpoint section, as TRVs were available from other jurisdictions.

Note: Bolded Toxicity Reference Values (TRVs) were used in the risk assessment. Unless otherwise stated, the most conservative of the available TRVs was chosen (i.e., the lowest).

SF = Slope Factors n/a = Not available; µg/m^3 = microgram per cubic metre; mg/kg/day = milligram per kilogram per day

4.6.4 Risk Characterization

Long-term health effects were evaluated by calculating Hazard Quotients (HQs) for chemicals that do not cause cancer (i.e., arsenic, cobalt, and iron) and Incremental Lifetime Cancer Risks (ILCRs) for chemicals which cause cancer (i.e., arsenic and carcinogenic PAHs).

The estimated HQs for the Baseline Case and Project Case for the non-carcinogenic effects of COPCs (i.e., arsenic, cobalt and iron) are provided in the tables below for the recreational user (Table 4.6-16), worker (Table 4.6-17), and resident/worker (Table 4.6-18). Estimated HQs that are greater than 1 are indicated by bolded and shaded formatting.

Table 4.6-16: Total Hazard Quotients for the Recreational User

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Maximum Point of Impingement	0.5	**5**
Marian River Receptor	0.4	**2**
Hislop Lake Receptor	0.4	0.4
Bea Lake Receptor	0.4	0.4
Cobalt		
Maximum Point of Impingement	0.3	0.7
Iron		
Maximum Point of Impingement	0.1	1

Note: Bold and shaded values exceed the target HQ of 1.

Table 4.6-17: Total Hazard Quotients for the Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp	0.1	0.2
Cobalt		
Worker Camp	0.01	0.1
Iron		
Worker Camp	0.02	0.02

Table 4.6-18: Total Hazard Quotients for the Resident/Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp/Maximum Point of Impingement	0.3	**2**
Worker Camp/Marian River Receptor	0.3	0.4
Worker Camp/Hislop Lake Receptor	0.3	0.4
Worker Camp/Bea Lake Receptor	0.3	0.3
Cobalt		
Worker Camp/Maximum Point of Impingement	0.2	0.4
Worker Camp/Marian River Receptor	0.2	0.2
Worker Camp/Hislop Lake Receptor	0.2	0.2
Worker Camp/Bea Lake Receptor	0.2	0.2
Iron		
Worker Camp/Maximum Point of Impingement	0.09	0.6
Worker Camp/Marian River Receptor	0.07	0.1
Worker Camp/Hislop Lake Receptor	0.07	0.1
Worker Camp/Bea Lake Receptor	0.07	0.1

Note: Bold and shaded values exceed the target HQ of 1.

The relative contributions to the total HQs for each pathway are shown for the recreational user (toddler life stage) for the Baseline Case and Project Case in Figures 4.6-1 and 4.6-2, respectively. In the Baseline Case, the highest contributions are from soil incidental ingestion and caribou ingestion; however, in the Project Case, the relative contribution of the berry ingestion to the total HQ increases substantially at the MPOI location.

A similar distribution of risks was observed for the Baseline Case for the Resident/Worker receptor as shown in Figure 4.6-3 below. For the Project Case (Figure 4.6-4), the greatest contribution is from water ingestion, followed by berry ingestion.

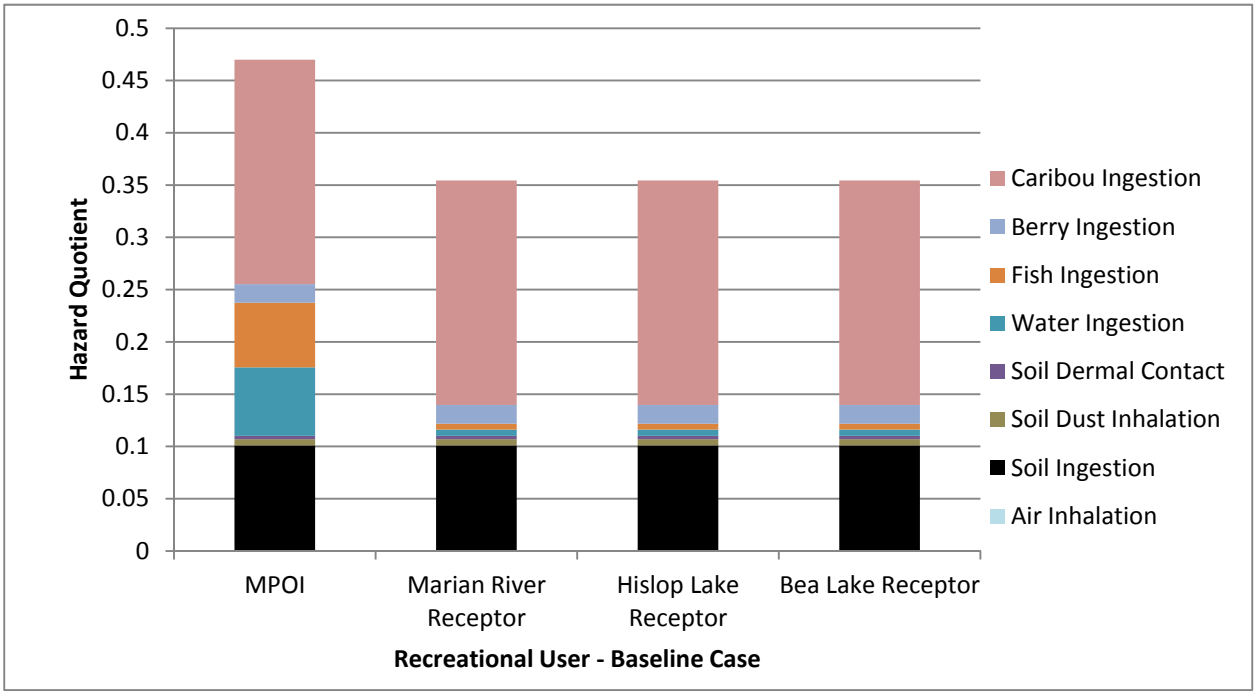


Figure 4.6-1: Hazard Quotients for the Baseline Case for the Recreational User at each Location for Arsenic

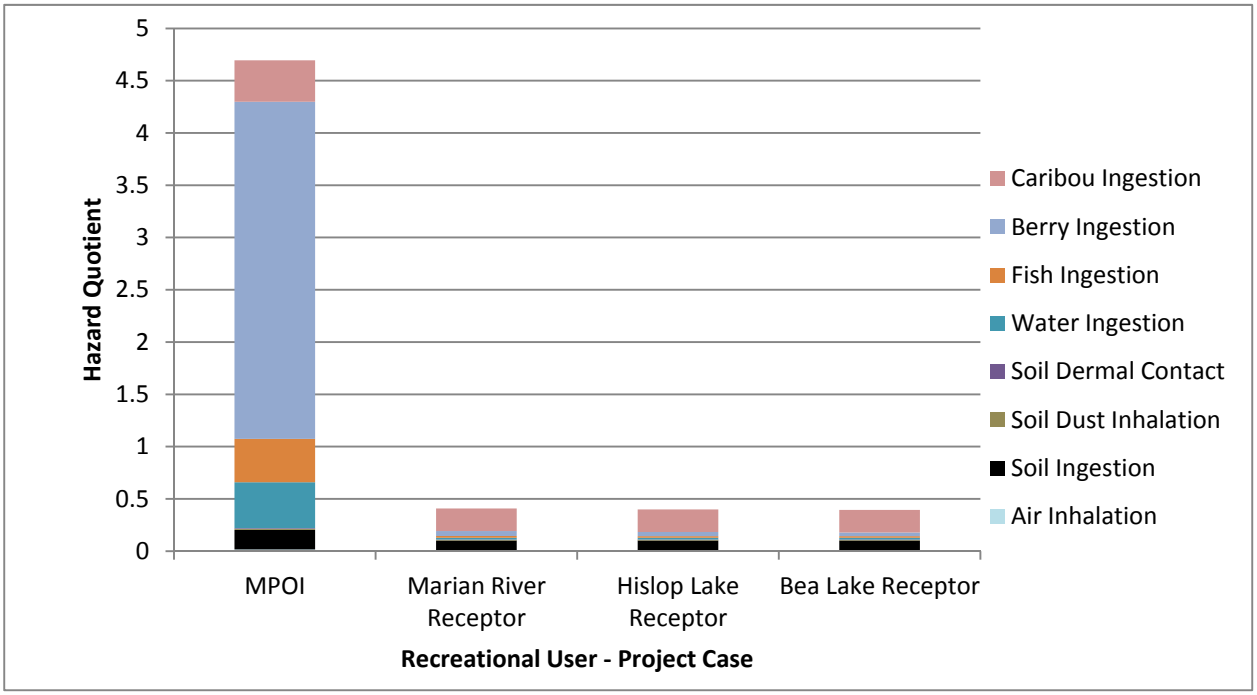


Figure 4.6-2: Hazard Quotients for the Project Case for the Recreational User at each Location for Arsenic

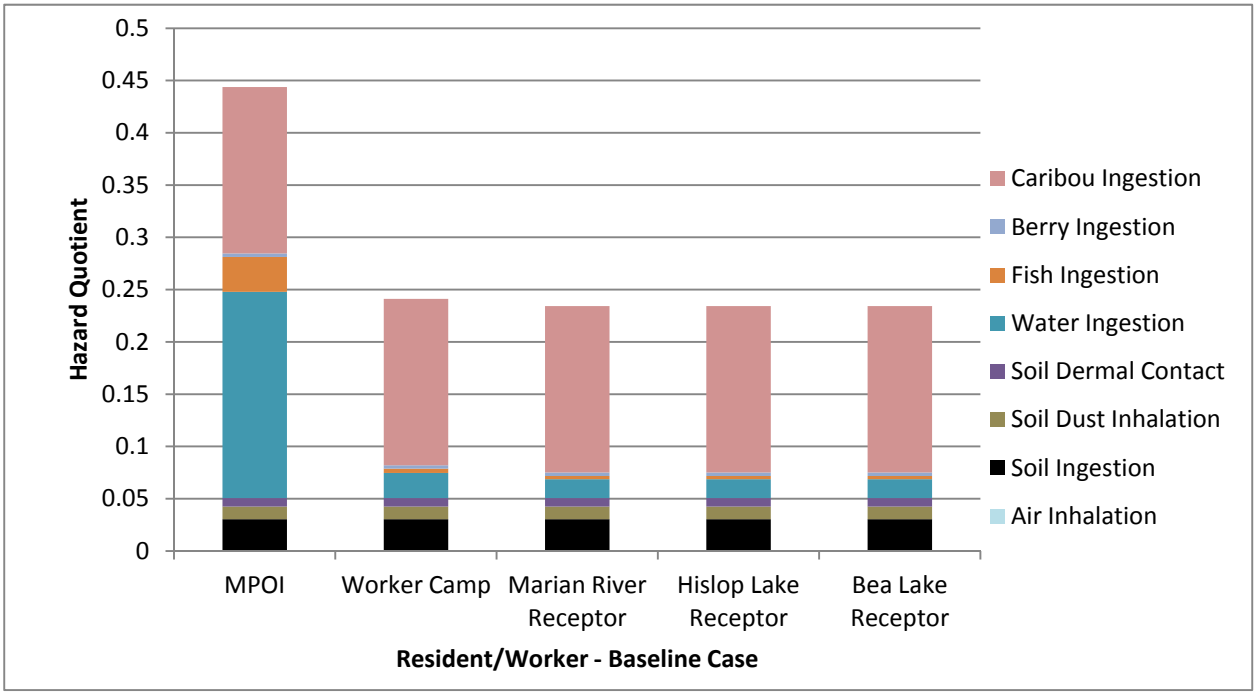


Figure 4.6-3: Hazard Quotients for the Baseline Case for the Resident/Worker at each Location for Arsenic

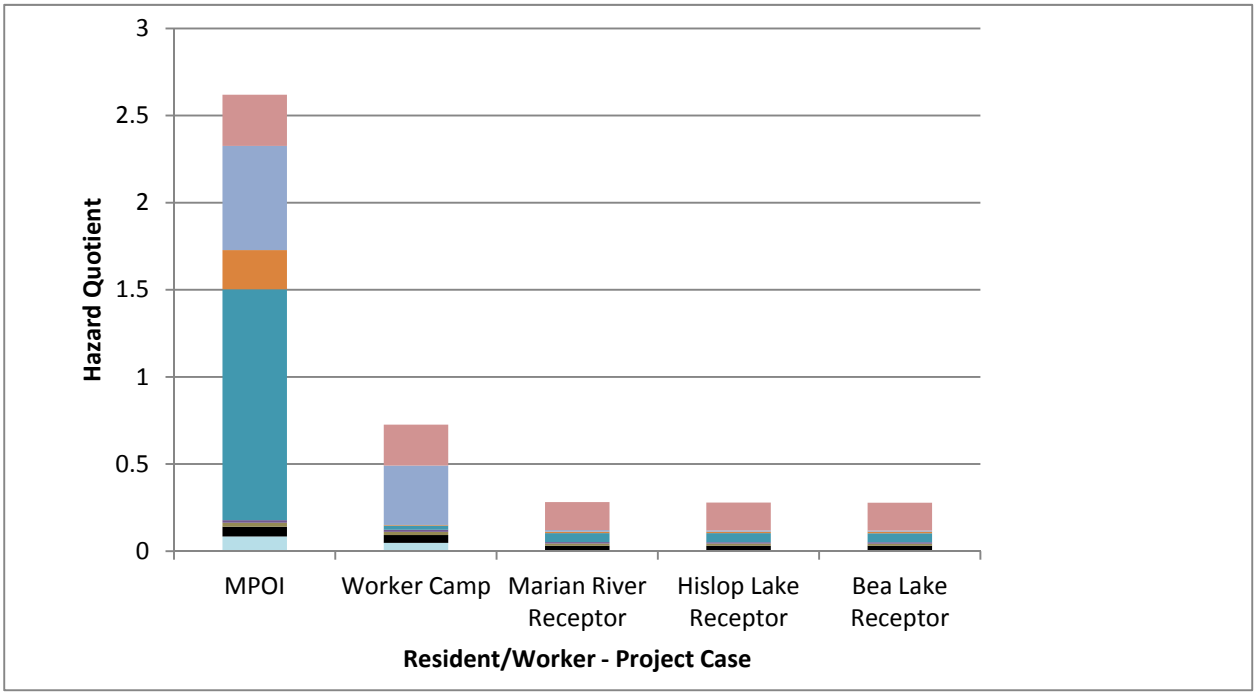


Figure 4.6-4: Hazard Quotients for the Project Case for the Resident/Worker at each Location for Arsenic

The estimated risks generated for carcinogens are based on the ILCR which is the increased lifetime risk attributed to chemical exposure above background cancer risks caused by genetics, lifestyle, and other non-chemical factors. Health Canada (Health Canada 2004; Health Canada 2007) and Alberta Health and Wellness (2006) consider cancer risks from chemical exposure to be essentially negligible if the ILCR is less than 1 in 100 000 (1×10^{-5}). For example, an ILCR less than or equal to 0.00001 for a carcinogen represents an incremental cancer risk above background of less than 1 in 100 000, which is considered a negligible health impact.

The estimated ILCRs for the Baseline Case and Project Case for COPCs that are classified as carcinogenic (i.e., arsenic and carcinogenic PAHs) are provided in the tables below for the recreational user (Table 4.6-19) and resident/worker (Table 4.6-20). Note that carcinogenic PAHs were retained only for the MPOI location; therefore, the only receptors for which ILCRs were calculated are the recreational user and the resident/worker given that the worker was only considered to be at the Worker Camp location. Estimated ILCRs that are greater than 1×10^{-5} (1.0E-05) are indicated by bolded and shaded formatting.

Table 4.6-19: Total ILCRs for Carcinogens for the Recreational User

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Maximum Point of Impingement	**1.4E-04**	**1.0E-03**
Marian River Receptor	**1.0E-04**	**4.6E-04**
Hislop Lake Receptor	**1.0E-04**	**1.1E-04**
Bea Lake Receptor	**1.0E-04**	**1.1E-04**
Carcinogenic PAHs		
Maximum Point of Impingement	6.0E-06	6.4E-06

Note: Bold and shaded values exceed the target HQ of 1.

Table 4.6-20: Total ILCRs for Carcinogens for the Resident/Worker

Chemicals of Potential Concern/ Location	Baseline Case	Project Case
Arsenic		
Worker Camp/Maximum Point of Impingement	**1.6E-04**	**4.4E-03**
Worker Camp/Marian River Receptor	**1.5E-04**	**3.7E-03**
Worker Camp/Hislop Lake Receptor	**1.5E-04**	**3.0E-03**
Worker Camp/Bea Lake Receptor	**1.5E-04**	**3.0E-03**
Carcinogenic PAHs		
Maximum Point of Impingement	4.3E-06	4.6E-06

Note: Bold and shaded values exceed the target HQ of 1.

The relative contributions to the total ILCRs for each pathway are shown for the recreational user (composite) for the Baseline Case and Project Case in Figures 4.6-5 and 4.6-6, respectively. In the Baseline Case, the contribution from caribou ingestion is most substantial at all locations; however, in the Project Case, the relative contribution of the berry ingestion pathway to the total ILCR increases substantially at the MPOI location.

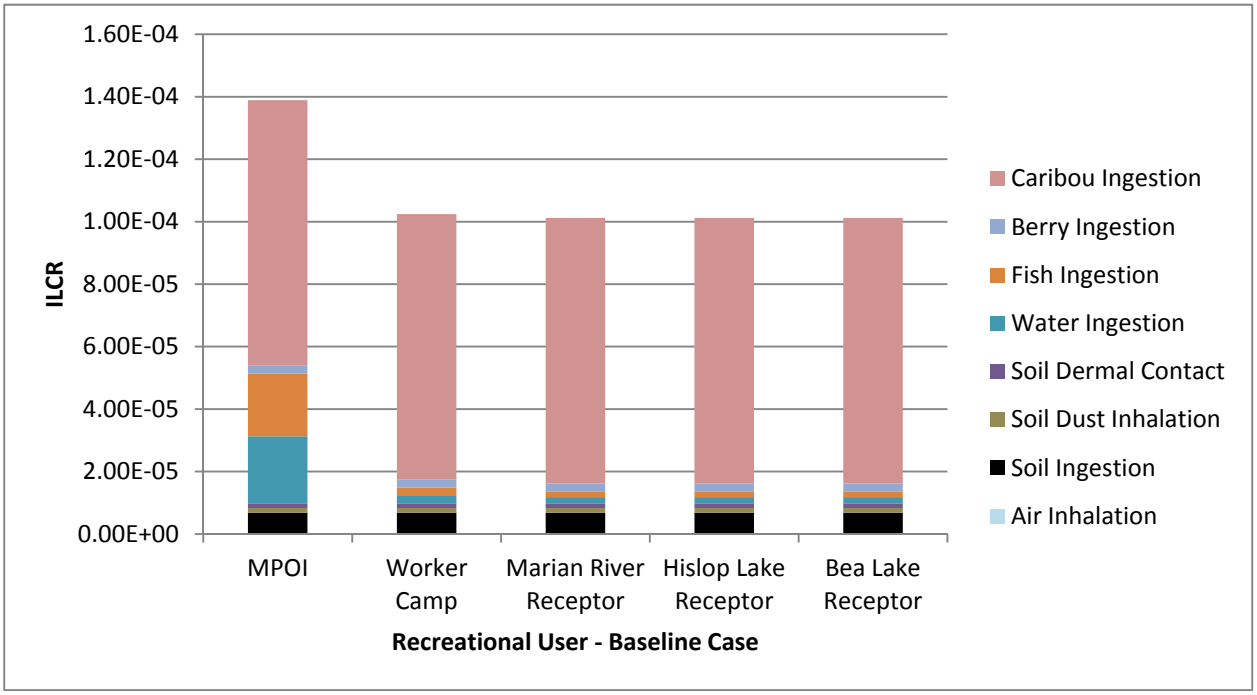


Figure 4.6-5: Incremental Lifetime Cancer Risks for the Baseline Case for the Recreational User at each Location for Arsenic

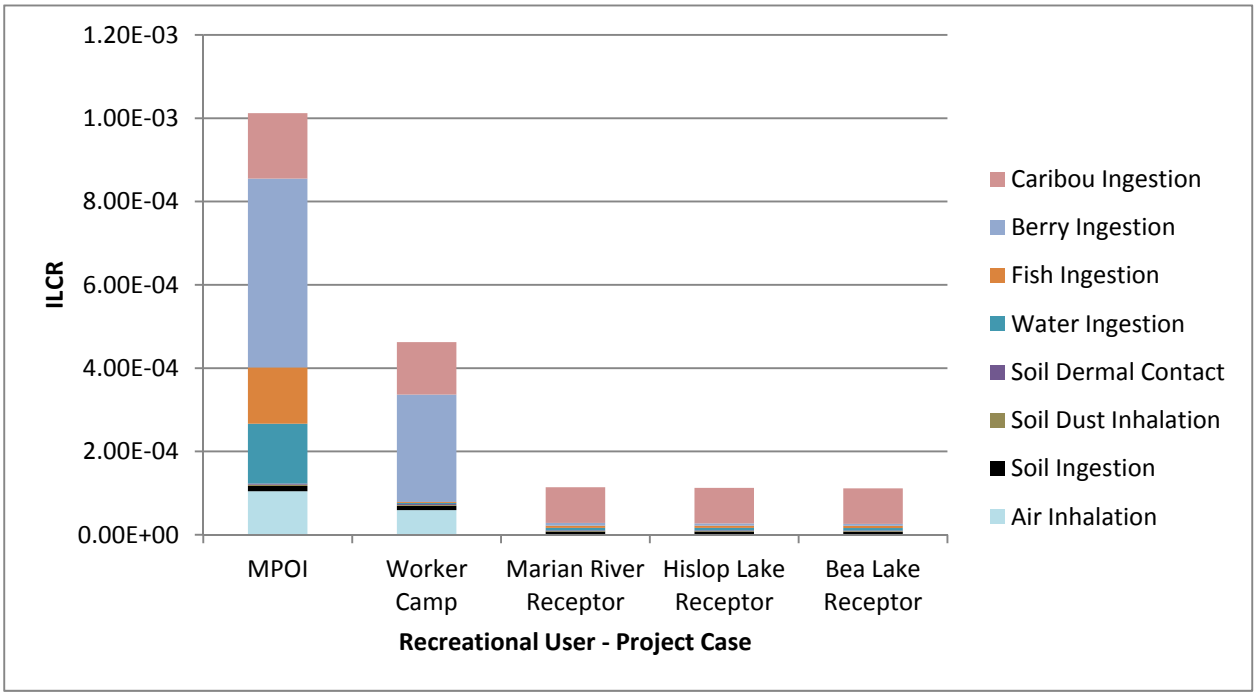


Figure 4.6-6: Incremental Lifetime Cancer Risks for the Project Case for the Recreational User at each Location for Arsenic

As with the HQs, the ILCRs for arsenic showed a similar distribution for the Resident/Worker receptor as shown in Figures 4.6-7 and 4.6-8 below.

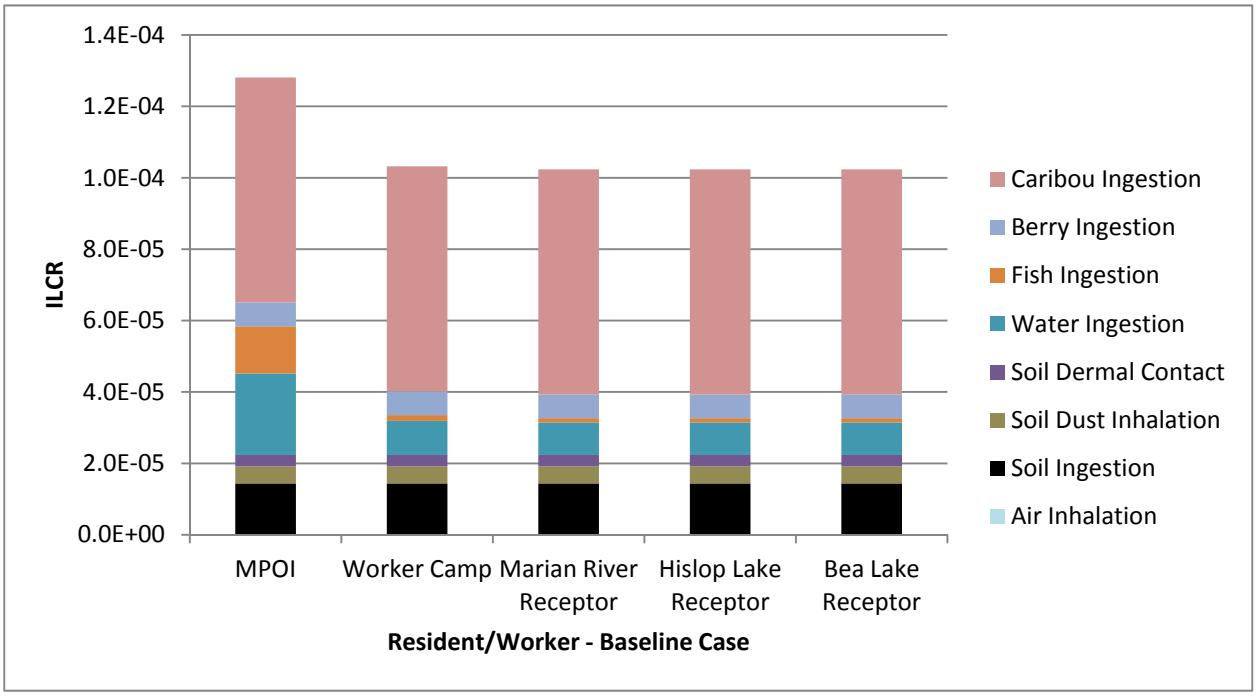


Figure 4.6-7: Incremental Lifetime Cancer Risks for the Baseline Case for the Resident/Worker at each Location for Arsenic

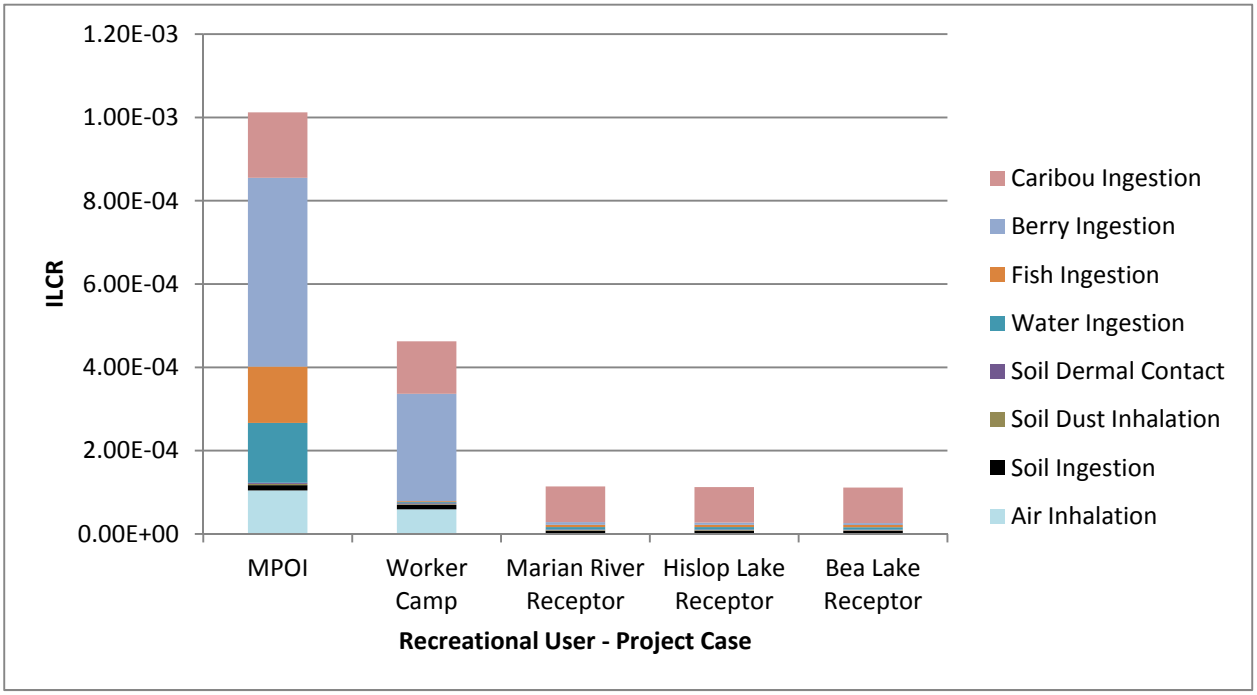


Figure 4.6-8: Incremental Lifetime Cancer Risks for the Project Case for the Resident/Worker at each Location for Arsenic

4.6.4.1 Chemical Mixtures

According to Health Canada (Health Canada 2004; Health Canada 2007), HQs and ILCRs for COPCs that have similar target organs, effects and mechanisms of action should be added together to estimate a total risk for a particular toxicological effect. The COPCs and their target organs and toxicological effects are summarized in Table 4.6-21 below.

Table 4.6-21: Target Organs and Toxicological Effects for the Chemicals of Potential Concern in the Multi-Media Risk Assessment

Chemicals of Potential Concern	Target Organ / Toxicological Effect(s)
Arsenic	
Non-cancer effects for the oral pathway	Skin (hyperpigmentation, keratosis) and possible vascular complications
Non-cancer effects for the inhalation pathway	Pulmonary effects
Cancer effects for the oral pathway	Skin cancer
Cancer effects for the inhalation pathway	Lung cancer
Cobalt	
Non-cancer effects for the oral pathway	Heart (cardiomyopathy)
Non-cancer effects for the inhalation pathway	Interstitial lung disease
Iron	
Non-cancer effects for the oral pathway	Gastrointestinal effects
Carcinogenic PAHs	
Cancer effects for the oral pathway	Gastric tumours
Cancer effects for the inhalation pathway	Respiratory tract tumours

For the Multi-Media Risk Assessment, only the carcinogenic PAHs have similar target organs, effects and mechanisms of action. Thus, estimated risks were only summed for the carcinogenic PAHs in the Multi-Media Risk Assessment.

4.6.5 Uncertainty Analysis

There is always uncertainty associated with risk estimations, depending on the uncertainty and variability associated with the available information. When information is uncertain, it is standard practice in a risk assessment to make assumptions that are biased towards safety so that even if there is uncertainty, human health will still be protected. Every effort was made to ensure that assumptions were specific to the communities being evaluated and where data were limited or unavailable, conservative estimations of exposure were adopted.

There are several levels of safety applied in this assessment. For example, the risk assessment assumed that a person will live in the RSA for their entire lives. It further assumes that this person is a susceptible child or elder who will be exposed to reasonable maximum releases from the NICO Project every day that the facility is operating. The assessment also assumes that local residents are carrying out traditional activities (i.e., hunting, trapping or plant gathering) at the MPOI location. As well, it was assumed that a toddler may accompany an

adult to traplines at the MPOI. Thus, if the risk assessment indicates that predicted risk levels are acceptable for these “maximally exposed” people, then it can be concluded that all people will be protected.

There is also uncertainty associated with estimating toxicity reference values. Toxicity reference values are based on toxicity information available from government databases and published scientific literature. The majority of toxicity information comes from the results of experiments with laboratory animals. Some additional information on human health effects may also be available for some substances where cases of workplace exposures and associated health effects have been documented. There is uncertainty in extrapolating from animal studies and workplace case studies to the possible effects that may result from exposure to releases from the NICO Project. To add a layer of safety, it is standard practice in a risk assessment to assume that people are more sensitive to the toxic effects of a chemical than laboratory animals. Therefore, the toxicity reference values for human health are set much lower than the animal toxicity threshold (typically 100 to 1000 times lower). This large margin of safety ensures that exceeding these toxicity reference values by small amounts will not measurably increase the risk of adverse health effects.

4.6.6 Magnitude of Effects Assessment

For COPCs and locations where estimated HQs were greater than 1 or estimated ILCRs were greater than one-in-one hundred thousand, a magnitude of effects assessment was completed. Arsenic had estimated HQs and ILCRs greater than the target values for the recreational user and resident/worker receptors at the MPOI, Marian River, Hislop Lake, and Bea Lake receptor locations.

The objective of the magnitude of effects assessment is to determine whether the NICO Project has a negligible, low, moderate, or high potential magnitude of risk for the scenarios described above with respect to arsenic. As introduced earlier, an estimated HQ or ILCR that is greater than the target level does not necessarily indicate that a risk is probable; rather, it indicates that further analysis of the assumptions made in the exposure and risk estimates is required. There were several conservative assumptions incorporated into the risk estimates as described in the uncertainty analysis above (Section 4.6.5), and these assumptions may have led to an overestimation of risk. The following analysis was carried out to identify the sources of uncertainty in the assessment and determine the likely level of risk associated with chronic exposure to arsenic at the selected receptor locations for the identified receptors:

- comparison of the magnitude(s) of the calculated HQ to the target HQ of 1;
- comparison of the magnitude(s) of the calculated ILCR to the target ILCR of 1E-05;
- comparison of Project Case risks to Baseline Case risks;
- assessment of the conservatism in the air modelling approach used to predict future concentrations;
- assessment of the conservatism in the exposure assumptions;
- assessment of the conservatism in the chronic toxicity reference values for each COPC; and
- assessment of the potential long-term health effects that may occur at the predicted concentrations.

The magnitude of effects assessment indicated that the NICO Project would likely have an overall negligible potential for chronic health effects (Table 4.6-22).

Table 4.6-22: Magnitude of Effects Assessment for Arsenic (Chronic)

Analysis Criteria	Discussion
Comparison of the Project Case and Baseline Case risks to the target HQ of 1 and target ILCR of 1E-05	***Non-Cancer Risks:*** The Baseline Case HQ estimated for the recreational user (toddler) at the MPOI (0.5) was less than the target HQ of 1. The Baseline Case HQs estimated for the resident/worker (adult) at all locations was 0.3, which is less than the target HQ of 1. The Project Case HQ estimated for the recreational user (toddler) at the MPOI (5) was greater than the target HQ of 1. The Project Case HQ estimated for the resident/worker (adult) at the MPOI (2) was greater than the target HQ of 1. ***Cancer Risks:*** The Baseline Case ILCRs estimated for the recreational user (composite) at the MPOI (1.4E-04), Marian River (1.0E-04), Hislop Lake (1.0E-04) and Bea Lake (1.0E-04) locations were greater than the target ILCR of 1E-05. The Baseline Case ILCRs estimated for the resident/worker (adult) at the MPOI (1.3E-04), Marian River (1.0E-04), Hislop Lake (1.0E-04) and Bea Lake (1.0E-04) locations were greater than the target ILCR of 1E-05. The Project Case ILCRs estimated for the recreational user (composite) at the MPOI (1.0E-03), Marian River (1.1E-04), Hislop Lake (1.1E-04) and Bea Lake (1.1E-04) locations were greater than the target ILCR of 1E-05. The Project Case ILCRs estimated for the resident/worker (adult) at the MPOI (1.8E-03), Marian River (3.1E-04), Hislop Lake (3.0E-04) and Bea Lake (3.0E-04) locations were greater than the target ILCR of 1E-05.
Comparison of the Project Case risks to Baseline Case risks	***Non-Cancer Risks:*** The Project Case HQs for the recreational user (toddler) and the resident/worker (adult) were approximately 10 times greater than the Baseline Case HQs. ***Cancer Risks:*** The Project Case ILCRs for the recreational user (composite) and the resident/worker (adult) at the MPOI location were just over one order of magnitude greater than the Baseline Case ILCRs, and the Project Case ILCRs for the other locations were within an order of magnitude of the Baseline Case ILCRs.
Conservatism and uncertainty in modelling predictions	The concentrations of arsenic in air, soil and water for the Project Case were the predicted annual average concentrations for the highest emission year from the 18-year operations phase of the NICO Project. It was conservatively assumed that each receptor was exposed to these worst-case concentrations throughout their exposure periods (i.e., 75 years for recreational users [i.e., infant through adult life stages] and 55 years for resident/workers [i.e., adult life stage only]).

Table 4.6-22: Magnitude of Effects Assessment for Arsenic (Chronic) (continued)

Analysis Criteria	Discussion
	The predicted concentrations of arsenic in surface water for the Project Case are the 95th percentile of the predicted annual average concentrations during the maximum emission year during the operations phase. The predicted arsenic concentrations used in the HHRA were 0.0441 mg/L for the MPOI location (compared to the measured baseline of 0.0066 mg/L), 0.0008 mg/L for the Worker Camp location (value remained unchanged as a result of the NICO Project), and 0.0017 mg/L for the culturally significant locations of Marian River, Hislop Lake, and Bea Lake (compared to the measured baseline concentration of 0.0006 mg/L at the Marian River). That is, as a result of the Project, arsenic concentrations were predicted to increase by a factor of almost 7 at the MPOI and by a factor of 3 at the culturally significant locations (surface water concentrations did not change at the Worker Camp). The concentrations of arsenic in soil and vegetation (berry) for the Project Case were predicted based upon the maximum wet and dry deposition rates estimated for the highest emission year from the 18-year operations phase of the Project. The predicted concentrations of arsenic in soil at the MPOI, Worker Camp, Marian River, Hislop Lake, and Bea Lake locations were 280.73, 225.05, 153.39, 153.00, and 152.79 mg/kg, respectively (baseline arsenic at all locations was 152.13 mg/kg). That is, as a result of the Project, arsenic concentrations were predicted to increase by a factor of almost 2 at the MPOI and by a factor of 1.5 at the Worker Camp (soil concentrations did not change appreciably for the three culturally significant locations). The predicted concentrations of arsenic in berries at the MPOI, Worker Camp, Marian River, Hislop Lake and Bea Lake locations were 16.18, 9.20, 0.24, 0.19 and 0.17 mg/kg, respectively (baseline arsenic at all locations was 0.09 mg/kg). That is, as a result of the NICO Project, arsenic concentrations were predicted to increase by a factor of almost 200 at the MPOI, a factor of 100 at the Worker Camp, a factor of almost 3 at Marian River, and a factor of approximately 2 at Hislop Lake and Bea Lake. The concentrations of arsenic in caribou for the Project Case were predicted based upon the predicted concentrations of arsenic in soil and a tissue uptake factors obtained from the scientific literature. The concentrations of arsenic in caribou tissue were 0.3 mg/kg for the Base Case at all locations and 0.56 mg/kg at the MPOI, 0.45 mg/kg at the Worker Camp, and 0.31 mg/kg at the three culturally significant locations (Marian River, Hislop Lake, and Bea Lake). That is, caribou concentrations increase by a factor of 2 at the MPOI and by 50% at the Worker Camp, and remain essentially unchanged at the culturally significant locations. The predictive modeling assumed that caribou would spend all of their time at any of these locations, which is overly conservative and unrealistic, particularly for the MPOI and Worker Camp locations because no suitable food sources or habitat are available to the caribou in these locations. It is more likely that caribou would only be present at the MPOI location on a transient basis, and not present at the Worker Camp. Therefore, the predictions of caribou tissue during the Project Case were considered to be conservative for the MPOI and Worker Camp locations. The predicted change in caribou tissue concentrations for the culturally significant locations is considered to be the more likely result and therefore, negligible impacts to caribou tissue were expected as a result of the NICO Project. Concentrations of arsenic in fish were predicted based upon site-specific water quality and fish tissue data available for Nico Lake, Peanut Lake, and Burke Lake. A comparison of baseline fish tissue data to baseline water quality data was carried out to generate a fish uptake factor of 59.7 (i.e., for every 1 mg/L of arsenic in water, the concentration in fish tissue would be 59.7 mg/kg). This uptake factor incorporated both fish muscle and liver data, and there was a small difference between the UFs derived for each tissue type (muscle = 67.8, liver = 51.6). Water-to-fish BCF values from the literature are somewhat higher: 280 from SRS (1999), and values of 44, 100, and 333 as cited in US EPA (1999). Given that the fish UF was site-specific, it is considered to be a reasonable predictor of fish tissue concentrations. Using the site-specific BCF, the predicted fish tissue concentrations of arsenic used in the RA were 2.63 mg/kg for the MPOI location (compared to the measured baseline of 0.392 mg/kg), 0.048 mg/kg for the Worker Camp location (value remained unchanged as a result of the Project), and 0.103 mg/kg for the culturally significant locations of Marian River, Hislop Lake and Bea Lake (compared to the measured baseline concentration of 0.036 mg/kg based upon the Marian River).

Table 4.6-22: Magnitude of Effects Assessment for Arsenic (Chronic) (continued)

Analysis Criteria	Discussion
Conservatism and uncertainty in receptor exposure assumptions	Receptors at the MPOI were considered to be present at that location for 24 hours per day for 30 days per year throughout their lives, while the likelihood of a receptor remaining at this location is quite low given that it is not associated with any culturally significant or traditional uses.
Conservatism in the toxicity reference values	***Non-Cancer Effects:*** The Health Canada oral RfD is based upon an epidemiological study wherein a NOAEL and a LOAEL were observed. An uncertainty factor of 3 was applied to the NOAEL for insufficient data on reproductive toxicity. The RIVM tolerable concentration in air (TCA) of 1 µg/m³ is based on the trivalent form of arsenic, and is derived from the LOAEC of 10 µg/m^3 for lung cancer in humans. An uncertainty factor of 10 was applied for human variation in susceptibility. ***Cancer Effects:*** The Health Canada Oral Slope Factor (1.8 mg/kg-day) is based skin cancer from a cohort of 40,421 individuals in 37 villages in Taiwan principally exposed via drinking water. The data was US EPA model adjusted to take into account for rates of skin cancer and ingestion rates of Canadians. No documentation is available. The Health Canada Inhalation Unit Risk (0.0064 µg/m³) is based on an epidemiological study of lung cancer in occupationally exposed workers, in which the TC_{05} was 7.83 µg/m³. No uncertainty or modifying factors were applied.
Potential chronic health effects	***Non-Cancer Effects:*** Blackfoot disease (the loss of circulation in the fingers and toes) is the primary effect of chronic arsenic exposure on the cardiovascular system. Peripheral neuropathy, characterized by numbness in hands and feet, is the most common neurological side effect of chronic 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. There is some evidence that arsenic may cause developmental effects. 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. ***Cancer Effects:*** 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.

Table 4.6-22: Magnitude of Effects Assessment for Arsenic (Chronic) (continued)

Analysis Criteria	Discussion
Magnitude of effect	The predicted concentrations of arsenic in soil, water, and air resulted in total HQs and ILCRs greater than the target risk levels. There is a notable change between Baseline Case and Project Case risk estimates and there were several conservative assumptions incorporated into the predictive modelling. The TRVs used to assess potential risks were associated with a low level of uncertainty, while the air modeling results are considered to provide a conservative estimate of exposure. The elevated risks were associated with berry ingestion at the MPOI location, which was evaluated as a worst-case scenario given that there are no known culturally significant uses at this location. Risk levels estimated at the known culturally significant locations of Marian River, Hislop Lake and Bea Lake were much lower and associated with negligible risks. As a result, the overall magnitude of effect is considered to be negligible to low. Specifically for the ingestion of fish and ingestion of caribou pathways, which are of significant interest for native populations in the north, the incremental changes between baseline and Project cases are negligible, indicating that changes to tissue concentrations of arsenic in fish and caribou are not anticipated to have a significant effect on health.

HQ = hazard quotient; HHRA = human health risk assessment; MPOI = Maximum Point of Impingement; mg/L = milligram per litre; mg/kg = milligram per kilogram

5.0 SUMMARY OF HUMAN HEALTH RESULTS AND CONCLUSIONS

5.1 Acute Air Quality Risk Assessment

The acute air quality risk assessment indicated that predicted acute health effects due to short-term exposure to COPCs in air were negligible to low. Air quality predictions were provided for the MPOI, the Worker Camp, Marian River, Hislop Lake, Bea Lake, Gamètì, Whatì, Wekweetì, Behchokö, and Yellowknife. The 1-hour and 24-hour peak concentrations of the chemicals that are expected to be associated with emissions due to the NICO Project were compared to acute toxicity thresholds at each of these locations. While some peak concentrations exceeded the acute toxicity thresholds, the infrequency of exceedances and the conservatism in the air modelling indicated that the overall magnitude of potential acute health effects would be negligible for most COPCs. For arsenic, there were several exceedances of the 1-hour and 24-hour thresholds based upon the predictive air modelling carried out in support of the RA. However, there was a high degree of uncertainty associated with the air predictions given that no dust suppression was considered for the winter months, even though the ground would be frozen and covered in snow, resulting in negligible dust generation. As a result, the magnitude of effects due to arsenic is considered to be low.

5.2 Chronic Air Quality Risk Assessment

The chronic air quality risk assessment indicated that predicted chronic health effects due to long-term exposure to COPCs in air were negligible. Air quality predictions were provided for all of the aforementioned receptor locations for the peak annual average concentrations of the chemicals associated with emissions due to the NICO Project. These concentrations were assessed in terms of exposure doses and compared to chronic toxicity thresholds. All estimated HQs and ILCRs were negligible and the overall magnitude of potential chronic health effects would be negligible.

5.3 Particulate Matter Risk Assessment

The particulate matter risk assessment indicated that predicted health effects due to chronic exposure to particulate matter in air were negligible. The predicted 24-hour peak PM_{10} and $PM_{2.5}$ concentrations were compared to the Canada-Wide Standards and Reference Levels and exceedances were noted. These peak concentrations were evaluated in terms of the Federal-Provincial Working Group on Air Quality Objectives and Guidelines SUM15 and SUM25 approaches. These methods consider the incremental increases of particulate matter and the associated increased rates of health effects per increment. Fewer than 1 case per million population was estimated for each potential health effect, indicating negligible potential chronic health effects due to particulate matter.

5.4 Multi-Media Risk Assessment

As discussed in the magnitude of effects section above, the incremental changes to concentrations of arsenic in air, soil, and water were considered to result in negligible to low changes to human health for the receptors assessed in the Multi-Media Risk Assessment. Conservative assumptions were incorporated into the modelling, including the assumption that the annual average concentrations predicted for the highest emissions year out of the 18 years of operations phase would be present at those levels throughout the receptor's lifespan (i.e., 75 years for the resident and 55 years for the worker [adult life stage only]). In reality, much lower exposure doses would be expected during the other 17 years of operations, the other phases of the NICO Project, and when the NICO Project is in the post-closure phase and ongoing emissions are not occurring. Considering that the highest emissions year is ongoing throughout the lifespan of a given receptor is overly conservative and results in

overestimates of exposure and risk. Second, assuming that a recreational receptor is present at the MPOI location for 30 days per year is expected to be an overestimate; no culturally significant or traditional uses are known to be present proximal to the MPOI location and it is much more likely that receptors would be only transiently exposed to the MPOI. The MPOI location is likely more representative of the worst-case conditions that an on-site worker could be exposed to while not at the on-site worker camp. Therefore, the MPOI assessment for the recreational user is considered to be overly conservative.

The TRVs for arsenic were considered to provide reasonable predictions of levels at which health effects may occur. This substance has been widely studied by several agencies including Health Canada and its associated TRVs are considered to be representative of the best current science for arsenic. As a result, the TRVs are not associated with a high level of uncertainty or conservatism.

The estimated levels of exposure and risk for the recreational user were considered to be conservative taking all potential contributing factors into account. The baseline risk levels for arsenic were greater than the target risk levels, which was mainly attributable to elevated background concentrations of arsenic in the region. The mean baseline concentration of arsenic in soil was used in the assessment to maintain consistency amongst the human health and wildlife assessment and to maintain a conservative approach. It should be noted that the median concentration is approximately one order of magnitude lower than the mean and should the median be used in the RA, the magnitude of risks due to pathways affected by soil concentrations would decrease. However, it remains that the incremental changes in arsenic concentrations due to the NICO Project were negligible to low and are not expected to be associated with changes to human health.

Specifically for the ingestion of fish and ingestion of caribou pathways, which are of significant interest for First Nation populations in the north; the predicted HQs and ILCRs for fish ingestion are less than the target risk levels (5.3E-06) and the predicted HQs and ILCRs for caribou ingestion, while slightly greater than target risk levels at 8.5E-05, do not change significantly from baseline. Given that the incremental changes between Baseline and Project cases are negligible, indicating that changes to tissue concentrations of arsenic in fish and caribou are not anticipated to have a significant effect on human health.

With respect to the cumulative assessment, the current assessment was carried out for the operations year with the highest emissions and it was assumed that this level of exposure would persist throughout a receptor's lifespan. The rates of exposure during the other years of operations, other phases of the NICO Project, and when the NICO Project is concluded, would be expected to be much lower. Therefore, any potential contribution to exposure due to current or foreseeable projects is expected to be well within the exposure doses predicted in this assessment. As a result, changes in health due to the Project in addition to other developments were also considered to be negligible.

5.5 Conclusions

Overall, negligible to low acute and chronic health effects are expected as a result of the predictive modelling and conservative assumptions carried out as part of the HHRA. It is emphasized that the baseline risk levels for arsenic were greater than the target risk levels, which was mainly attributable to elevated background concentrations of arsenic in the region. The incremental changes to the estimated risk levels as a result of the NICO Project were negligible to low, indicating that the NICO Project would not be expected to be associated with changes to human health. However, it is recommended that an ongoing environmental monitoring program include air quality, water quality, soil quality, and fish tissue concentrations.

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7.0 ACRONYMS AND ABBREVIATIONS

%	percent
<	less than
>	more than
≤	less than or equal to
µg/g	micrograms per gram
µg/L	micrograms per litre
mg/kg	milligrams per kilogram
mg/L	milligrams per litre
$CaCO_3$	Calcium carbonate
CCC	Criterion Continuous Concentration
CCME	Canadian Council of Ministers of the Environment
CDF	Co-Disposal Facility
CoPC	Chemicals of Potential Concern
CSM	Conceptual Site Model
DAR	Developer's Assessment Report
EDI	Estimated Daily Intake
e.g.	For example (from Latin exempli gratia)
ETF	Effluent Treatment Facility
et al.	and others (from Latin et alia)
Fortune	Fortune Minerals Limited
GNWT	Government of the Northwest Territories
HHRA	Human Health Risk Assessment
HQ	Hazard Quotient
i.e.	that is (from Latin id est)
ILCR	Incremental Lifetime Cancer Risks
KLOI	Key Lines of Inquiry
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LSA	Local Study Area
MPOI	Maximum Point of Impingement
MVRB	Mackenzie Valley Review Board
NICO Project	NICO Cobalt-Gold-Copper-Bismuth Project
NO_2	Nitrogen dioxide
NOEC	No Observed Effect Concentration
NWT	Northwest Territories

the Plant	Mineral Process Plant
PAH	Polycyclic Aromatic Hydrocarbons
RA	Risk Assessment
RfC	Reference Concentration
RfD	Reference Dose
RSA	Regional Study Area
SCP	Seepage Collection Pond
SF	Slope Factors
SO_2	Sulphur dioxide
SSWQO	Site-specific Water Quality Objective
STP	Sewage Treatment Plant
TOR	Terms of Reference
TRV	Toxicity Reference Value
US EPA	United States Environmental Protection Agency
VOC	Volatile Organic Compounds

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APPENDIX A

Screening Tables for the Acute and Chronic Air Quality Risk Assessments

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
CAC								
Carbon monoxide	n/a	36,200	23,000	15,000	30,000	**15,000**	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available. The CalEPA OEHHA threshold is based on effects of angina in people with known cardiovascular diseases that are exercising heavily. The AAAQO threshold is based on the oxygen carrying capacity of blood and is adopted from the National Ambient Air Quality Objective (NAAQO) The WHO threshold is based on the Coburn-Foster-Kane exponential equation, which takes into account all the known physiological variables affecting carbon monoxide uptake. The threshold was determined so the carboxyhaemoglobin level of 2.5% is not exceeded, even when a subject engages in light or moderate exercise. The GNWT threshold is adopted from the NAAQO
Hydrogen Chloride	n/a	n/a	2,100	**75**	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on upper respiratory system symptoms of sore throat; nasal discharge. The AAAQO threshold is adopted from Texas
Hydrogen Fluoride	n/a	n/a	240	**4.9**	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on irritation to the eyes, nose, and throat. The AAAQO threshold is adopted from Texas
Hydrogen Sulphide	n/a	n/a	42	14	n/a	**14**	n/a	The CalEPA OEHHA threshold is based on physiological responses to odor, headaches, and nausea. The AAAQO threshold is based on odour perception. The GNWT threshold is adopted from the AAAQO
Nitrogen dioxide	n/a	400	470	400	**200**	400	n/a	The OMEE threshold is based on health effects, but supporting documentation is not available. The CalEPA OEHHA threshold is based on increased airway reactivity in asthmatics. The AAAQO is based on the NAAQO derived from a maximum acceptable limit at which odour will be perceived. For NO_2 (a criteria pollutant), the AAAQO, which is based on the NAAQO, was selected. The WHO threshold is based on an increase in bronchial responsiveness in asthmatics. The GNWT Threshold is based on the NAAQO
Nitric Oxide	n/a	n/a	n/a	n/a	n/a	n/a	**300 (1000) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Nitrogen Oxide	n/a	**500**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Sulphur dioxide	n/a	690	660	450	n/a	**450**	n/a	The OMEE threshold is based on health effects, but supporting documentation is not available. The CalEPA OEHHA threshold is based on impairment of airway function (bronchoconstriction) especially in asthmatics. The AAAQO is based on the NAAQO derived from a maximum acceptable limit that will minimize the effects of pulmonary function. The GNWT threshold is based on the NAAQO derived from a maximum acceptable limit that will minimize the effects of pulmonary function

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Green House Gases (GHGs)								
Carbon Dioxide	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available
Methane	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available
Nitrous Oxide	n/a	n/a	n/a	n/a	n/a	n/a	**4500 (15000) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Particulate Matter								
Total suspended Partciulates	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available
Particulates <10 microns (PM10)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available
Particulates <2.5 microns (PM2.5)	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available
Volatile Organic Compounds (VOCs)								
Acetone	n/a	n/a	n/a	**5,900**	n/a	n/a	5,900 (19000) (TCEQ)	The AAAQO is based on the TCEQ value. The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Acrolein	6.9 [(j)]	n/a	**2.5**	n/a	n/a	n/a	n/a	The ATSDR threshold was based on the LOAEL for a decrease in respiratory rate and nose and throat irritation in a study of human volunteers. The LOAEL was corrected for intermittent exposure and an uncertainty factor was applied. The CalEPA OEHHA threshold was based on a geometric mean of two reference exposure levels (2.3 µg/m^3 and2.7 µg/m3) from two studies for subjective ocular irritation in human volunteers.
Aldehydes (surrogate: acetaldehyde)	n/a	n/a	470	**90**	n/a	n/a	n/a	Acetaldehyde was used as a surrogate for aldehydes. The CalEPA OEHHA threshold is based on a LOAEL from a human study. The critical effects of the study include sensory irritation in bronchi, eyes, nose and throat. For the AAAQO, Acetaldehyde was used as a surrogate for aldehyde and is adopted from Texas.
Benzene	n/a	n/a	1,300	30	n/a	n/a	**170 (560) (TCEQ)**	The CalEPA OEHHA threshold is based on health effects (reproductive and developmental toxicity). The TCEQ threshold is based on hematotoxic effects (depressed peripheral lymphocytes and depressed mitogen-induced blastogenesis of femoral B-lymphocytes in male mice). A cumulative uncertainty factor of 90 was applied (3 for interspecies variation, 10 for intraspecies variation, 3 for extrapolation from a LOAEL to a NOAEL, and 1 for database uncertainty). The effects screening level was determined based on a target hazard quotient of 0.3. The AAAQO threshold was adopted from the previous TCEQ value; the TCEQ threshold has since been updated; however, Alberta has not yet adopted the updated value. The AAAQO of 30 µg/m^3 for benzene is based on the former TCEQ health –based short-term ESL. The value was derived by applying a factor of ten to the TCEQ long-term (annual) ESL of 3 µg/m^3.
1,3-Butadiene	n/a	n/a	n/a	n/a	n/a	n/a	**510 (1700) (TCEQ)**	The TCEQ threshold is based on an odour endpoint, as a value based on a health endpoint was not available.
C_2-C_8 aliphatics (surrogate: cyclohex	n/a	n/a	n/a	n/a	n/a	n/a	**3,400 (11000) (TCEQ)**	Cyclohexane was used as a surrogate for the C_2-C_8 aliphatics. The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
C_9-C_{16} aliphatics (surrogate: decane)	n/a	**60,000**	n/a	n/a	n/a	n/a	10,000 (33000) (TCEQ)	Decane was used as a surrogate for the C_9-C_{16} aliphatics. The OMEE threshold for *n*-decane is based on endpoints of health and odour; supporting documentation was not available. The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
C_{16+} aliphatics (surrogate: decane)	n/a	**60,000**	n/a	n/a	n/a	n/a	10,000 (33000) (TCEQ)	No 1-hr thresholds were available for C_{16+} aliphatics. The 1-hr threshold for C_9-C_{16} aliphatics which is based on decane was conservatively used in the risk assessment (60,000 µg/m^3).
C_9-C_{16} aromatics (ethylbenzene)	n/a	n/a	n/a	**2,000**	n/a	n/a	2,000 (6600) (TCEQ)	Ethylbenzene was used as a surrogate for the C_9-C_{16} aromatics. The TCEQ threshold is based on an odour endpoint; a health endpoint was not available. This screening level is currently under review. The AAAQO threshold was adopted from the TCEQ threshold.
Ethylbenzene	n/a	n/a	n/a	**2,000**	n/a	n/a	2,000 (6600) (TCEQ)	The TCEQ threshold is based on an odour endpoint; a health endpoint was not available. This screening level is currently under review. The AAAQO threshold was adopted from the TCEQ threshold.
Chlorobenzenes	n/a	n/a	n/a	n/a	n/a	n/a	**460 (1500) (TCEQ)**	The TCEQ limit for Chlorobenzene is based on a health endpoint, but supporting documentation is not available.
Chloromethane	**1000 (0.5 ppm)**	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold was available, so the 24 hour threshold was adopted. The ATSDR MRL is based on motor coordination and damages to cerebellar granule cells.
Dichloromethane	n/a	n/a	n/a	n/a	n/a	n/a	**260 (860) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Formaldehyde	n/a	n/a	**55**	65	100 (30 Minutes	n/a	n/a	The CalEPA OEHHA threshold was based on the benchmark concentration using the dose-response for eye irritation in humans; an intraspecies uncertainty factor of 10 was applied to account for potential asthma exacerbation. The AAAQO threshold is based on respiratory effects in humans. The WHO guideline is based on the lowest concentration that has been associated with nose and throat irritation in humans. This concentration would also be associated with negligible risk of upper respiratory tract cancer in humans. The WHO also reports a detection threshold of 30 to 600 µg/m^3.
Ketones (surrogate: Methyl Ethyl Ket	n/a	n/a	**13,000**	n/a	n/a	n/a	n/a	The CalEPA OEHHA threshold was based on the LOAEL for eye, nose, and throat irritation in humans. An uncertainty factor of 60 was applied; 6 for use of a LOAEL and 10 for sensitive individuals.
Styrenes	n/a	n/a	21,000	**215**	70 (30 min)	n/a	n/a	The CalEPA OEHHA threshold is based on eye and upper respiratory irritation The AAAQO threshold is adopted from Texas The WHO guideline is based on the odour detection limit for a 30 minute average.
Toluene	n/a	n/a	37,000	**1,880**	n/a	n/a	640 (odour), 4,500 (health) (TCEQ)	The CalEPA OEHHA threshold is based on a LOAEL (100 ppm) for headaches, dizziness, and slight eye and nose irritation in human males exposed for 6 hrs. The LOAEL was extrapolated to represent 1 hr exposure (98 ppm) and an uncertainty factor of 10 was applied for intraspecies differences. The TCEQ threshold for toluene is based on an odour endpoint. There is also a health-based threshold (4,500 µg/m^3; at a target HQ = 0.3) based on eye and nose irritation, headaches, dizziness and intoxication in humans. An uncertainty factor of 10 was applied for intraspecies variability to account for sensitive subpopulations. The AAAQO threshold was adopted from Texas. The Texas threshold has been updated; however, Alberta has not yet adopted the updated value.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
1,1,1-Trichloroethane	n/a	n/a	n/a	n/a	n/a	n/a	**10800 (36000) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Trimethylbenzenes	n/a	n/a	n/a	n/a	n/a	n/a	**1,250 (4160) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Vinyl Chloride	n/a	n/a	180,000	**130**	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on mild headache and dryness of eyes and nose in healthy humans. The AAAQO threshold is adopted from Texas
Xylenes	n/a	3,000 (10 min)	22,000	**2,300**	n/a	n/a	n/a	The OMEE has an odour-based threshold of 3,000 µg/m^3 for xylenes; however, Alberta has not yet adopted the updated value. The CalEPA OEHHA threshold is based on a LOAEL (198 ppm) for eye, nose and throat irritation in humans exposed for 30 minutes. The LOAEL was extrapolated to 1 hour (50 ppm) and an uncertainty factor of 10 was applied for intraspecies differences. The AAAQO threshold was adopted from Ontario which was based on an odour threshold.
Dioxins/Furans								
Chlorinated dibenzo-p-dioxins (CDDs	n/a	**0.000015 (TEQ/m3) (30 min)**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Polycyclic Aromatic Hydrocarbons								
Acenaphthene	n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Acenaphthylene	n/a	**10.5**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a median jurisdictional limit (1/2 hour averaging time).
Anthracene	n/a	**0.6**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a median jurisdictional limit (1/2 hour averaging time).
Benzo(a)anthracene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Benzo(a)pyrene	n/a	**0.0033**	n/a	n/a	n/a	n/a	n/a	The OMEE point of impingement guideline is for a single facility and is based on a health endpoint with1/2 hour averaging time. Supporting documentation is not available.
Benzo(b)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Benzo(b+k)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Benzo(g,h,i)perylene	n/a	**3.6**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a jurisdictional limit (1/2 averaging time).
Benzo(k)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Chrysene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Dibenzo(a.h)anthracene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Fluoranthene	n/a	**420**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a median jurisdictional limit (1/2 hour averaging time).
Fluorene	n/a	n/a	n/a	n/a	n/a	n/a	**10 (33) (TCEQ)**	The TCEQ threshold for fluorene is based on a health endpoint, but supporting documentation is not available.
Indeno(1,2,3-cd)pyrene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
2-Methylnaphthalene	n/a	**30**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a jurisdictional limit (1/2 hour averaging time).
Naphthalene	n/a	**22.5**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold is available. The OMEE 24-hr threshold was conservatively adopted and is based on health, but supporting documentation is not available.
Phenanthrene	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Pyrene	n/a	**0.6**	n/a	n/a	n/a	n/a	n/a	The OMEE screening level is a median jurisdictional limit (1/2 hour averaging time).
Metals								
Aluminum	n/a	**120**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold was available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold for aluminum oxide is based on particulate matter, but supporting documentation is not available.
Antimony	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Arsenic	n/a	n/a	0.2	**0.1**	n/a	n/a	0.1 (0.33) (TCEQ)	The CalEPA OEHHA threshold is based on a LOAEL for decreased fetal weight in mice following maternal inhalation exposure. An uncertainty factor of 1,000 was applied: 10 for extrapolation from animals to humans, 10 for sensitive individuals and 10 for use of a LOAEL. The TCEQ threshold for arsenic is based decreased fetal body weights of maternally exposed rats to arsenic trioxide. The LOAEL was 2900 ug/m^3 and the NOAEL was 260 ug/m^3. A POD_{HEC} was derived and uncertainty factors were applied (3 for extrapolation from animals to humans, 10 for database uncertainty, 10 to acocunt for potential sensitive human subpopulations). to derive an ReV (Reference Value) The ReV was then adjusted for arsenic. This value is currently under review The AAAQO threshold was adopted from Texas.
Barium	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Beryllium	n/a	**0.01**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold was available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold is based on a health endpoint, but supporting documentation is not available.
Bismuth	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Boron	n/a	**120**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold was available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold is based on a particulate endpoint; a threshold based on a health endpoint is not available. Supporting documentation is not available.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Bromine	n/a	n/a	n/a	n/a	n/a	n/a	**7 (23) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Cadmium	n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Calcium	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Chromium (III)[h]	n/a	n/a	n/a	1	n/a	n/a	**3.6 (12) (TCEQ)**	The TCEQ threshold applies to all chromium compounds except hexavalent compounds. The threshold is based on a health endpoint. The critical effect is increased precursor enzymes that are early indicators of lung damage. The AAAQO threshold was adopted from the previous TCEQ value.
Chromium (VI)	n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available. This threshold is currently under review.
Cobalt	n/a	n/a	n/a	n/a	n/a	n/a	**0.2 (0.67) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Copper	n/a	n/a	**100**	n/a	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on the NOAEL for metal fume fever in human workers exposed to copper dust. An uncertainty factor of 10 was applied to the NOAEL for sensitive individuals.
Gallium	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 1-hr thresholds are available.
Indium	n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Iron	n/a	n/a	n/a	n/a	n/a	n/a	**10 (33) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Lanthanum	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available. Lanthanum oxide was used
Lead	n/a	n/a	n/a	**1.5**	n/a	n/a	0.15 (quarterly average) 1.5 (three-month)	Texas defers to the US National Ambient Air Quality Standards for lead (1.5 µg/m^3 as the quarterly average primary standard, and 0.15 µg/m^3 as the rolling three month average primary standard). Alberta adopted the quarterly average primary standard from TCEQ.
Lithium	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available. Lithium chloride was used
Manganese	n/a	n/a	n/a	**2**	n/a	n/a	2 (6.7) (TCEQ)	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available. Alberta adopted the quarterly average primary standard from TCEQ.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Magnesium	n/a	n/a	n/a	n/a	n/a	n/a	**100 (330) (TCEQ)**	The TCEQ limit for magnesium chloride is based on a health endpoint, but supporting documentation is not available.
Mercury	n/a	n/a	**0.6**	n/a	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on nervous system and developmental effects in offspring following maternal exposure to mercury vapour during pregnancy.
Molybdenum	n/a	**120**	n/a	n/a	n/a	n/a	**30 (100) (TCEQ)**	No 1-hr threshold available. The 24-hr threshold was conservatively used. The OMEE threshold is based on particulate endpoint, but supporting documentation is not available.
Nickel	n/a	n/a	6	6	n/a	n/a	**0.15 (0.5) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available. The CalEPA OEHHA threshold is based on a LOAEL (67 µg/m^3) for respiratory and immune system effects noted in metal plating workers with occupational asthma exposed to nickel as $NiSO_4$ for 30 minutes. The LOAEL was converted to 1-hr exposure and a factor of 10 was applied for LOAEL uncertainty. The AAAQO threshold was adopted from California.
Palladium	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Phosphorus	n/a	n/a	n/a	n/a	n/a	n/a	**10 (33) (TCEQ)**	The TCEQ limit for phosphorus trichloride is based on a health endpoint, but supporting documentation is not available.
Potassium	n/a	n/a	n/a	n/a	n/a	n/a	**20 (66) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Rubidum	n/a	n/a	n/a	n/a	n/a	n/a	**25 (83) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Selenium	n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.7) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Silicon	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Silver	n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33) (TCEQ)**	The TCEQ threshold is based on a health endpoint, but supporting documentation is not available.
Sodium	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit for sodium chloride is based onparticulate matter, but supporting documentation is not available.
Strontium	n/a	**120**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold is based on particulate endpoint, but supporting documentation is not available.
Thallium	n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Tin	n/a	**10**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold is based on a health endpoint, but supporting documentation is not available.

Table A.1: Selection of Acute (1-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Titanium	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on particulate matter, but supporting documentation is not available.
Tungsten	n/a	n/a	n/a	n/a	n/a	n/a	**10 (33) (TCEQ)**	The TCEQ limit (soluble compounds) is based on particulate matter, but supporting documentation is not available.
Uranium	n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6) (TCEQ)**	The TCEQ threshold for soluble uranium is based on a health endpoint, however supporting documentation is not available.
Vanadium	n/a	n/a	**30**	n/a	n/a	n/a	n/a	The CalEPA OEHHA threshold is based on eye irritation and coughing and increased mucus production in humans exposed to vanadium pentoxide. An uncertainty factor of 10 was applied to account for intraspecies variation.
Yttrium	n/a	n/a	n/a	n/a	n/a	n/a	**10 (33) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.
Zinc	n/a	**120**	n/a	n/a	n/a	n/a	n/a	No 1-hr threshold available. The 24-hr threshold was conservatively used. The OMEE 24-hr threshold is based on particulate endpoint, but supporting documentation is not available.
Zirconium	n/a	n/a	n/a	n/a	n/a	n/a	**50 (160) (TCEQ)**	The TCEQ limit is based on a health endpoint, but supporting documentation is not available.

(a) Agency for Toxic Substances & Disease Registry (ATSDR 2010, internet site).

(b) Ontario Ministry of Energy and the Environment (OMEE 2008).

(c) Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2009, internet site).

(d) Alberta Ambient Air Quality Objectives (AAAQO; AENV 2009, internet site).

(e) World Health Organization (WHO 2000, 2005).

(f) NWT Ambient Air Quality Standards (GNWT; NWT ENR 2005, internet site).

(g) Texas Commission on Environmental Quality (TCEQ 2010, internet site). Values for TCEQ were only provided where values from the preferred sources were not available. For non-carcinogens, the screening level derived by TCEQ was based on a HQ=0.3. Therefore, the values in brackets represent the screening level adjusted to a HQ=1 for non-carcinogens.

(h) The chromium threshold is based upon divalent and trivalent chromium species.

n/a = Not available.

Note: Shaded and bold acute thresholds were used in the risk assessment. See Section 2.1 for a discussion on the hierarchy for selecting the acute thresholds.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Particulate Matter								
Total Suspended Particulate (TSP)	n/a	120	n/a	100	n/a	**120**	120	The OMEE threshold is based on visibility, however supporting documentation in not available. The AENV threshold is a health-based standard but supporting documentation is not available. The Other value represents the NAAQO maximum desirable average concentration over a 24-hour period The GNWT threshold bas based on the NAAQO maximum desirable level
Particulate Matter (PM10)	n/a	n/a	n/a	**30**	50	n/a	n/a	The AAAQO is the Canada wide standard.
Particulate Matter (PM2.5)	n/a	30	n/a	30	25	**30**	n/a	The OMEE threshold is adopted from the health-based Canada Wide Standard, which is intended to be protective of respiratory effects. The NWT threshold is also adopted from the Canada-Wide Standard, which is based upon respiratory and cardiovascular effects in humans. The GNWT threshold is adopted from the Canada wide standard The WHO threshold is based on multi-city studies conducted in Europe and the United States reporting short-term mortality effects.
Acid Gases								
Carbon monoxide	n/a	15700 (8 Hours)	n/a	6,000 (8 hours)	10,000 (8 hours)	**6000 (8 Hours) (5000 ppbv)**	6000 (8 Hours)	The OMEE threshold is based on a health endpoint, and 8 hours of exposure, but supporting documentation is not available. Supporting documentation for the 8 hour AAAQO for carbon monoxide is not available. The WHO threshold is a time-weighted average 8 hour exposure determined so that the COHb level of 2.5% is not exceeded (threshold for human health effect). The GNWT threshold is adopted from the NAAQO The other value is the NAAQO determined from a 8 hour exposure period
Hydrogen chloride	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Hydrogen fluoride	**16 (0.02 ppm)**	n/a	n/a	n/a	n/a	n/a	8.7 (29) (TCEQ)	The ATSDR MRL is based on upper respiratory inflammation in humans. The TCEQ Chronic limit is based on a health endpoint, but supporting documentation is not available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Hydrogen sulphide	97.3 (0.07 ppm)	7	n/a	**4**	150	**4**	n/a	The ATSDR threshold is based on a LOAEL for a 30% alteration in two measures of lung function for 2 out of 10 persons with asthma. The OMEE threshold is based on a health endpoint, but supporting documentation is not available. The AAAQO is based on odour perception. The WHO guideline is based on eye irritation The GNWT threshold is adopted from the NAAQO
Nitric oxide	n/a	n/a	n/a	n/a	n/a	n/a	**30 (100) (TCEQ)**	The TCEQ Chronic limit is based on a health endpoint, but supporting documentation is not available.
Nitrogen dioxide	n/a	200	n/a	200	n/a	**200 (106 ppbv)**	n/a	The OMEE threshold is based on health effects; however, supporting documentation is not available. The AAAQO threshold was adopted from the health-based Canadian National Ambient Air Quality Objective (NAAQO). The GNWT threshold was adopted from the NAAQO
Nitrogen oxide	n/a	**200**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Nitrous dioxide	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Sulphur dioxide	26.2 (0.01 ppm)	275	n/a	125	20	**150 (57 ppbv)**	150	The ATSDR MRL is based on increased airway resistance in asthmatics. The OMEE threshold is based on health and vegetation effects, but supporting documentation is not available. The AAAQO is based on effects on vegetation (begonia, bluegrass, aspen and forests). The WHO (2005) threshold is based on multiple epidemiological studies wherein mortality, morbidity and lung function were adversely affected. Since no consistent value was apparent in the studies, the guideline was set to 20 µg/m^3 to be conservative, though no particular study was used to arrive at this value. The AAAQO value was adopted from the European Union and is based on human health The GNWT Threshold is adopted from the NAAQO maximum desirable average concentration over a 24-hour period. The Other value represents the NAAQO maximum desirable average concentration over a 24-hour period.
Green House Gases (GHGs)								
Carbon dioxide	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available.
Methane	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available.
Nitrous oxide	n/a	**9000**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Volatile Organic Compounds (VOCs)								
Acetone	53233 (26 ppm) (4 hours)	**11880**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on neurobehavioural effects in humans exposed for 4 hours. The OMEE threshold is based on irritation and neurological effects. The OMEE used a weight of evidence approach to select a value of 594 mg/m^3 as the LOAEL to be used as the basis for deriving an AAQC to prevent irritation and neurological effects with exposure to acetone. A total uncertainty factor of 50 was applied to the LOAEL, giving a value of 12,000 µg/m^3 (the 24-hour AAQC). A total uncertainty factor of 50 was applied (a factor of 10 to account for sensitive individuals within the population, and a factor of 5 to account for the extrapolation from a LOAEL to a NOAEL and to account for the lack of toxicological data characterizing the dose-response (for the endpoint of irritation) and chronic effects of acetone).
Acrolein	n/a	**0.08**	0.7 (8 Hours)	n/a	n/a	n/a	n/a	The OMEE derived a threshold based on a LOAEL (920 µg/m^3; nasal cavity effects in rats) from 3 animal studies. The LOAEL was adjusted based on 6 hours daily exposure, 5 days per week and applying a regional gas dose ratio of 0.14 resulting in a LOAEL of 23 µg/m^3. A total uncertainty factor of 300 was applied (3 to extrapolate from a LOAEL to a NOAEL, 3 for interspecies extrapolation, 3 to extrapolate from subchronic to chronic exposure, and 10 for intraspecies variability, to account for sensitive individuals). The CalEPA threshold is based on lesions in the respiratory epithelium, and derived from an 8 hour exposure.
Aldehydes (surrogate: acetaldehyde)	n/a	**500**	300 (8 Hours)	n/a	n/a	n/a	n/a	The OMEE threshold is based on health effects, but supporting documentation is not available. The CalEPA REL for acetaldehyde based on degeneration of the olfactory nasal epithelium (8 hour exposure).
Benzene	**28.71**[h]	n/a	n/a	n/a	n/a	n/a	n/a	The ATSDR minimal risk level was based on the LOAEL for decreased lymphocytes in mice (0.009 ppm). The LOAEL was conservatively adjusted from 6 hrs for 6 days exposure to a 24-hour exposure and an uncertainty factor of 300 was applied; 10 for use of a LOAEL, 3 for extrapolation from animals to humans, and 10 for sensitive individuals.
1,3-Butadiene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hour thresholds were available for 1,3-butadiene. The selected 1-hour threshold was based upon odour because no acute health-based threshold was available, and the chronic threshold was based upon a cancer endpoint, which is not applicable to a 24-hour exposure period.
C_2-C_8 aliphatics (surrogate: cyclohexane)	n/a	**6,100**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold for cyclohexane is based on a health endpoint (developmental effects as reduced offspring weights in laboratory animals). The 24-hour OMEE threshold of 6,100 µg/m^3 is proposed as the new air quality standard for cyclohexane, but it has not yet been finalized.
C_9-C_{16} aliphatics (surrogate: cyclohexane)	n/a	**6,100**	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold for the C_9-C_{16} aliphatic surrogate, decane, was available. The 24-hr threshold for C_2-C_8 aliphatics, cyclohexane, was conservatively used in the risk assessment.
C_{16+} aliphatics (surrogate: cyclohexane)	n/a	**6,100**	n/a	n/a	n/a	n/a	n/a	No 24-hr thresholds were available for the C_{16}+ aliphatics. The 24-hr threshold for C_2-C_8 aliphatics, which was based on cyclohexane, was conservatively used in the risk assessment.
C_9-C_{16} aromatics (surrogate: ethylbenzene	21739 (5 ppm) (8hr/day – 5 days)	**1,000**	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available. The 24-hr threshold for ethylbenzene was conservatively used in the risk assessment. Refer to discussion for ethylbenzene. The ATSDR MRL is based on a BMCL for auditory threshold shifts in rats exposed for 8 hours/day for 5 days.
Chlorobenzenes	n/a	n/a	n/a	n/a	n/a	n/a	**50**	No 24 hour threshold was available, so the chronic threshold was adopted. The U.S. EPA RSL value for chlorobenzene was selected and supporting documentation was available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Chloromethane	**1000 (0.5 ppm)**	n/a	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on motor coordination and damages to cerebellar granule cells.
Dichloromethane	n/a	n/a	n/a	n/a	**450**	n/a	n/a	The WHO threshold is based on health effects and supporting documentation is available.
Ethylbenzene	21739 (5 ppm) (8hr/day – 5 days)	**1,000**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on irritant effects to exposed humans in the workplace. The OMEE threshold for ethylbenzene is based on a health endpoint, but supporting documentation is not available.
Formaldehyde	49.1 (0.04 ppm)	**65**	9 (8 Hour)	n/a	n/a	n/a	n/a	The ATSDR MRL is based on clinical symptoms and nasal alterations in humans The OMEE threshold is based on a health endpoint, but supporting documentation is not available. The CalEPA threshold is based on nasal obstruction and discomfort, lower airway discomfort and eye irritations. It is based on 8 hours of exposure.
Ketones (surrogate: methyl ethyl ketone)	n/a	**1,000**	n/a	n/a	n/a	n/a	n/a	No 24-hr thresholds were available for ketones. The 24-hr threshold for methyl ethyl ketone was conservatively used in the risk assessment The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Styrenes	n/a	**400**	n/a	n/a	n/a	n/a	n/a	The ATSDR is based on alterations in tests of reaction time, memor The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Toluene	3,800	6,700 (odour); 5,000 (health)	n/a	**400**	n/a	n/a	n/a	The ATSDR minimal risk level of 1 ppm (3.8 mg/m3) was based on the NOAEL (10 ppm) for neurological effects in male humans exposed for 6 hours for 4 consecutive days. An uncertainty factor of 10 was applied to account for human variability. ATSDR converted this value to a 24-hr threshold. The OMEE has developed a toluene threshold based on odour (6,700 µg/m^3; geometric mean) and a threshold based on adverse human health effects (5,000 µg/m^3). The OMEE threshold is derived from an average NOAEL (128 mg/m^3) from several (mostly chronic) occupational studies where a deficit in neurological function was the critical effect. The NOAEL was adjusted for working hours per day and 5 days per week. An uncertainty factor of The AAAQO 24-hour threshold was adopted from Michigan and Washington. The Michigan and Washington thresholds have been updated (5,000 µg/m^3); however, Alberta has not yet adopted the updated values
1,1,1-Trichloroethane	**10900 (2 ppm)**	**115,000**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on reduced performance in psychomotor tests in human volunteers. The OMEE threshold for 1,1,1-trichloroethane is health-based, but no supporting documentation was available.
Trimethylbenzenes (surrogate: 1,2,4-trime	n/a	**220**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold for 1,2,3-, 1,2,4- and 1,2,5-trimethylbenzene is based on several subchronic rat studies where central nervous system effects (behavioural effects) were observed. A NOAEL of 123 mg/m^3 was derived and adjusted for continuous time. A safety factor of 100 was applied (3 for interspecies extrapolation, 10 for intraspecies variability and 3 for subchronic to chronic extrapolation) to derive the 24 hour threshold of approximately 220 µg/m^3
Vinyl Chloride	1250 (0.5 ppm)	**1**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL was based on developmental effects on mice. The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
								The OMEE threshold for xylene is based on neurological effects. The California chronic REL of 700 µg/m^3 was used to derive Ontario's health-based 24-hour ambient air quality criteria (AAQC) of 730 µg/m^3. The REL was based on a study where human workers were exposed to an average of 62 mg/m^3 xylene for seven years. Some of the workers experienced eye

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Xylenes	n/a	730	n/a	**700**	n/a	n/a	n/a	The AAAQO threshold was adopted from California. The California REL of 700 µg/m^3 is a chronic value based on occupational inhalation exposure of factory workers to xylenes. The workers experienced a dose related increase in the prevalence of eye irritation, sore throat, floating sensation and poor appetite. A total uncertainty factor of 30 was applied (3 for extrapolation from a LOAEL to a NOAEL and 10 for intraspecies variability).
Dioxins/Furans								
Chlorinated dibenzo-p-dioxins (CCDs)	n/a	**(TEQ/m^3)**	n/a	n/a	n/a	n/a		The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Individual PAHs								
Acenaphthene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available,
Acenaphthylene	n/a	**3.5**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Anthracene	n/a	**0.2**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Benzo(a)anthracene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available,
Benzo(a)pyrene	n/a	**0.0011**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting information is not available.
Benzo(b)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Benzo(g,h,i)perylene	n/a	**1.2**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Benzo(b+k)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Benzo(k)fluoranthene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Chrysene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Dibenzo(a,h)anthracene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Fluoranthene	n/a	**140**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Fluorene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Indeno(1,2,3-cd)pyrene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
2-Methylnaphthalene	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Naphthalene	n/a	**22.5**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting information is not available.
Phenanthrene	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available.
Pyrene	n/a	**0.2**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Metals								
Aluminum	n/a	**120**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold for aluminum oxide is based on a particulate endpoint, but supporting documentation is not available.
Antimony	n/a	**25**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Arsenic	n/a	**0.3**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Barium	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Beryllium	n/a	**0.01**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Bismuth	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ Chronic threshold is based on paritculate matter. Supporting documentation is not available.
Boron	300	**120**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on increased nasal secretions in humans. The OMEE threshold is based on a particulate endpoint; a threshold based on a health endpoint is not available. Supporting documentation is not available.
Bromine	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available
Cadmium	0.03	**0.025**	n/a	n/a	n/a	n/a	n/a	The ATSDR MRL is based on respiratory effects in rats exposed to cadmium oxide. The OMEE threshold is based on kidney effects and carcinogenicity in humans associated with exposure to cadmium and cadmium compounds, and is based on extrapolation to low concentration from occupational exposure studies
Calcium	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ Chronic threshold is based on health effects, but supporting documentation is not available
Chromium (III) [i]	n/a	**1.5**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is for di- and tri-valent chromium, and is based on a health endpoint, but supporting documentation is not available.
Chromium (VI)	n/a	**1.5**	n/a	n/a	n/a	n/a	**0.041 (0.14) (TCEQ)**	No 24 Hour threshold was found for Chromium (VI), so the Chronic Chromium (III) threshold was adopted. The TCEQ value was selected in the absence of other values and is based upon particulate matter with a health endpoint.
Cobalt	n/a	**0.1**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Copper	n/a	**50**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Gallium	n/a	n/a	n/a	n/a	n/a	n/a	n/a	No 24-Hr thresholds are available.
Indium	n/a	**0.4**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available
Iron	n/a	**4**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is for metallic iron and is based on a soiling endpoint; a threshold based on a health endpoint was not available.
Lanthanum	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Lead	n/a	**0.5**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on neurological effects in children. This threshold is based on the Cal/EPA (2001-as cited in OMEE 2008) approach for the derivation of health-based air standards for lead, which is based on the identification of the airborne lead concentration associated with a 5% probability of children in a reference population exceeding a LOAEL.
Lithium	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Magnesium	n/a	**120**	n/a	n/a	n/a	n/a	n/a	No 24-hr threshold was available for magnesium. The 24-hr threshold for magnesium oxide was conservatively used in the risk assessment. The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Manganese	n/a	**2.5**	0.17 (8 Hour)	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available. The CalEPA REL is based on impairment of neurobehavioural function from 8 hours of exposure.
Mercury	n/a	**2**	0.06 (8 Hour)	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available. The CalEPA REL is based on the impairment of neurobehavioural functions from an 8-hour exposure.
Molybdenum	n/a	**120**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on particulate endpoint, but supporting documentation is not available.
Nickel	n/a	**2**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is not based on a health endpoint (it is based on vegetation effects); however, since no other value was available, it was used in the assessment.
Palladium	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Phosphorus	n/a	n/a	n/a	n/a	n/a	n/a	**0.2 (0.66) (TCEQ)**	The TCEQ Chronic value for phosphorus trichloride was selected in the absence of other values.
Potassium	n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.6) (TCEQ)**	The TCEQ Chronic threshold is based on health effects, but supporting documentation is not available
Rubidium	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Selenium	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Silicon	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Silver	n/a	**1**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Sodium	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ Chronic threshold is based onparticulate matter, but supporting documentation is not available
Strontium	n/a	**120**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on particulate endpoint, but supporting documentation is not available.

Table A.2: Selection of Acute (24-hour) Air Thresholds

Parameter	ATSDR[a] [µg/m^3]	OMEE[b] [µg/m^3]	CalEPA OEHHA[c] [µg/m^3]	AENV[d] [µg/m^3]	WHO[e] [µg/m^3]	GNWT[f] [µg/m^3]	Other[g] [µg/m^3]	Basis of Threshold
Thallium	n/a	**0.24**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Tin	n/a	**10**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on a health endpoint, but supporting documentation is not available.
Titanium	n/a	n/a	n/a	n/a	n/a	n/a	**5 (16) (TCEQ)**	The TCEQ Chronic threshold is based on particulate matter. Supporting documentation is not available.
Tungsten	n/a	**4**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Uranium	n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.16) (TCEQ)**	The TCEQ Chronic threshold is based on health effects, but supporting documentation is not available
Vanadium	n/a	2	n/a	n/a	**1**	n/a	n/a	The OMEE threshold is based on a health endpoint (no supporting documentation is available). The WHO threshold is based on a LOAEL (20 µg/m^3) for upper respiratory tract symptoms observed in occupational studies. A protection factor of 20 was applied.
Yttrium	n/a	**2.4**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.
Zinc	n/a	**120**	n/a	n/a	n/a	n/a	n/a	The OMEE threshold is based on particulate endpoint, but supporting documentation is not available.
Zirconium	n/a	**20**	n/a	n/a	n/a	n/a	n/a	The OMEE median jurisdictional limit is based on a health endpoint, but supporting information is not available.

(a) Agency for Toxic Substances & Disease Registry (ATSDR 2010, internet site).

(b) Ontario Ministry of Energy and the Environment (OMEE 2005, 2008).

(c) California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2009, internet site).

(d) Alberta Ambient Air Quality Objectives (AAAQO; AENV 2009, internet site).

(e) World Health Organization (WHO 2000, 2005).

(f) NWT Ambient Air Quality Standards (GNWT; NWT ENR 2005, internet site).

(g) Texas Commission on Environmental Quality (TCEQ 2010, internet site). Values for TCEQ were only provided where values from the preferred sources were not available. For non-carcinogens, the screening level derived by TCEQ was based on a HQ=0.3. Therefore, the values in brackets represent the screening level adjusted to a HQ=1 for non-carcinogens.

(h) The benzene threshold from ATSDR (0.009 ppm) was converted to µg/m^3 using the following conversion: 1 ppm = 3.19 mg/m^3 (ATSDR 2010, internet site).

(i) The chromium threshold from OMEE is based upon divalent and trivalent chromium species.

n/a = Not available.

Notes: Shaded and bold acute thresholds were used in the risk assessment. See Section XX for a discussion on the hierarchy for selecting the acute thresholds.

Table A.3: Screening of Predicted 1-Hour Air Concentrations for the Acute Air Quality Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
Acid/Greenhouse Gases																	
Carbon Monoxide	15000	3.5E+02	3.5E+02	3.5E+02	4.0E+02	4.0E+02	4.0E+02	3.5E+02	3.5E+02	1.4E+03	9.7E+02	1.4E+03	3.5E+02	3.9E+02	No	Yes	No
Nitrogen Dioxide	200	6.3E+00	7.8E+00	6.3E+00	8.3E+01	7.9E+01	8.3E+01	2.7E+01	1.8E+01	**4.7E+02**	**2.0E+02**	**4.7E+02**	2.6E+01	2.8E+01	Yes	Yes	**Yes**
Sulphur Dioxide	450	5.3E-01	5.3E-01	5.2E-01	1.2E+00	1.1E+00	1.0E+00	5.2E-01	5.5E-01	3.2E+00	1.0E+01	1.0E+01	5.7E-01	6.2E-01	No	Yes	No
Volatile Organic Compounds																	
1,1,1-Trichlorethane	36000	1.2E-08	2.5E-08	1.1E-08	2.7E-07	3.2E-07	2.7E-07	1.2E-08	2.7E-08	2.7E-06	3.7E-06	3.7E-06	5.0E-07	5.5E-07	No	Yes	No
1,3-Butadiene	1700	6.2E-05	5.1E-05	5.0E-05	1.2E-03	1.0E-03	8.1E-04	5.9E-05	9.2E-05	7.3E-03	2.3E-02	2.3E-02	1.4E-05	1.5E-05	No	Yes	No
Acetone	5900	4.4E-03	3.6E-03	3.6E-03	8.8E-02	7.4E-02	5.7E-02	4.2E-03	6.5E-03	5.2E-01	1.6E+00	1.6E+00	0.0E+00	0.0E+00	No	Yes	No
Acrolein	2.5	1.5E-03	1.2E-03	1.2E-03	3.0E-02	2.5E-02	1.9E-02	1.4E-03	2.2E-03	1.7E-01	5.5E-01	5.5E-01	1.8E-04	2.0E-04	No	Yes	No
Aldehydes	90	2.0E-02	1.6E-02	1.6E-02	4.0E-01	3.4E-01	2.6E-01	1.9E-02	3.0E-02	2.4E+00	7.4E+00	7.4E+00	5.9E-04	6.5E-04	No	Yes	No
Benzene	560	2.3E-03	2.1E-03	2.0E-03	4.2E-02	4.2E-02	4.3E-02	6.4E-03	5.3E-03	9.7E-01	3.8E-01	9.7E-01	1.8E-02	2.0E-02	No	Yes	No
C16+ Aliphatics	60000	6.9E-04	5.6E-04	5.6E-04	1.4E-02	1.2E-02	8.9E-03	6.6E-04	1.0E-03	8.1E-02	2.5E-01	2.5E-01	0.0E+00	0.0E+00	No	Yes	No
C2-C8 Aliphatics	11000	1.3E-02	1.1E-02	1.1E-02	2.0E-01	1.9E-01	2.0E-01	2.4E-02	2.6E-02	3.6E+00	2.6E+00	3.6E+00	6.5E-02	7.2E-02	No	Yes	No
C9-16 Aliphatics	60000	8.5E-04	7.0E-04	6.9E-04	1.7E-02	1.4E-02	1.1E-02	8.1E-04	1.3E-03	1.0E-01	3.1E-01	3.1E-01	0.0E+00	0.0E+00	No	Yes	No
C9-16 Aromatics	2000	1.2E-03	1.0E-03	9.9E-04	2.5E-02	2.1E-02	1.6E-02	1.2E-03	1.8E-03	1.4E-01	4.5E-01	4.5E-01	0.0E+00	0.0E+00	No	Yes	No
Chlorobenzenes	1500	1.3E-07	2.8E-07	1.2E-07	3.0E-06	3.5E-06	3.0E-06	1.4E-07	3.0E-07	2.9E-05	4.0E-05	4.0E-05	5.5E-06	6.1E-06	No	Yes	No
Chloromethane	1000	3.3E-07	7.0E-07	3.1E-07	7.7E-06	9.0E-06	7.7E-06	3.5E-07	7.6E-07	7.5E-05	1.0E-04	1.0E-04	1.4E-05	1.6E-05	No	Yes	No
Dichloromethane	860	3.7E-07	7.8E-07	3.4E-07	8.6E-06	1.0E-05	8.6E-06	3.9E-07	8.4E-07	8.3E-05	1.1E-04	1.1E-04	1.6E-05	1.7E-05	No	Yes	No
Ethylbenzene	2000	9.5E-05	7.8E-05	7.6E-05	1.9E-03	1.6E-03	1.2E-03	9.0E-05	1.4E-04	1.1E-02	3.5E-02	3.5E-02	2.8E-05	3.0E-05	No	Yes	No
Formaldehyde	55	4.7E-03	3.7E-03	3.8E-03	9.0E-02	7.6E-02	6.0E-02	4.4E-03	7.0E-03	5.3E-01	1.7E+00	1.7E+00	1.8E-03	2.0E-03	No	Yes	No
Ketones	13000	1.5E-03	1.2E-03	1.2E-03	3.0E-02	2.5E-02	1.9E-02	1.4E-03	2.2E-03	1.8E-01	5.6E-01	5.6E-01	0.0E+00	0.0E+00	No	Yes	No
Styrenes	215	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
Toluene	1880	1.6E-03	1.7E-03	1.5E-03	3.1E-02	3.1E-02	2.6E-02	2.6E-03	3.4E-03	3.6E-01	3.1E-01	3.6E-01	1.8E-02	1.9E-02	No	Yes	No
Trimethylbenzenes	4160	2.3E-04	1.9E-04	1.8E-04	4.6E-03	3.8E-03	3.0E-03	2.2E-04	3.4E-04	2.7E-02	8.4E-02	8.4E-02	0.0E+00	0.0E+00	No	Yes	No
Vinyl Chloride	130	1.1E-07	2.4E-07	1.0E-07	2.6E-06	3.0E-06	2.6E-06	1.2E-07	2.6E-07	2.5E-05	3.5E-05	3.5E-05	4.8E-06	5.2E-06	No	Yes	No
Xylenes	2300	1.1E-03	8.8E-04	9.1E-04	1.6E-02	1.4E-02	1.6E-02	1.7E-03	2.0E-03	2.5E-01	2.4E-01	2.5E-01	4.5E-03	5.0E-03	No	Yes	No
Dioxins/Furans																	
Total Dioxins/Furans	1.50E-05	4.7E-12	1.0E-11	4.5E-12	1.0E-10	1.2E-10	1.0E-10	4.7E-12	1.0E-11	1.0E-09	1.4E-09	1.4E-09	9.2E-12	1.0E-11	No	Yes	No
Polyaromatic Hydrocarbons																	
2-Methylnaphthalene	30	1.1E-04	9.0E-05	8.8E-05	2.2E-03	1.9E-03	1.4E-03	1.0E-04	1.6E-04	1.2E-02	4.0E-02	4.0E-02	0.0E+00	0.0E+00	No	Yes	No
Acenapthene	3.3	1.3E-05	1.2E-05	1.1E-05	2.5E-04	2.4E-04	2.5E-04	3.8E-05	3.0E-05	5.8E-03	2.1E-03	5.8E-03	3.6E-05	4.0E-05	No	Yes	No
Acenapthylene	10.5	2.9E-05	2.6E-05	2.5E-05	5.1E-04	5.0E-04	5.1E-04	7.6E-05	6.5E-05	1.2E-02	4.7E-03	1.2E-02	7.2E-05	7.9E-05	No	Yes	No
Anthracene	0.6	5.4E-06	4.5E-06	4.6E-06	8.1E-05	7.9E-05	8.2E-05	1.0E-05	1.1E-05	1.6E-03	1.0E-03	1.6E-03	9.6E-06	1.1E-05	No	Yes	No
Benzo(a)anthracene	1.6	1.8E-06	1.6E-06	1.5E-06	3.3E-05	3.2E-05	3.3E-05	5.1E-06	4.1E-06	7.8E-04	2.9E-04	7.8E-04	4.8E-06	5.3E-06	No	Yes	No
Benzo(a)pyrene	0.0033	9.9E-07	7.8E-07	8.3E-07	1.6E-05	1.3E-05	1.4E-05	1.1E-06	1.7E-06	1.7E-04	2.6E-04	2.6E-04	1.0E-06	1.1E-06	No	Yes	No
Benzo(b+k)fluoranthene	1.6	5.8E-06	4.9E-06	5.0E-06	8.8E-05	8.3E-05	8.8E-05	1.0E-05	1.1E-05	1.6E-03	1.2E-03	1.6E-03	9.5E-06	1.0E-05	No	Yes	No
Benzo(g,h,i)perylene	3.6	1.7E-06	1.4E-06	1.4E-06	2.6E-05	2.3E-05	2.5E-05	2.4E-06	3.1E-06	3.6E-04	4.0E-04	4.0E-04	2.2E-06	2.4E-06	No	Yes	No
Chrysene	1.6	3.9E-06	3.7E-06	3.4E-06	7.8E-05	7.6E-05	8.1E-05	1.3E-05	9.5E-06	1.9E-03	6.5E-04	1.9E-03	1.2E-05	1.3E-05	No	Yes	No
Dibenzo(a,h)anthracene	1.6	8.8E-07	7.3E-07	7.4E-07	1.3E-05	1.2E-05	1.3E-05	1.5E-06	1.7E-06	2.2E-04	1.8E-04	2.2E-04	1.3E-06	1.5E-06	No	Yes	No
Fluoranthene	420	1.5E-05	1.3E-05	1.3E-05	2.4E-04	2.4E-04	2.4E-04	3.4E-05	3.2E-05	5.1E-03	2.6E-03	5.1E-03	3.1E-05	3.4E-05	No	Yes	No
Fluorene	33	4.1E-05	3.7E-05	3.6E-05	7.2E-04	7.0E-04	7.2E-04	1.1E-04	9.2E-05	1.6E-02	6.7E-03	1.6E-02	9.9E-05	1.1E-04	No	Yes	No
Indeno(1,2,3-c,d)pyrene	1.6	7.8E-07	6.8E-07	6.7E-07	1.2E-05	1.2E-05	1.2E-05	1.7E-06	1.6E-06	2.6E-04	1.4E-04	2.6E-04	1.6E-06	1.8E-06	No	Yes	No
Naphthalene	22.5	4.7E-04	4.1E-04	4.0E-04	7.6E-03	7.5E-03	7.5E-03	1.1E-03	1.0E-03	1.6E-01	7.9E-02	1.6E-01	1.0E-03	1.1E-03	No	Yes	No
Phenanthrene	1.6	1.2E-04	1.1E-04	1.0E-04	2.2E-03	2.1E-03	2.2E-03	3.4E-04	2.7E-04	5.1E-02	1.9E-02	5.1E-02	3.2E-04	3.5E-04	No	Yes	No
Pyrene	0.6	1.7E-05	1.4E-05	1.4E-05	2.5E-04	2.4E-04	2.6E-04	3.1E-05	3.4E-05	4.7E-03	3.2E-03	4.7E-03	2.9E-05	3.2E-05	No	Yes	No
Metals																	
aluminum	120	4.3E-02	5.0E-03	1.1E-02	3.7E+00	2.8E+00	1.7E+00	2.5E-02	1.7E-02	6.9E+01	**1.5E+02**	**1.5E+02**	3.8E-04	4.2E-04	Yes	Yes	No [1]
antimony	16	1.4E-05	1.4E-05	7.0E-06	8.4E-04	6.4E-04	4.4E-04	7.7E-06	1.4E-05	1.6E-02	3.0E-02	3.0E-02	6.1E-05	6.7E-05	No	Yes	No
arsenic	0.1	1.3E-03	1.6E-04	3.4E-04	**1.1E-01**	9.1E-02	5.7E-02	7.5E-04	5.6E-04	**2.5E+00**	**3.6E+00**	**3.6E+00**	4.1E-05	4.5E-05	Yes	Yes	**Yes**
barium	16	4.6E-04	5.3E-05	1.1E-04	4.0E-02	3.1E-02	1.8E-02	2.6E-04	1.8E-04	7.4E-01	1.6E+00	1.6E+00	3.0E-06	3.3E-06	No	Yes	No
beryllium	0.01	1.5E-06	1.8E-07	3.8E-07	1.3E-04	1.0E-04	6.0E-05	8.6E-07	6.0E-07	2.5E-03	5.1E-03	5.1E-03	5.4E-09	6.0E-09	No	Yes	No
bismuth	160	5.0E-04	5.9E-05	1.3E-04	4.2E-02	3.3E-02	2.0E-02	2.9E-04	2.0E-04	8.2E-01	1.6E+00	1.6E+00	0.0E+00	0.0E+00	No	Yes	No
boron	120	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
bromine	23	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
cadmium	0.33	6.3E-05	7.2E-05	5.8E-05	2.2E-03	2.0E-03	2.1E-03	3.0E-04	1.5E-04	4.7E-02	1.7E-02	4.7E-02	2.9E-04	3.1E-04	No	Yes	No
calcium	160	1.6E-02	1.8E-03	3.9E-03	1.3E+00	1.0E+00	6.4E-01	8.9E-03	6.3E-03	2.6E+01	4.8E+01	4.8E+01	0.0E+00	0.0E+00	No	Yes	No
chromium	12	5.5E-05	1.9E-05	2.2E-05	3.9E-03	3.0E-03	1.7E-03	5.1E-05	2.6E-05	7.0E-02	1.5E-01	1.5E-01	8.8E-05	9.6E-05	No	Yes	No
chromium 6	0.33	8.6E-09	2.1E-08	7.8E-09	2.8E-07	2.9E-07	2.6E-07	8.2E-09	2.1E-08	2.8E-06	3.7E-06	3.7E-06	4.8E-08	5.3E-08	No	Yes	No
cobalt	0.66	1.7E-04	2.2E-05	5.0E-05	1.4E-02	1.1E-02	7.1E-03	9.7E-05	7.2E-05	3.0E-01	4.5E-01	4.5E-01	4.8E-05	5.2E-05	No	Yes	No

Table A.3: Screening of Predicted 1-Hour Air Concentrations for the Acute Air Quality Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
copper	100	1.4E-04	4.6E-05	5.1E-05	1.0E-02	7.8E-03	4.7E-03	7.8E-05	5.6E-05	1.9E-01	4.0E-01	4.0E-01	1.8E-04	2.0E-04	No	Yes	No
gallium	NG	9.4E-06	9.5E-06	8.3E-06	3.2E-04	2.9E-04	3.0E-04	4.9E-05	2.5E-05	7.8E-03	2.8E-03	7.8E-03	4.8E-05	5.2E-05	No	Yes	No
indium	3.3	5.6E-05	5.7E-05	5.0E-05	1.9E-03	1.7E-03	1.8E-03	2.9E-04	1.5E-04	4.7E-02	1.7E-02	4.7E-02	2.9E-04	3.1E-04	No	Yes	No
iron	33	9.1E-02	1.1E-02	2.3E-02	7.7E+00	6.0E+00	3.6E+00	5.2E-02	3.6E-02	**1.5E+02**	**3.0E+02**	**3.0E+02**	2.4E-04	2.6E-04	Yes	Yes	No [1]
lanthanum	160	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
lead	1.5	2.1E-04	4.0E-04	2.1E-04	1.2E-03	1.4E-03	9.1E-04	9.8E-05	3.4E-04	9.3E-03	1.9E-02	1.9E-02	2.1E-03	2.3E-03	No	Yes	No
lithium	33	1.3E-05	1.5E-06	3.2E-06	1.1E-03	8.6E-04	5.1E-04	7.4E-06	5.1E-06	2.1E-02	4.5E-02	4.5E-02	0.0E+00	0.0E+00	No	Yes	No
magnesium	330	2.0E-02	2.4E-03	5.1E-03	1.8E+00	1.4E+00	8.0E-01	1.2E-02	8.1E-03	3.3E+01	7.0E+01	7.0E+01	0.0E+00	0.0E+00	No	Yes	No
manganese	2	5.1E-04	6.2E-05	1.4E-04	4.1E-02	3.3E-02	2.1E-02	2.9E-04	2.1E-04	8.4E-01	1.6E+00	1.6E+00	4.8E-05	5.2E-05	No	Yes	No
mercury	0.6	5.4E-06	1.1E-05	5.4E-06	7.0E-05	7.9E-05	6.5E-05	2.6E-06	9.0E-06	6.9E-04	9.1E-04	9.1E-04	5.4E-05	5.9E-05	No	Yes	No
molybdenum	120	4.3E-06	5.1E-07	1.1E-06	3.5E-04	2.8E-04	1.7E-04	2.4E-06	1.7E-06	7.0E-03	1.4E-02	1.4E-02	1.5E-07	1.7E-07	No	Yes	No
nickel	6	1.5E-05	1.8E-05	9.3E-06	1.0E-03	8.0E-04	5.0E-04	8.3E-06	1.5E-05	2.0E-02	3.9E-02	3.9E-02	9.2E-05	1.0E-04	No	Yes	No
palladium	160	9.4E-06	9.5E-06	8.3E-06	3.2E-04	2.9E-04	3.0E-04	4.9E-05	2.5E-05	7.8E-03	2.8E-03	7.8E-03	4.8E-05	5.2E-05	No	Yes	No
phosphorus	33	1.8E-04	2.2E-05	5.2E-05	1.6E-02	1.2E-02	6.8E-03	1.0E-04	7.1E-05	2.8E-01	6.2E-01	6.2E-01	4.8E-05	5.2E-05	No	Yes	No
potassium	66	4.3E-02	5.0E-03	1.1E-02	3.8E+00	2.9E+00	1.7E+00	2.5E-02	1.7E-02	**7.0E+01**	**1.5E+02**	**1.5E+02**	0.0E+00	0.0E+00	Yes	Yes	No [1]
rubidium	83	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
selenium	6.6	4.1E-06	6.2E-07	1.2E-06	3.4E-04	2.6E-04	1.5E-04	2.3E-06	1.6E-06	6.2E-03	1.3E-02	1.3E-02	2.4E-06	2.7E-06	No	Yes	No
silicon	160	5.9E-04	6.0E-04	5.2E-04	2.0E-02	1.8E-02	1.9E-02	3.1E-03	1.6E-03	4.9E-01	1.7E-01	4.9E-01	3.0E-03	3.3E-03	No	Yes	No
silver	0.33	9.5E-06	1.0E-05	8.6E-06	3.3E-04	3.0E-04	3.1E-04	4.9E-05	2.5E-05	7.8E-03	2.9E-03	7.8E-03	4.8E-05	5.2E-05	No	Yes	No
sodium	160	4.5E-03	5.3E-04	1.1E-03	3.9E-01	3.0E-01	1.8E-01	2.6E-03	1.8E-03	7.3E+00	1.5E+01	1.5E+01	0.0E+00	0.0E+00	No	Yes	No
strontium	120	1.8E-05	2.1E-06	4.4E-06	1.6E-03	1.2E-03	7.0E-04	1.0E-05	7.0E-06	2.9E-02	6.2E-02	6.2E-02	0.0E+00	0.0E+00	No	Yes	No
thallium	3.3	4.8E-07	5.5E-08	1.2E-07	4.1E-05	3.1E-05	1.9E-05	2.7E-07	1.9E-07	7.6E-04	1.6E-03	1.6E-03	0.0E+00	0.0E+00	No	Yes	No
tin	10	4.8E-06	5.7E-07	1.2E-06	4.1E-04	3.2E-04	1.9E-04	2.8E-06	1.9E-06	7.9E-03	1.6E-02	1.6E-02	0.0E+00	0.0E+00	No	Yes	No
titanium	160	1.4E-03	1.7E-04	3.6E-04	1.2E-01	9.5E-02	5.7E-02	8.2E-04	5.7E-04	2.3E+00	4.9E+00	4.9E+00	0.0E+00	0.0E+00	No	Yes	No
tungsten	33	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
uranium	1.6	5.4E-06	6.3E-07	1.4E-06	4.5E-04	3.6E-04	2.2E-04	3.1E-06	2.2E-06	8.9E-03	1.8E-02	1.8E-02	0.0E+00	0.0E+00	No	Yes	No
vanadium	30	3.2E-05	3.9E-06	8.7E-06	2.7E-03	2.1E-03	1.2E-03	1.8E-05	1.3E-05	5.1E-02	1.1E-01	1.1E-01	6.2E-06	6.8E-06	No	Yes	No
yttrium	33	1.2E-05	1.4E-06	2.9E-06	1.0E-03	7.8E-04	4.7E-04	6.8E-06	4.7E-06	1.9E-02	3.9E-02	3.9E-02	0.0E+00	0.0E+00	No	Yes	No
zinc	120	9.1E-05	8.5E-05	7.4E-05	3.9E-03	3.2E-03	2.6E-03	3.4E-04	1.7E-04	5.9E-02	9.2E-02	9.2E-02	3.3E-04	3.7E-04	No	Yes	No
zirconium	160	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No

Notes:
Units are in $\mu g/m^3$
Outlined and bold values exceed the air threshold
NG = No Guideline
[1] These chemicals were not retained as COPCs because their standards are based upon particulate matter rather than health (particulate matter was assessed in Section 4.5).

Table A.4: Screening of Predicted 24-Hour Air Concentrations for the Acute Air Quality Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
Acid/Greenhouse Gases																	
Carbon Monoxide[1]	6000	3.5E+02	3.5E+02	3.5E+02	3.7E+02	3.7E+02	3.7E+02	3.5E+02	3.5E+02	7.9E+02	6.9E+02	7.9E+02	3.5E+02	3.8E+02	No	Yes	No
Nitrogen Dioxide	200	2.7E+00	3.5E+00	3.3E+00	3.2E+01	3.3E+01	6.4E+01	4.3E+00	6.5E+00	2.0E+02	1.3E+02	2.0E+02	3.5E+00	3.9E+00	No	Yes	No
Sulphur Dioxide	150	5.1E-01	5.1E-01	5.1E-01	6.1E-01	6.6E-01	6.5E-01	5.1E-01	5.2E-01	1.4E+00	4.2E+00	4.2E+00	5.3E-01	5.8E-01	No	Yes	No
Volatile Organic Compounds																	
1,1,1-Trichlorethane	10900	2.8E-09	8.5E-09	4.3E-09	4.3E-08	4.4E-08	9.5E-08	6.7E-09	8.2E-09	5.2E-07	1.2E-06	1.2E-06	7.1E-08	7.9E-08	No	Yes	No
1,3-Butadiene	NG	1.6E-05	2.2E-05	1.7E-05	2.8E-04	3.0E-04	2.2E-04	2.3E-05	3.4E-05	3.3E-03	9.9E-03	9.9E-03	1.9E-06	2.1E-06	No	Yes	No
Acetone	11880	1.1E-03	1.5E-03	1.2E-03	2.0E-02	2.1E-02	1.6E-02	1.6E-03	2.4E-03	2.3E-01	7.0E-01	7.0E-01	0.0E+00	0.0E+00	No	Yes	No
Acrolein	0.08	3.8E-04	5.2E-04	4.2E-04	6.8E-03	7.3E-03	5.3E-03	5.4E-04	8.1E-04	7.8E-02	2.4E-01	2.4E-01	6.6E-05	7.3E-05	Yes	Yes	**Yes**
Aldehydes	500	5.1E-03	7.0E-03	5.6E-03	9.1E-02	9.8E-02	7.1E-02	7.2E-03	1.1E-02	1.1E+00	3.2E+00	3.2E+00	2.1E-04	2.3E-04	No	Yes	No
Benzene	28.71	5.0E-04	8.2E-04	7.7E-04	7.8E-03	9.2E-03	1.6E-02	9.8E-04	1.7E-03	3.3E-01	1.7E-01	3.3E-01	6.5E-03	7.2E-03	No	Yes	No
C16+ Aliphatics	6100	1.8E-04	2.4E-04	1.9E-04	3.1E-03	3.4E-03	2.4E-03	2.5E-04	3.7E-04	3.6E-02	1.1E-01	1.1E-01	0.0E+00	0.0E+00	No	Yes	No
C2-C8 Aliphatics	6100	2.9E-03	4.6E-03	4.1E-03	4.5E-02	5.6E-02	7.4E-02	5.3E-03	8.7E-03	1.2E+00	1.3E+00	1.3E+00	2.3E-02	2.6E-02	No	Yes	No
C9-16 Aliphatics	6100	2.2E-04	3.0E-04	2.4E-04	3.9E-03	4.1E-03	3.0E-03	3.1E-04	4.6E-04	4.5E-02	1.4E-01	1.4E-01	0.0E+00	0.0E+00	No	Yes	No
C9-16 Aromatics	1000	3.1E-04	4.3E-04	3.4E-04	5.6E-03	6.0E-03	4.3E-03	4.4E-04	6.7E-04	6.4E-02	1.9E-01	1.9E-01	0.0E+00	0.0E+00	No	Yes	No
Chlorobenzenes	50	3.1E-08	9.3E-08	4.7E-08	4.7E-07	4.8E-07	1.0E-06	7.3E-08	9.1E-08	5.7E-06	1.3E-05	1.3E-05	7.8E-07	8.6E-07	No	Yes	No
Chloromethane	1000	8.0E-08	2.4E-07	1.2E-07	1.2E-06	1.2E-06	2.7E-06	1.9E-07	2.3E-07	1.5E-05	3.4E-05	3.4E-05	2.0E-06	2.2E-06	No	Yes	No
Dichloromethane	450	8.9E-08	2.6E-07	1.3E-07	1.3E-06	1.4E-06	3.0E-06	2.1E-07	2.6E-07	1.6E-05	3.8E-05	3.8E-05	2.2E-06	2.5E-06	No	Yes	No
Ethylbenzene	1000	2.4E-05	3.3E-05	2.6E-05	4.3E-04	4.6E-04	3.3E-04	3.4E-05	5.1E-05	5.0E-03	1.5E-02	1.5E-02	3.9E-06	4.3E-06	No	Yes	No
Formaldehyde	65	1.2E-03	1.6E-03	1.3E-03	2.1E-02	2.2E-02	1.7E-02	1.7E-03	2.5E-03	2.4E-01	7.2E-01	7.2E-01	6.6E-04	7.3E-04	No	Yes	No
Ketones	1000	3.8E-04	5.2E-04	4.2E-04	6.9E-03	7.3E-03	5.3E-03	5.4E-04	8.2E-04	7.9E-02	2.4E-01	2.4E-01	0.0E+00	0.0E+00	No	Yes	No
Styrenes	400	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
Toluene	400	3.9E-04	7.0E-04	5.9E-04	6.0E-03	7.0E-03	1.1E-02	7.9E-04	1.2E-03	1.2E-01	1.5E-01	1.5E-01	2.5E-03	2.8E-03	No	Yes	No
Trimethylbenzenes	220	5.8E-05	7.9E-05	6.3E-05	1.0E-03	1.1E-03	8.1E-04	8.2E-05	1.2E-04	1.2E-02	3.6E-02	3.6E-02	0.0E+00	0.0E+00	No	Yes	No
Vinyl Chloride	1	2.7E-08	8.0E-08	4.0E-08	4.1E-07	4.2E-07	9.0E-07	6.3E-08	7.8E-08	4.9E-06	1.1E-05	1.1E-05	6.7E-07	7.4E-07	No	Yes	No
Xylenes	700	2.4E-04	3.8E-04	3.3E-04	3.8E-03	4.6E-03	5.7E-03	4.2E-04	6.8E-04	8.3E-02	1.1E-01	1.1E-01	1.6E-03	1.8E-03	No	Yes	No
Dioxins/Furans																	
Total Dioxins/Furans	1.50E-05	1.1E-12	3.4E-12	1.6E-12	1.6E-11	1.7E-11	3.6E-11	2.5E-12	3.2E-12	2.0E-10	4.5E-10	4.5E-10	3.0E-12	3.3E-12	No	Yes	No
Polyaromatic Hydrocarbons																	
2-Methylnaphthalene	10	2.8E-05	3.8E-05	3.0E-05	4.9E-04	5.3E-04	3.8E-04	3.9E-05	5.9E-05	5.4E-03	1.7E-02	1.7E-02	0.0E+00	0.0E+00	No	Yes	No
Acenapthene	NG	2.8E-06	4.5E-06	4.3E-06	4.6E-05	5.0E-05	9.6E-05	5.5E-06	9.7E-06	2.0E-03	9.8E-04	2.0E-03	3.7E-06	4.1E-06	No	Yes	No
Acenapthylene	3.5	6.2E-06	1.0E-05	9.5E-06	9.4E-05	1.1E-04	2.0E-04	1.2E-05	2.1E-05	3.9E-03	2.1E-03	3.9E-03	7.4E-06	8.1E-06	No	Yes	No
Anthracene	0.2	1.2E-06	1.9E-06	1.7E-06	1.8E-05	2.2E-05	3.1E-05	2.1E-06	3.6E-06	5.3E-04	4.7E-04	5.3E-04	9.8E-07	1.1E-06	No	Yes	No
Benzo(a)anthracene	NG	3.8E-07	6.1E-07	5.9E-07	6.1E-06	6.8E-06	1.3E-05	7.4E-07	1.3E-06	2.6E-04	1.3E-04	2.6E-04	5.0E-07	5.5E-07	No	Yes	No
Benzo(a)pyrene	0.0011	2.2E-07	3.4E-07	3.0E-07	3.7E-06	4.3E-06	4.7E-06	3.8E-07	5.9E-07	5.6E-05	1.2E-04	1.2E-04	1.0E-07	1.1E-07	No	Yes	No
Benzo(b+k)fluoranthene	NG	1.3E-06	2.0E-06	1.8E-06	2.0E-05	2.4E-05	3.2E-05	2.3E-06	3.8E-06	5.2E-04	5.5E-04	5.5E-04	9.7E-07	1.1E-06	No	Yes	No
Benzo(g,h,i)perylene	1.2	3.8E-07	5.9E-07	5.2E-07	6.1E-06	7.3E-06	8.7E-06	6.6E-07	1.1E-06	1.2E-04	1.8E-04	1.8E-04	2.2E-07	2.4E-07	No	Yes	No
Chrysene	NG	8.3E-07	1.4E-06	1.3E-06	1.5E-05	1.5E-05	3.1E-05	1.7E-06	3.0E-06	6.5E-04	3.1E-04	6.5E-04	1.2E-06	1.3E-06	No	Yes	No
Dibenzo(a,h)anthracene	NG	1.9E-07	3.1E-07	2.7E-07	3.0E-06	3.7E-06	4.8E-06	3.5E-07	5.6E-07	7.4E-05	8.6E-05	8.6E-05	1.4E-07	1.5E-07	No	Yes	No
Fluoranthene	140	3.3E-06	5.3E-06	4.8E-06	4.8E-05	6.2E-05	9.3E-05	6.1E-06	1.0E-05	1.7E-03	1.2E-03	1.7E-03	3.2E-06	3.5E-06	No	Yes	No
Fluorene	NG	8.8E-06	1.4E-05	1.3E-05	1.3E-04	1.6E-04	2.8E-04	1.7E-05	2.9E-05	5.4E-03	3.0E-03	5.4E-03	1.0E-05	1.1E-05	No	Yes	No
Indeno(1,2,3-c,d)pyrene	NG	1.7E-07	2.7E-07	2.5E-07	2.5E-06	3.2E-06	4.8E-06	3.2E-07	5.4E-07	8.8E-05	6.1E-05	8.8E-05	1.7E-07	1.8E-07	No	Yes	No
Naphthalene	22.5	1.0E-04	1.6E-04	1.5E-04	1.4E-03	1.9E-03	2.9E-03	1.9E-04	3.2E-04	5.5E-02	3.4E-02	5.5E-02	1.0E-04	1.1E-04	No	Yes	No
Phenanthrene	NG	2.5E-05	4.1E-05	3.9E-05	4.0E-04	4.5E-04	8.5E-04	4.9E-05	8.7E-05	1.7E-02	8.7E-03	1.7E-02	3.3E-05	3.6E-05	No	Yes	No
Pyrene	0.2	3.7E-06	5.9E-06	5.3E-06	5.6E-05	7.0E-05	9.5E-05	6.7E-06	1.1E-05	1.6E-03	1.5E-03	1.6E-03	3.0E-06	3.3E-06	No	Yes	No
Metals																	
aluminum	120	1.2E-02	1.0E-03	3.2E-03	1.6E+00	1.4E+00	7.4E-01	5.6E-03	5.0E-03	4.3E+01	2.6E+01	7.9E+01	3.8E-05	4.2E-05	No	Yes	No [2]
antimony	25	3.8E-06	4.2E-06	2.6E-06	3.6E-04	3.0E-04	2.2E-04	3.4E-06	4.4E-06	8.7E-03	1.6E-02	1.6E-02	1.9E-05	2.1E-05	No	Yes	No
arsenic	0.3	3.7E-04	3.2E-05	9.6E-05	4.5E-02	3.8E-02	2.5E-02	1.7E-04	1.6E-04	1.4E+01	2.6E+01	2.6E+01	1.3E-05	1.4E-05	Yes	Yes	**Yes**
barium	10	1.2E-04	1.1E-05	3.4E-05	1.7E-02	1.5E-02	7.8E-03	6.0E-05	5.3E-05	4.7E-01	8.6E-01	8.6E-01	9.2E-07	1.0E-06	No	Yes	No
beryllium	0.01	4.1E-07	3.5E-08	1.1E-07	5.6E-05	4.7E-05	2.6E-05	2.0E-07	1.8E-07	1.5E-03	2.7E-03	2.7E-03	1.7E-09	1.9E-09	No	Yes	No
bismuth	16	1.4E-04	1.2E-05	3.7E-05	1.8E-02	1.6E-02	8.8E-03	6.5E-05	5.9E-05	4.8E-01	8.9E-01	8.9E-01	0.0E+00	0.0E+00	No	Yes	No
boron	120	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
bromine	20	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
cadmium	0.025	2.1E-05	2.6E-05	2.7E-05	3.9E-04	4.3E-04	8.0E-04	3.8E-05	4.8E-05	1.6E-02	8.9E-03	1.6E-02	3.5E-05	3.8E-05	No	Yes	No
calcium	16	4.3E-03	3.6E-04	1.1E-03	5.6E-01	4.7E-01	2.8E-01	2.0E-03	1.9E-03	1.4E+01	2.6E+01	2.6E+01	0.0E+00	0.0E+00	Yes	Yes	No [2]
chromium	1.5	1.5E-05	7.2E-06	8.3E-06	1.7E-03	1.4E-03	8.6E-04	9.1E-06	1.2E-05	4.4E-02	8.1E-02	8.1E-02	2.7E-05	3.0E-05	No	Yes	No
chromium 6	0.14	2.6E-09	5.3E-09	3.0E-09	4.3E-08	4.4E-08	9.1E-08	4.8E-09	6.1E-09	5.5E-07	1.2E-06	1.2E-06	1.5E-08	1.7E-08	No	Yes	No
cobalt	0.1	4.9E-05	4.5E-06	1.5E-05	5.7E-03	4.8E-03	3.1E-03	2.2E-05	2.5E-05	1.4E-01	2.5E-01	2.5E-01	4.8E-06	5.3E-06	Yes	Yes	**Yes**

Table A.4: Screening of Predicted 24-Hour Air Concentrations for the Acute Air Quality Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
copper	50	3.7E-05	1.5E-05	1.8E-05	4.4E-03	3.7E-03	2.3E-03	1.9E-05	2.4E-05	1.1E-01	2.1E-01	2.1E-01	5.6E-05	6.1E-05	No	Yes	No
gallium	NG	3.2E-06	3.4E-06	4.0E-06	6.0E-05	6.6E-05	1.2E-04	5.4E-06	6.8E-06	2.6E-03	1.3E-03	2.6E-03	4.8E-06	5.3E-06	No	Yes	No
indium	0.4	1.9E-05	2.0E-05	2.4E-05	3.6E-04	4.0E-04	7.1E-04	3.2E-05	4.1E-05	1.6E-02	8.0E-03	1.6E-02	2.9E-05	3.2E-05	No	Yes	No
iron	4	2.5E-02	2.1E-03	6.7E-03	3.3E+00	2.8E+00	1.6E+00	1.2E-02	1.1E-02	1.4E+01	2.6E+01	2.6E+01	2.4E-05	2.6E-05	Yes	Yes	No [2]
lanthanum	20	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
lead	0.5	3.6E-05	1.0E-04	7.4E-05	3.1E-04	3.1E-04	3.7E-04	4.5E-05	1.2E-04	5.3E-03	1.0E-02	1.0E-02	6.4E-04	7.0E-04	No	Yes	No
lithium	20	3.5E-06	3.0E-07	9.6E-07	4.9E-04	4.1E-04	2.2E-04	1.7E-06	1.5E-06	1.3E-02	2.4E-02	2.4E-02	0.0E+00	0.0E+00	No	Yes	No
magnesium	120	5.5E-03	4.8E-04	1.5E-03	7.6E-01	6.4E-01	3.5E-01	2.7E-03	2.4E-03	2.0E+01	3.8E+01	3.8E+01	0.0E+00	0.0E+00	No	Yes	No
manganese	2.5	1.4E-04	1.3E-05	3.9E-05	1.8E-02	1.5E-02	9.1E-03	6.6E-05	6.5E-05	4.5E-01	8.5E-01	8.5E-01	4.8E-06	5.3E-06	No	Yes	No
mercury	2	9.4E-07	3.0E-06	1.9E-06	1.1E-05	1.1E-05	2.3E-05	1.2E-06	3.1E-06	1.3E-04	3.1E-04	3.1E-04	1.7E-05	1.8E-05	No	Yes	No
molybdenum	120	1.2E-06	1.0E-07	3.2E-07	1.5E-04	1.3E-04	7.6E-05	5.6E-07	5.1E-07	3.9E-03	7.4E-03	7.4E-03	4.8E-08	5.2E-08	No	Yes	No
nickel	2	4.0E-06	5.1E-06	3.3E-06	4.5E-04	3.8E-04	2.4E-04	2.3E-06	5.4E-06	1.1E-02	2.1E-02	2.1E-02	2.8E-05	3.1E-05	No	Yes	No
palladium	10	3.2E-06	3.4E-06	4.0E-06	6.0E-05	6.6E-05	1.2E-04	5.4E-06	6.8E-06	2.6E-03	1.3E-03	2.6E-03	4.8E-06	5.3E-06	No	Yes	No
phosphorus	0.66	4.9E-05	4.8E-06	1.6E-05	6.6E-03	5.6E-03	3.0E-03	2.3E-05	2.5E-05	1.8E-01	3.3E-01	3.3E-01	4.8E-06	5.3E-06	No	Yes	No
potassium	6.6	1.2E-02	1.0E-03	3.2E-03	1.6E+00	1.4E+00	7.4E-01	5.6E-03	5.0E-03	1.8E-01	3.3E-01	1.6E+00	0.0E+00	0.0E+00	No	Yes	No
rubidium	10	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
selenium	10	1.1E-06	1.8E-07	3.5E-07	1.4E-04	1.2E-04	6.9E-05	5.2E-07	5.2E-07	3.9E-03	7.1E-03	7.1E-03	7.6E-07	8.3E-07	No	Yes	No
silicon	20	2.0E-04	2.1E-04	2.5E-04	3.8E-03	4.2E-03	7.4E-03	3.4E-04	4.3E-04	1.7E-01	8.4E-02	1.7E-01	3.0E-04	3.3E-04	No	Yes	No
silver	1	3.2E-06	3.5E-06	4.1E-06	6.2E-05	7.0E-05	1.2E-04	5.5E-06	7.0E-06	2.6E-03	1.5E-03	2.6E-03	4.8E-06	5.3E-06	No	Yes	No
sodium	16	1.2E-03	1.1E-04	3.3E-04	1.7E-01	1.4E-01	7.8E-02	5.9E-04	5.3E-04	4.4E+00	8.2E+00	8.2E+00	0.0E+00	0.0E+00	No	Yes	No
strontium	120	4.8E-06	4.2E-07	1.3E-06	6.7E-04	5.6E-04	3.1E-04	2.3E-06	2.1E-06	1.8E-02	3.3E-02	3.3E-02	0.0E+00	0.0E+00	No	Yes	No
thallium	0.24	1.3E-07	1.1E-08	3.5E-08	1.8E-05	1.5E-05	8.1E-06	6.2E-08	5.5E-08	4.8E-04	8.8E-04	8.8E-04	0.0E+00	0.0E+00	No	Yes	No
tin	10	1.3E-06	1.1E-07	3.6E-07	1.8E-04	1.5E-04	8.5E-05	6.3E-07	5.7E-07	4.7E-03	8.6E-03	8.6E-03	0.0E+00	0.0E+00	No	Yes	No
titanium	16	3.9E-04	3.4E-05	1.1E-04	5.3E-02	4.5E-02	2.5E-02	1.9E-04	1.7E-04	1.4E+00	2.6E+00	2.6E+00	0.0E+00	0.0E+00	No	Yes	No
tungsten	4	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
uranium	0.16	1.5E-06	1.3E-07	4.0E-07	2.0E-04	1.7E-04	9.5E-05	7.0E-07	6.4E-07	5.1E-03	9.5E-03	9.5E-03	0.0E+00	0.0E+00	No	Yes	No
vanadium	1	8.8E-06	9.5E-07	2.5E-06	1.2E-03	9.9E-04	5.5E-04	4.2E-06	4.0E-06	3.1E-02	5.8E-02	5.8E-02	1.9E-06	2.1E-06	No	Yes	No
yttrium	2.4	3.2E-06	2.8E-07	8.7E-07	4.4E-04	3.7E-04	2.1E-04	1.5E-06	1.4E-06	1.1E-02	2.1E-02	2.1E-02	0.0E+00	0.0E+00	No	Yes	No
zinc	120	2.9E-05	3.0E-05	3.4E-05	1.3E-03	1.2E-03	1.2E-03	4.5E-05	5.7E-05	2.7E-02	5.1E-02	5.1E-02	4.1E-05	4.5E-05	No	Yes	No
zirconium	20	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No

Notes:
Units are in $\mu g/m^3$
Outlined and bold values exceed the air threshold
NG = No Guideline
[1] Carbon monoxide threshold and air predictions are for 8-hour averaging period.
[2] These chemicals were not retained as COPCs because their standards are based upon particulate matter rather than health (particulate matter was assessed in Section 4.5).

Table A.5: Selection of Chronic (Annual) Air Thresholds

Parameter		Air Screening Levels and Guidelines [µg/m^3]							Decision criteria	Value Used [µg/m3]
		AENV[a] (HQ=1.0)	U.S. EPA[b] (HQ=1.0, RL=10^{-6})	ATSDR[c] (HQ=1.0, RL=10^{-6})	CalEPA OEHHA[d] (HQ=1.0, RL = 10^{-6})	WHO[e] (HQ=1.0, RL = 10^{-6})	GNWT[f] (HQ=1.0)	Other[g]		
Particulate Matter										
Total Suspended Particulate (TSP)		60	n/a	n/a	n/a	n/a	**60**	n/a	The project is located in the NWTs, therefore using a NWTs specific threshold is most appropriate.	60
Particulate matter (PM10)		n/a	n/a	n/a	n/a	**25**	n/a	n/a	The WHO value was selected as it was the lowest value and supporting documentation was available.	25
Particulate matter (PM2.5)		n/a	n/a	n/a	n/a	**10**	n/a	n/a	The WHO value was selected as it was the lowest value and supporting documentation was available.	10
Acid Gases										
Carbon monoxide		n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available.	n/a
Hydrogen chloride		n/a	20	n/a	**9**	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	9
Hydrogen fluoride		n/a	14	n/a	**14**	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	14
Hydrogen sulphide		n/a	2	**2**	10	n/a	n/a	n/a	The ATSDR chronic MRL was the lowest threshold and supporting documentation was available.	2
Nitric oxide		n/a	n/a	n/a	n/a	n/a	n/a	**25 (83)**	The TCEQ value was selected in the absence of other values.	83
Nitrogen oxide		n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available	n/a
Nitrous dioxide NO_2 (Non-Carcinogenic)		60	n/a	n/a	n/a	40	**60**	n/a	The project is located in the NWTs, therefore using a NWTs specific threshold is most appropriate.	60
Sulphur dioxide SO_2 (Non-Carcinogenic)		30	n/a	n/a	n/a	50	**30**	n/a	The project is located in the NWTs, therefore using a NWTs specific threshold is most appropriate.	30
Green House Gases (GHGs)										
Carbon dioxide		n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available	n/a
Methane		n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available	n/a
Nitrous oxide		n/a	n/a	n/a	n/a	n/a	n/a	**250 (830)**	The TCEQ value was selected in the absence of other values	830
Volatile Organic Compounds (VOCs)										
Aldehydes (surrogate: acetaldehyde)	Non-carcinogenic	n/a	9	n/a	140	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	0.37
	Carcinogenic	n/a	1.1	n/a	**0.37**	n/a	n/a	n/a		
Acetone (Non-Carcinogenic)		n/a	31,000	**30,880**	n/a	n/a	n/a	n/a	The ATSDR chronic MRL was the lowest threshold and supporting documentation was available.	30,880
Acrolein (Non-Carcinogenic)		n/a	**0.02**	n/a	0.35	n/a	n/a	n/a	The U.S. EPA RSL was the lowest threshold and supporting documentation was available.	0.02
Benzene	Non-Carcinogenic	n/a	30	9.6	60	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	0.034
	Carcinogenic	n/a	0.31	n/a	**0.034**	0.17	n/a	n/a		
1,3-Butadiene	Non-Carcinogenic	n/a	2	n/a	20	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	0.0059
	Carcinogenic	n/a	0.081	n/a	**0.0059**	n/a	n/a	n/a		

Table A.5: Selection of Chronic (Annual) Air Thresholds

Parameter		Air Screening Levels and Guidelines [µg/m^3]							Decision criteria	Value Used [µg/m3]
		AENV[a] (HQ=1.0)	U.S. EPA[b] (HQ=1.0, RL=10^{-6})	ATSDR[c] (HQ=1.0, RL=10^{-6})	CalEPA OEHHA[d] (HQ=1.0, RL = 10^{-6})	WHO[e] (HQ=1.0, RL = 10^{-6})	GNWT[f] (HQ=1.0)	Other[g]		
C_2-C_8 aliphatics (surrogate: cyclohexane) (Non-Carcinogenic)		n/a	**6,000**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	6,000
C_9-C_{16} aliphatic (surrogate: decane) (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**1000 (3300)**	The TCEQ value for decane was selected in the absence of other values.	3,300
C_{16+} aliphatic (surrogate: nonacosane) (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**10 (33)**	The TCEQ value for nonocosane (C17 and above alkane) was selected in the absence of other values.	33
C_9-C_{16} aromatic (surrogate: ethylbenzene)	Non-carcinogenic	n/a	1,000	**261**	2,000	n/a	n/a	n/a	The ATSDR chronic MRL was the lowest threshold and supporting documentation was available.	261
Chlorobenzenes (Non-carcinogenic)		n/a	50	n/a	1000	n/a	n/a	n/a	The U.S. EPA RSL value for chlorobenzenewas selected and supporting documentation was available.	50
Chloromethane (Non-carcinogenic)		n/a	90	103	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	90
Dichloromethane		n/a	n/a	n/a	n/a	**450**	n/a	n/a	The WHO value was selected as it was the lowest value and supporting documentation was available.	450
Ethylbenzene	Non-carcinogenic	n/a	1,000	261	2,000	n/a	n/a	n/a	The CalEPA OEHHA adjusted unit risk was the lowest threshold and supporting documentation was available.	0.4
	Carcinogenic	n/a	0.4	n/a	**0.4**	n/a	n/a	n/a		
Formaldehyde	Non-carcinogenic	n/a	9.8	9.8	9	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	0.077
	Carcinogenic	n/a	**0.077**	n/a	0.17	n/a	n/a	n/a		
Ketones (surrogate: methyl ethyl ketone) (Non-Carcinogenic)		n/a	**5,000**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	5,000
Styrenes		n/a	1000	1000	**900**	n/a	n/a	n/a	The CalEPA OEHHA REL was the most conservative threshold and supporting documentation was available	900
Toluene (Non-Carcinogenic)		n/a	5,000	300	**300**	n/a	n/a	n/a	The CalEPA OEHHA REL was the most conservative threshold and supporting documentation was available.	300
1,1,1-Trichloroethane (Non-Carcinogenic)		n/a	**5,000**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	5,000
Trimethylbenzenes (surrogate: 1,2,4-trimethylbenzene) (Non-Carcinogenic)		n/a	**7**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	7
Vinyl chloride	Non-carcinogenic	n/a	100	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available.	0.23
	Carcinogenic	n/a	**0.23**	n/a	n/a	n/a	n/a	n/a		
Xylenes (Non-Carcinogenic)		n/a	**100**	217	700	n/a		n/a	The U.S. EPA RSL was the lowest value and supporting documentation was available.	100
Dioxins/Furans										
2,3,7,8-Tetrachloro-dibenzo-*p*-dioxin (TCDD) (Carcinogenic)		n/a	**2.60E-08**	n/a	4E-5 (all)	n/a	n/a	n/a	The U.S. EPA RSL was the lowest value and supporting documentation was available.	2.60E-08
Dioxins, polychlorinated, dibenzo (all congeners)		n/a	n/a	n/a	n/a	n/a	n/a	**3.00E-09 (TCEQ)**	The TCEQ value was selected in the absence of other values.	3.00E-09

Table A.5: Selection of Chronic (Annual) Air Thresholds

Parameter		Air Screening Levels and Guidelines [µg/m^3]							Decision criteria	Value Used [µg/m3]
		AENV[a] (HQ=1.0)	U.S. EPA[b] (HQ=1.0, RL=10^{-6})	ATSDR[c] (HQ=1.0, RL=10^{-6})	CalEPA OEHHA[d] (HQ=1.0, RL = 10^{-6})	WHO[e] (HQ=1.0, RL = 10^{-6})	GNWT[f] (HQ=1.0)	Other[g]		
Individual PAHs										
Acenaphthene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.01)**	The TCEQ value was selected in the absence of other values. Supporting documentation is not available; to be conservative this compound was assessed as a carcinogen.	0.01
Acenapthylene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.01)**	The TCEQ value was selected in the absence of other values. Supporting documentation is not available; to be conservative this compound was assessed as a carcinogen.	0.01
Anthracene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.005)**	The TCEQ value was selected in the absence of other values. Supporting documentation is not available; to be conservative this compound was assessed as a carcinogen.	0.005
Benzo(a)anthracene (Carcinogenic)		n/a	**0.0091**	n/a	0.0091	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available	0.0091
Benzo(a)pyrene (Carcinogenic)		n/a	0.0091	n/a	0.00091	**0.000012**	n/a	n/a	The WHO was selected as it is the lowest value and it is based on health.	0.000012
Benzo(b)fluoranthene (Carcinogenic)		n/a	**0.0091**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL value was selected and supporting documentation was available.	0.0091
Benzo(b+k)fluoranthene (Carcinogenic)		n/a	**0.0091**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available.	0.0091
Benzo(g,h,i)perylene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.005)**	The TCEQ value was selected in the absence of other values.	0.005
Benzo(k)fluoranthene (Carcinogenic)		n/a	**0.0091**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available	0.0091
Chrysene (Carcinogenic)		n/a	**0.091**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available	0.091
Dibenzo(a,h)anthracene (Carcinogenic)		n/a	**0.00083**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available	0.0008
Fluoranthene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.005)**	The TCEQ value was selected in the absence of other values.	0.005
Fluorene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**1 (0.1)**	The TCEQ value was selected in the absence of other values.	0.1
Indeno(1,2,3-cd)pyrene (Carcinogenic)		n/a	**0.0091**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as supporting documentation was available	0.0091
2-Methylnaphthalene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**3 (0.3)**	The TCEQ value was selected in the absence of other values.	0.3
Naphthalene	Non-Carcinogenic	n/a	3	3	9	n/a	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	0.029
	Carcinogenic	n/a	**0.029**	n/a	n/a	50 (5)	n/a	n/a		
Phenanthrene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.005)**	The TCEQ value was selected in the absence of other values.	0.005
Pyrene (Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.05 (0.005)**	The TCEQ value was selected in the absence of other values.	0.005
Metals										
Aluminum (Non-Carcinogenic)		n/a	**5**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	5
Antimony (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.5 (1.6)**	The TCEQ value was selected in the absence of other values.	1.6

Table A.5: Selection of Chronic (Annual) Air Thresholds

Parameter		Air Screening Levels and Guidelines [µg/m^3]							Decision criteria	Value Used [µg/m3]
		AENV[a] (HQ=1.0)	U.S. EPA[b] (HQ=1.0, RL=10^{-6})	ATSDR[c] (HQ=1.0, RL=10^{-6})	CalEPA OEHHA[d] (HQ=1.0, RL = 10^{-6})	WHO[e] (HQ=1.0, RL = 10^{-6})	GNWT[f] (HQ=1.0)	Other[g]		
Arsenic	(Non-Carcinogenic)	0.01	0.015	n/a	0.015	n/a	n/a	n/a	The CalEPA value was selected as it was the lowest value and supporting documentation was available.	0.015
	(Carcinogenic)	n/a	**0.00023**	n/a	0.0003	0.00066	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	0.00023
Barium (Non-Carcinogenic)		n/a	**0.5**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	0.5
Beryllium	Non-Carcinogenic	n/a	0.02	0.02	0.007	n/a	n/a	n/a	The CalEPA value was selected as it was the lowest value and supporting documentation was available.	0.00042
	Carcinogenic	n/a	0.00042	n/a	**0.00042**	n/a	n/a	n/a		
Bismuth (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value was selected in the absence of other values and is based upon particulate matter.	16
Boron (Non-Carcinogenic)		n/a	**20**	n/a	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	20
Bromine		n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33)**	The TCEQ value was selected in the absence of other values.	0.33
Cadmium	Non-Carcinogenic	n/a	0.01	0.01	0.02	0.005	n/a	n/a	The Cal EPA value was selected as it was the lowest value and supporting documentation was available.	0.00024
	Carcinogenic	n/a	0.00055	n/a	**0.00024**	n/a	n/a	n/a		
Calcium		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value for calcium chloride was selected in the absence of other values.	16
Chromium III (Non-Carcinogenic)[o]		n/a	n/a	n/a	n/a	n/a	n/a	**0.041 (0.14)**	The TCEQ value was selected in the absence of other values and is based upon particulate matter.	0.14
Chromium VI (Carcinogenic)		n/a	0.000012	0.005	**0.0000067**	0.000025	n/a	n/a	The Cal EPA value was selected as it was the lowest value and supporting documentation was available.	0.0000067
Cobalt (Non-Carcinogenic)		n/a	**0.006**	0.1	n/a	n/a	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	0.006
Copper (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3)**	The TCEQ value was selected in the absence of other values.	3.3
Gallium		n/a	n/a	n/a	n/a	n/a	n/a	n/a	No values were available	n/a
Indium		n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33)**	The TCEQ value was selected in the absence of other values.	0.33
Iron (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3)**	The TCEQ value was selected in the absence of other values.	3.3
Lanthanum		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value for lanthanum oxide was selected in the absence of other values.	16
Lead	Non-Carcinogenic	n/a	n/a	n/a	n/a	n/a		n/a	The CalEPA value was selected as it was the lowest value and supporting documentation was available.	0.083
	Carcinogenic	n/a	n/a	n/a	**0.083**	n/a	n/a	n/a		
Lithium		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value for lithium chloride (PM) was selected in the absence of other values.	16
Magnesium		n/a	n/a	n/a	n/a	n/a	n/a	**10 (33)**	The TCEQ value for magnesium chloride was selected in the absence of other values.	33
Manganese (Non-Carcinogenic)		0.2	**0.05**	n/a	0.09	0.15	n/a	n/a	The U.S. EPA RSL was selected as it was the lowest value and supporting documentation was available.	0.05
Mercury (Non-Carcinogenic)		n/a	0.03	n/a	**0.03**	1	n/a	n/a	The CalEPA value was selected as it was the lowest value and supporting documentation was available.	0.03
Molybdenum (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**3 (10)**	The TCEQ value was selected in the absence of other values and is based upon particulate matter.	10

Table A.5: Selection of Chronic (Annual) Air Thresholds

Parameter		Air Screening Levels and Guidelines [µg/m^3]							Decision criteria	Value Used [µg/m3]
		AENV[a] (HQ=1.0)	U.S. EPA[b] (HQ=1.0, RL=10^{-6})	ATSDR[c] (HQ=1.0, RL=10^{-6})	CalEPA OEHHA[d] (HQ=1.0, RL = 10^{-6})	WHO[e] (HQ=1.0, RL = 10^{-6})	GNWT[f] (HQ=1.0)	Other[g]		
Nickel	Non-Carcinogenic	0.05	0.09	0.09	0.05	n/a	n/a	n/a	The WHO value was selected as it was the lowest value and supporting documentation was available.	0.0025
	Carcinogenic	n/a	0.0038	n/a	0.0038	**0.0025**	n/a	n/a		
Palladium		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value was selected in the absence of other values, and is based on particulate matter.	16
Phosphorus		n/a	n/a	n/a	n/a	n/a	n/a	**0.2 (0.66)**	The TCEQ value for phosphorus trichloride was selected in the absence of other values.	0.66
Potassium		n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.6)**	The TCEQ value was selected in the absence of other values.	6.6
Rubidium		n/a	n/a	n/a	n/a	n/a	n/a	**2.5 (8.3)**	The TCEQ value was selected in the absence of other values.	8.3
Selenium (Non-Carcinogenic)		n/a	20	n/a	**20**	n/a	n/a	n/a	The CalEPA value was selected as it was the lowest value and supporting documentation was available.	20
Silicon		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value was selected in the absence of other values, and is based on particulate matter.	16
Silver (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**0.01 (0.03)**	The TCEQ value was selected in the absence of other values.	0.03
Sodium		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value was selected in the absence of other values, and is based on particulate matter.	16
Strontium (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.6)**	The TCEQ value was selected in the absence of other values, and is based on particulate matter.	6.6
Thallium		n/a	n/a	n/a	n/a	n/a	n/a	**0.1 (0.33)**	The TCEQ value was selected in the absence of other values.	0.33
Tin		n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.6)**	The TCEQ value for tin oxide was selected in the absence of other values, and is based on particulate matter.	6.6
Titanium (Non-Carcinogenic)		n/a	**0.1**	0.1	n/a	n/a	n/a	n/a	The US EPA value for titanium tetrachloride was selected as it was the lowest value and supporting documentation was available.	0.1
Tungsten		n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3)**	The TCEQ value for soluble tungsten was was selected in the absence of other values, and is based on particulate matter.	3.3
Vanadium (Non-Carcinogenic)		n/a	0.1	**0.1**	n/a	n/a	n/a	n/a	The ATSDR MRL was selected as it was the lowest value and supporting documentation was available.	0.1
Uranium (Non-Carcinogenic)		n/a	0.3	**0.3**	n/a	n/a	n/a	n/a	The ATSDR MRL was selected as it was the lowest value and supporting documentation was available.	0.3
Yttrium		n/a	n/a	n/a	n/a	n/a	n/a	**1 (3.3)**	The TCEQ value was selected in the absence of other values.	3.3
Zinc (Non-Carcinogenic)		n/a	n/a	n/a	n/a	n/a	n/a	**2 (6.6)**	The TCEQ value was selected in the absence of other values and is based upon particulate matter.	6.6
Zirconium		n/a	n/a	n/a	n/a	n/a	n/a	**5 (16)**	The TCEQ value was selected in the absence of other values.	16

[a] Alberta Ambient Air Quality Objectives (AAAQO; AENV 2009, internet site)..

[b] United States Environmental Protection Agency; Regional Screening Level Table; Residential Air Screening Level (U.S. EPA 2009, internet site).

[c] Agnecy for Toxic Substances and Disease Registry; Chronic Duration Minimal Risk Levels (ATSDR 2010, internet site).

[d] California Environmental Protection Agency Office of Environmental Health Hazard Assessment (CalEPA OEHHA 2009, internet site). For non-carcinogens, chronic reference exposure levels (RELs) are shown. For carcinogens, inhalation unit risks (ba

[e] World Health Organization (WHO 2000, 2005).

[f] NWT Ambient Air Quality Standards (GNWT; NWT ENR 2005, internet site).

[g] Other source is, unless otherwise noted, the Texas Commission on Environmental Quality (TCEQ 2010, internet site). Values for TCEQ were only provided where values from the preferred sources were not available. For non-carcinogens, the screenin

n/a = Not available.

Note: HQ = Risk level (Hazard Quotient) used by regulatory agency for developing screening levels/guidelines for non-carcinogens.

RL = Risk level used by regulatory agency for developing screening levels/guidelines for carcinogens.

Table A.6: Screening of Annual Average Air Concentrations for the Chronic Air Quality Assessment and Multi-Media Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Lowest Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
Acid/Greenhouse Gases																	
Nitrogen Dioxide	60	1.2E+00	1.1E+00	1.1E+00	2.7E+00	3.3E+00	4.8E+00	1.2E+00	1.2E+00	5.1E+01	**6.8E+01**	6.8E+01	1.1E+00	1.2E+00	Yes	Yes	**Yes**
Sulphur Dioxide	30	5.0E-01	5.0E-01	5.0E-01	5.1E-01	5.1E-01	5.1E-01	5.0E-01	5.0E-01	5.8E-01	9.8E-01	9.8E-01	5.0E-01	5.5E-01	No	Yes	No
Volatile Organic Compounds																	
1,1,1-Trichlorethane	5000	3.6E-10	2.9E-10	3.1E-10	2.0E-09	2.2E-09	5.6E-09	3.7E-10	5.0E-10	1.1E-08	9.8E-08	9.8E-08	2.9E-09	3.1E-09	No	Yes	No
1,3-Butadiene	0.0059	1.4E-06	4.7E-07	9.5E-07	2.2E-05	2.3E-05	2.1E-05	1.1E-06	1.7E-06	3.4E-04	1.2E-03	1.2E-03	7.7E-08	8.5E-08	No	Yes	No
Acetone	30880	9.9E-05	3.3E-05	6.7E-05	1.5E-03	1.6E-03	1.5E-03	8.0E-05	1.2E-04	2.4E-02	8.5E-02	8.5E-02	0.0E+00	0.0E+00	No	Yes	No
Acrolein	0.02	3.4E-05	1.1E-05	2.3E-05	5.2E-04	5.4E-04	5.0E-04	2.7E-05	4.1E-05	8.2E-03	**2.9E-02**	2.9E-02	2.3E-06	2.5E-06	Yes	Yes	**Yes**
Aldehydes	0.37	4.5E-04	1.5E-04	3.0E-04	7.0E-03	7.3E-03	6.7E-03	3.6E-04	5.4E-04	1.1E-01	**3.8E-01**	3.8E-01	7.3E-06	8.0E-06	Yes	Yes	**Yes**
Benzene	0.034	5.4E-05	2.0E-05	3.9E-05	5.1E-04	6.9E-04	1.1E-03	6.2E-05	7.9E-05	1.5E-02	2.1E-02	2.1E-02	2.2E-04	2.5E-04	No	Yes	No
C16+ Aliphatics	33	1.5E-05	5.2E-06	1.0E-05	2.4E-04	2.5E-04	2.3E-04	1.2E-05	1.9E-05	3.7E-03	1.3E-02	1.3E-02	0.0E+00	0.0E+00	No	Yes	No
C2-C8 Aliphatics	6000	3.1E-04	1.1E-04	2.2E-04	3.5E-03	4.3E-03	5.5E-03	3.1E-04	4.2E-04	8.1E-02	1.7E-01	1.7E-01	8.0E-04	8.8E-04	No	Yes	No
C9-16 Aliphatics	3300	1.9E-05	6.4E-06	1.3E-05	3.0E-04	3.1E-04	2.8E-04	1.5E-05	2.3E-05	4.6E-03	1.6E-02	1.6E-02	0.0E+00	0.0E+00	No	Yes	No
C9-16 Aromatics	261	2.8E-05	9.2E-06	1.9E-05	4.3E-04	4.4E-04	4.1E-04	2.2E-05	3.3E-05	6.7E-03	2.3E-02	2.3E-02	0.0E+00	0.0E+00	No	Yes	No
Chlorobenzenes	50	4.0E-09	3.2E-09	3.4E-09	2.2E-08	2.4E-08	6.1E-08	4.1E-09	5.5E-09	1.2E-07	1.1E-06	1.1E-06	3.1E-08	3.5E-08	No	Yes	No
Chloromethane	90	1.0E-08	8.2E-09	8.7E-09	5.5E-08	6.1E-08	1.6E-07	1.1E-08	1.4E-08	3.2E-07	2.8E-06	2.8E-06	8.0E-08	8.8E-08	No	Yes	No
Dichloromethane	450	1.1E-08	9.1E-09	9.7E-09	6.1E-08	6.8E-08	1.7E-07	1.2E-08	1.6E-08	3.5E-07	3.1E-06	3.1E-06	8.9E-08	9.8E-08	No	Yes	No
Ethylbenzene	0.4	2.1E-06	7.2E-07	1.4E-06	3.3E-05	3.4E-05	3.2E-05	1.7E-06	2.6E-06	5.1E-04	1.8E-03	1.8E-03	1.6E-07	1.7E-07	No	Yes	No
Formaldehyde	0.17	1.0E-04	3.5E-05	7.1E-05	1.6E-03	1.7E-03	1.6E-03	8.6E-05	1.3E-04	2.6E-02	8.7E-02	8.7E-02	2.3E-05	2.5E-05	No	Yes	No
Ketones	5000	3.4E-05	1.1E-05	2.3E-05	5.3E-04	5.4E-04	5.0E-04	2.7E-05	4.1E-05	8.2E-03	2.9E-02	2.9E-02	0.0E+00	0.0E+00	No	Yes	No
Styrenes	900	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
Toluene	300	4.5E-05	2.2E-05	3.4E-05	4.4E-04	5.4E-04	7.7E-04	4.6E-05	6.2E-05	9.1E-03	2.1E-02	2.1E-02	1.0E-04	1.1E-04	No	Yes	No
Trimethylbenzenes	7	5.1E-06	1.7E-06	3.5E-06	8.0E-05	8.3E-05	7.6E-05	4.1E-06	6.2E-06	1.2E-03	4.4E-03	4.4E-03	0.0E+00	0.0E+00	No	Yes	No
Vinyl Chloride	0.16	3.4E-09	2.8E-09	2.9E-09	1.9E-08	2.1E-08	5.3E-08	3.5E-09	4.7E-09	1.1E-07	9.3E-07	9.3E-07	2.7E-08	3.0E-08	No	Yes	No
Xylenes	100	2.5E-05	8.8E-06	1.7E-05	3.0E-04	3.5E-04	4.3E-04	2.4E-05	3.3E-05	6.5E-03	1.5E-02	1.5E-02	5.6E-05	6.1E-05	No	Yes	No
Dioxins/Furans																	
Total Dioxins/Furans	6.40E-08	1.1E-12	3.4E-12	1.6E-12	1.6E-11	1.7E-11	3.6E-11	2.5E-12	3.2E-12	2.0E-10	4.5E-10	4.5E-10	1.7E-13	1.8E-13	No	Yes	No
Polyaromatic Hydrocarbons																	
2-Methylnaphthalene	0.3	2.5E-06	8.2E-07	1.7E-06	3.8E-05	3.9E-05	3.6E-05	2.0E-06	3.0E-06	5.7E-04	2.1E-03	2.1E-03	0.0E+00	0.0E+00	No	Yes	No
Acenapthene	0.01	3.0E-07	1.1E-07	2.2E-07	2.7E-06	3.7E-06	6.0E-06	3.5E-07	4.4E-07	8.5E-05	1.0E-04	1.0E-04	1.0E-07	1.1E-07	No	Yes	No
Acenapthylene	0.01	6.7E-07	2.5E-07	4.8E-07	6.4E-06	8.6E-06	1.3E-05	7.6E-07	9.7E-07	1.9E-04	2.7E-04	2.7E-04	2.0E-07	2.2E-07	No	Yes	No
Anthracene	0.005	1.2E-07	4.4E-08	8.7E-08	1.3E-06	1.7E-06	2.2E-06	1.3E-07	1.7E-07	3.2E-05	6.5E-05	6.5E-05	2.6E-08	2.9E-08	No	Yes	No
Benzo(a)anthracene	0.0087	4.1E-08	1.5E-08	3.0E-08	3.7E-07	5.1E-07	8.2E-07	4.8E-08	6.0E-08	1.1E-05	1.5E-05	1.5E-05	1.3E-08	1.5E-08	No	Yes	**Yes [1]**
Benzo(a)pyrene	0.000012	2.3E-08	7.9E-09	1.6E-08	2.9E-07	3.3E-07	3.8E-07	2.1E-08	2.9E-08	5.6E-06	**1.5E-05**	1.5E-05	2.8E-09	3.0E-09	Yes	Yes	**Yes [1]**
Benzo(b+k)fluoranthene	0.0087	1.3E-07	4.8E-08	9.4E-08	1.5E-06	1.9E-06	2.4E-06	1.4E-07	1.8E-07	3.5E-05	7.4E-05	7.4E-05	2.6E-08	2.9E-08	No	Yes	**Yes [1]**
Benzo(g,h,i)perylene	0.005	3.9E-08	1.4E-08	2.7E-08	4.8E-07	5.6E-07	6.7E-07	3.8E-08	5.2E-08	9.9E-06	2.4E-05	2.4E-05	6.0E-09	6.6E-09	No	Yes	**Yes [1]**
Chrysene	0.087	9.1E-08	3.4E-08	6.6E-08	7.8E-07	1.1E-06	1.9E-06	1.1E-07	1.4E-07	2.6E-05	2.9E-05	2.9E-05	3.3E-08	3.6E-08	No	Yes	**Yes [1]**
Dibenzo(a,h)anthracene	0.0008	2.0E-08	7.2E-09	1.4E-08	2.3E-07	2.8E-07	3.6E-07	2.0E-08	2.7E-08	5.2E-06	1.1E-05	1.1E-05	3.7E-09	4.1E-09	No	Yes	**Yes [1]**
Fluoranthene	0.005	3.5E-07	1.3E-07	2.5E-07	3.6E-06	4.7E-06	6.5E-06	3.8E-07	4.9E-07	9.4E-05	1.7E-04	1.7E-04	8.7E-08	9.5E-08	No	Yes	No
Fluorene	0.1	9.5E-07	3.5E-07	6.8E-07	9.2E-06	1.2E-05	1.8E-05	1.1E-06	1.4E-06	2.6E-04	4.0E-04	4.0E-04	2.7E-07	3.0E-07	No	Yes	No
Indeno(1,2,3-c,d)pyrene	0.0087	1.8E-08	6.6E-09	1.3E-08	1.9E-07	2.4E-07	3.4E-07	1.9E-08	2.5E-08	4.8E-06	8.6E-06	8.6E-06	4.4E-09	4.9E-09	No	Yes	**Yes [1]**
Naphthalene	0.072	1.1E-05	3.9E-06	7.7E-06	1.1E-04	1.4E-04	2.0E-04	1.2E-05	1.5E-05	2.9E-03	4.9E-03	4.9E-03	2.8E-06	3.1E-06	No	Yes	No
Phenanthrene	0.005	2.7E-06	1.0E-06	2.0E-06	2.5E-05	3.4E-05	5.4E-05	3.1E-06	4.0E-06	7.6E-04	9.9E-04	9.9E-04	8.8E-07	9.6E-07	No	Yes	No
Pyrene	0.005	3.9E-07	1.4E-07	2.7E-07	4.3E-06	5.3E-06	7.0E-06	4.0E-07	5.3E-07	1.0E-04	2.1E-04	2.1E-04	8.0E-08	8.8E-08	No	Yes	No
Metals																	
aluminum	5	4.7E-04	1.7E-05	1.2E-04	5.9E-02	3.4E-02	3.3E-02	1.1E-04	1.3E-04	4.0E+00	**7.8E+00**	7.8E+00	9.5E-07	1.0E-06	Yes	Yes	No [2]
antimony	1.6	3.0E-07	1.4E-07	1.9E-07	1.4E-05	8.6E-06	1.1E-05	2.0E-07	2.9E-07	8.6E-04	1.6E-03	1.6E-03	1.0E-06	1.1E-06	No	Yes	No
arsenic	0.0003	1.6E-05	5.8E-07	3.7E-06	**1.7E-03**	**1.3E-03**	**1.0E-03**	3.5E-06	4.2E-06	**1.1E-01**	**2.0E-01**	2.0E-01	7.0E-07	7.7E-07	Yes	Yes	**Yes**
barium	0.5	5.0E-06	1.8E-07	1.2E-06	6.3E-04	3.6E-04	3.5E-04	1.2E-06	1.4E-06	4.3E-02	8.4E-02	8.4E-02	5.0E-08	5.6E-08	No	Yes	No
beryllium	0.00042	1.7E-08	5.9E-10	4.1E-09	2.1E-06	1.2E-06	1.2E-06	3.9E-09	4.5E-09	1.4E-04	2.7E-04	2.7E-04	9.2E-11	1.0E-10	No	Yes	No
bismuth	16.6	5.6E-06	1.9E-07	1.4E-06	6.8E-04	4.1E-04	3.8E-04	1.3E-06	1.5E-06	4.6E-02	8.8E-02	8.8E-02	0.0E+00	0.0E+00	No	Yes	No
boron	20	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
bromine	0.33	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
cadmium	0.00024	2.1E-06	6.4E-07	1.5E-06	2.4E-05	3.0E-05	5.0E-05	2.3E-06	2.7E-06	**7.3E-04**	**1.0E-03**	1.0E-03	2.2E-06	2.4E-06	Yes	Yes	**Yes**
calcium	16	1.8E-04	6.0E-06	4.2E-05	2.1E-02	1.3E-02	1.2E-02	4.0E-05	4.7E-05	1.4E+00	2.6E+00	2.6E+00	0.0E+00	0.0E+00	No	Yes	No
chromium	0.14	9.6E-07	2.5E-07	4.8E-07	6.3E-05	3.8E-05	4.3E-05	6.1E-07	7.8E-07	4.1E-03	8.0E-03	8.0E-03	1.5E-06	1.7E-06	No	Yes	No
chromium 6	0.0000067	2.6E-10	1.5E-10	2.1E-10	1.8E-09	1.9E-09	5.2E-09	2.2E-10	3.1E-10	1.1E-08	1.0E-07	1.0E-07	8.2E-10	9.1E-10	No	Yes	No
cobalt	0.00027	2.2E-06	1.4E-07	6.6E-07	2.2E-04	1.6E-04	1.3E-04	7.6E-07	8.9E-07	**1.4E-02**	**2.5E-02**	2.5E-02	1.2E-07	1.3E-07	Yes	Yes	**Yes**

Table A.6: Screening of Annual Average Air Concentrations for the Chronic Air Quality Assessment and Multi-Media Risk Assessment

Parameter	Air Threshold	Behchoko	Wekweeti	Yellowknife	Marian River Receptor	Hislop Lake Receptor	Bea Lake Receptor	Gameti	Whati	Camp	MPOI	Maximum Concentration	Baseline	Baseline Maximum + 10%	Max Above Lowest Guideline?	Max Above Baseline Maximum + 10%?	Retained as a COPC?
copper	3.3	2.2E-06	5.2E-07	1.0E-06	1.7E-04	9.9E-05	1.1E-04	1.1E-06	1.5E-06	1.1E-02	2.1E-02	2.1E-02	3.1E-06	3.4E-06	No	Yes	No
gallium	NG	3.0E-07	6.8E-08	2.0E-07	3.6E-06	4.7E-06	7.4E-06	3.3E-07	3.7E-07	1.2E-04	1.6E-04	1.6E-04	1.2E-07	1.3E-07	No	Yes	No
indium	0.33	1.8E-06	4.1E-07	1.2E-06	2.1E-05	2.8E-05	4.4E-05	2.0E-06	2.2E-06	7.1E-04	9.5E-04	9.5E-04	7.2E-07	7.9E-07	No	Yes	No
iron	3.3	1.0E-03	3.5E-05	2.5E-04	1.2E-01	7.4E-02	7.0E-02	2.4E-04	2.7E-04	**8.3E+00**	**1.6E+01**	1.6E+01	6.0E-07	6.6E-07	Yes	Yes	No [2]
lanthanum	16	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
lead	0.083	3.1E-06	3.3E-06	3.0E-06	2.1E-05	1.9E-05	3.0E-05	2.9E-06	5.3E-06	6.4E-04	1.2E-03	1.2E-03	3.5E-05	3.8E-05	No	Yes	No
lithium	16	1.4E-07	4.9E-09	3.4E-08	1.8E-05	1.0E-05	9.9E-06	3.3E-08	3.8E-08	1.2E-03	2.4E-03	2.4E-03	0.0E+00	0.0E+00	No	Yes	No
magnesium	33	2.2E-04	7.7E-06	5.4E-05	2.8E-02	1.6E-02	1.6E-02	5.3E-05	6.1E-05	1.9E+00	3.7E+00	3.7E+00	0.0E+00	0.0E+00	No	Yes	No
manganese	0.05	6.1E-06	2.9E-07	1.6E-06	6.8E-04	4.3E-04	3.9E-04	1.7E-06	2.0E-06	4.5E-02	**8.4E-02**	8.4E-02	2.8E-07	3.0E-07	Yes	Yes	**Yes**
mercury	0.03	1.1E-07	9.8E-08	1.0E-07	5.8E-07	6.0E-07	1.4E-06	9.8E-08	1.7E-07	3.1E-06	2.5E-05	2.5E-05	9.1E-07	1.0E-06	No	Yes	No
molybdenum	10	4.9E-08	2.1E-09	1.2E-08	5.7E-06	3.5E-06	3.3E-06	1.2E-08	1.4E-08	3.8E-04	7.2E-04	7.2E-04	2.6E-09	2.9E-09	No	Yes	No
nickel	0.0025	3.2E-07	1.7E-07	2.0E-07	1.7E-05	1.1E-05	1.2E-05	1.9E-07	3.2E-07	1.1E-03	2.1E-03	2.1E-03	1.6E-06	1.7E-06	No	Yes	No
palladium	16	3.0E-07	6.8E-08	2.0E-07	3.6E-06	4.7E-06	7.4E-06	3.3E-07	3.7E-07	1.2E-04	1.6E-04	1.6E-04	1.2E-07	1.3E-07	No	Yes	No
phosphorus	0.66	2.2E-06	1.4E-07	6.6E-07	2.4E-04	1.4E-04	1.4E-04	7.8E-07	8.9E-07	1.6E-02	3.3E-02	3.3E-02	1.2E-07	1.3E-07	No	Yes	No
potassium	6.6	4.7E-04	1.6E-05	1.1E-04	5.9E-02	3.4E-02	3.3E-02	1.1E-04	1.3E-04	4.0E+00	**7.9E+00**	7.9E+00	0.0E+00	0.0E+00	Yes	Yes	No [2]
rubidium	8.3	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
selenium	20	5.0E-08	7.1E-09	1.7E-08	5.3E-06	3.1E-06	3.1E-06	1.7E-08	2.2E-08	3.6E-04	7.0E-04	7.0E-04	4.2E-08	4.6E-08	No	Yes	No
silicon	16	1.9E-05	4.3E-06	1.3E-05	2.3E-04	2.9E-04	4.7E-04	2.1E-05	2.3E-05	7.4E-03	1.0E-02	1.0E-02	7.5E-06	8.3E-06	No	Yes	No
silver	0.03	3.1E-07	7.5E-08	2.1E-07	3.9E-06	4.8E-06	7.7E-06	3.4E-07	3.9E-07	1.3E-04	1.9E-04	1.9E-04	1.2E-07	1.3E-07	No	Yes	No
sodium	16	5.0E-05	1.7E-06	1.2E-05	6.2E-03	3.6E-03	3.5E-03	1.2E-05	1.3E-05	4.1E-01	8.0E-01	8.0E-01	0.0E+00	0.0E+00	No	Yes	No
strontium	6.6	1.9E-07	6.7E-09	4.7E-08	2.4E-05	1.4E-05	1.4E-05	4.6E-08	5.3E-08	1.6E-03	3.2E-03	3.2E-03	0.0E+00	0.0E+00	No	Yes	No
thallium	0.33	5.2E-09	1.8E-10	1.3E-09	6.5E-07	3.7E-07	3.6E-07	1.2E-09	1.4E-09	4.4E-05	8.6E-05	8.6E-05	0.0E+00	0.0E+00	No	Yes	No
tin	6.6	5.4E-08	1.8E-09	1.3E-08	6.6E-06	3.9E-06	3.7E-06	1.2E-08	1.5E-08	4.4E-04	8.5E-04	8.5E-04	0.0E+00	0.0E+00	No	Yes	No
titanium	0.1	1.6E-05	5.4E-07	3.8E-06	2.0E-03	1.1E-03	1.1E-03	3.7E-06	4.3E-06	**1.3E-01**	**2.6E-01**	2.6E-01	0.0E+00	0.0E+00	Yes	Yes	No [2]
tungsten	3.3	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No
uranium	0.3	6.0E-08	2.1E-09	1.5E-08	7.3E-06	4.4E-06	4.1E-06	1.4E-08	1.6E-08	4.9E-04	9.3E-04	9.3E-04	0.0E+00	0.0E+00	No	Yes	No
vanadium	0.1	3.8E-07	3.1E-08	1.1E-07	4.3E-05	2.5E-05	2.5E-05	1.1E-07	1.3E-07	2.9E-03	5.7E-03	5.7E-03	1.1E-07	1.2E-07	No	Yes	No
yttrium	3.3	1.3E-07	4.5E-09	3.2E-08	1.6E-05	9.5E-06	9.0E-06	3.0E-08	3.5E-08	1.1E-03	2.1E-03	2.1E-03	0.0E+00	0.0E+00	No	Yes	No
zinc	6.6	2.8E-06	7.7E-07	1.8E-06	6.0E-05	5.3E-05	7.8E-05	2.7E-06	3.2E-06	3.1E-03	5.6E-03	5.6E-03	2.6E-06	2.8E-06	No	Yes	No
zirconium	16	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00	No	No	No

Notes:
Units are in $\mu g/m^3$
Outlined and bold values exceed the air threshold
NG = No Guideline
[1] Because benzo(a)pyrene exceeded its threshold, the other carcinogenic PAHs were also retained.
[2] These chemicals were not retained as COPCs because their standards are based upon particulate matter rather than health (particulate matter was assessed in Section 4.6).

APPENDIX B

Screening Tables for the Multi-Media Risk Assessment

Table B.1: Screening of Peak 95th Percentile Predicted Surface Water Concentrations

Parameter	Units	Health Canada Drinking Water Quality Guideline	Notes	Baseline Concentration	+10% Baseline	Nico Lake	Marian River	COPC?
Total Metals								
Aluminum	µg/L	100 / 200	[1]	72	79	**1,308**	**107**	No[2]
Antimony	µg/L	6		0.22	0.25	3.7	0.07	No
Arsenic	µg/L	10		6.9	7.6	**44**	1.7	Yes
Barium	µg/L	1000		10	11	24	30	No
Beryllium	µg/L	NG		0.51	0.56	0.15	0.03	No
Boron	µg/L	5000		181	199	25	35	No
Cadmium	µg/L	5		20	22	0.06	0.05	No
Chromium	µg/L	50		0.15	0.17	2.0	0.77	No
Cobalt	µg/L	NG		0.69	0.76	6.1	0.28	No
Copper	µg/L	1000	[1]	1.2	1.3	4.9	1.7	No
Iron	µg/L	300	[1]	448	493	**3,297**	**314**	No [3]
Lead	µg/L	10		0.61	0.68	1.80	0.29	No
Manganese	µg/L	50	[1]	65	71	78	63	No
Mercury	µg/L	1		0.05	0.06	0.03	0.08	No
Molybdenum	µg/L	NG		1.5	1.6	5.9	0.44	No
Nickel	µg/L	NG		0.87	0.96	1.6	2.0	No
Selenium	µg/L	10		0.25	0.27	2.6	0.72	No
Silver	µg/L	NG		0.61	0.67	0.10	0.01	No
Thallium	µg/L	NG		2.8	3.1	0.87	0.01	No
Uranium	µg/L	20		5.8	6.4	9.5	1.8	No
Vanadium	µg/L	NG		0.83	0.92	1.2	1.1	No
Zinc	µg/L	5000	[1]	4.3	4.7	28	24	No

Notes:

3.5 Outline and bold font indicates value >baseline+10% and >guideline.

NG No Guideline is available.

[1] The Health Canada Guideline is based on aesthetic objectives or Operational Guidance Values

[2] The Health Canada Guideline is that is available for aluminum is based on an Operational Guidance Value of 0.1 mg/L for conventional water treatment plants and 0.2 mg/L for other types of treatment plants. Given that the aluminum CDWG is not based on health, it was not retained as a COPC.

[3] Given that iron's CDWG is based upon an Aesthetic Guideline value and is not based on health, it was not retained as a COPC.

Table B.2: Screening of Maximum Predicted Soil Concentrations

Parameter	CCME Soil Guidelines (mg/kg) [a]	US EPA - Regional Screening Levels [b] (mg/kg)	Background + 10% [g]	Gameti	Whati	Hislop Lake	Marian River	Bea Lake	Worker Camp	MPOI	Retained as a COPC?
Total Metals											
Aluminum (Al)	NV	77000	23977	21797	21797	21821	21840	21819	24360	26821	No
Antimony (Sb)	NV	6.2	1.0	0.9	0.9	0.9	0.9	0.9	1.5	2.0	No
Arsenic (As)	12	--	167	152	152	153	153	153	**225**	**281**	**Yes**
Barium (Ba)	NV	3000	318	289	289	289	289	289	316	343	No
Beryllium (Be)	4	--	1.0	0.9	0.9	0.9	0.9	0.9	1.0	1.1	No
Bismuth (Bi)	NV	NV	3.8	3.4	3.4	3.7	3.9	3.7	33	60	No
Boron (B)	NV	3200	26	24	24	24	24	24	24	24	No
Cadmium (Cd)	14	--	0.28	0.28	0.26	0.25	0.25	0.25	0.29	0.31	No
Calcium (Ca)	NV	NV	5494	4995	4995	5004	5010	5002	5890	6665	No
Chromium (Cr)	220	--	20	18	18	18	18	18	20	23	No
Cobalt (Co)	50	--	41	37	37	37	37	37	46	**53**	**Yes**
Copper (Cu)	1100	--	160	145	145	145	146	145	152	159	No
Gallium (Ga)	NV	NV	NV	0.0046	0.0008	0.0002	0.0003	0.0003	0.005	0.007	No
Indium (In)	NV	NV	NV	0.03	0.005	0.001	0.002	0.002	0.03	0.04	No
Iron (Fe)	NV	11000	7454	6777	6777	6828	6866	6822	**12143**	**17104**	**Yes**
Lead (Pb)	140	--	7.4	6.8	6.7	6.7	6.8	6.7	7.1	7.4	No
Lithium (Li)	NV	32	5.6	5.1	5.1	5.1	5.1	5.1	5.9	6.6	No
Magnesium (Mg)	NV	NV	NV	0.04	0.05	11	20	10	1218	2380	No
Manganese (Mn)	NV	360	773	703	703	703	703	703	732	757	No
Mercury (Hg)	6.6	--	0.18	0.16	0.16	0.16	0.16	0.16	0.16	0.17	No
Molybdenum (Mo)	10	--	2.64	2.40	2.40	2.40	2.40	2.40	2.65	2.87	No
Nickel (Ni)	50 [c]	--	11	9.76	9.76	9.76	9.77	9.76	10	11	No
Palladium (Pd)	NV	NV	NV	0.005	0.0008	0.0002	0.0003	0.0003	0.005	0.007	No
Phosphorus (P)	NV	NV	NV	0.005	0.001	0.09	0.17	0.09	11	21	No
Potassium (K)	NV	NV	NV	0.08	0.1	23	43	22	2598	5119	No
Selenium (Se)	80	--	0.99	0.9	0.9	0.9	0.9	0.9	1.1	1.4	No
Silicon (Si)	NV	NV	NV	0.3	0.05	0.01	0.02	0.02	0.33	0.44	No
Silver (Ag)	20	--	0.23	0.22	0.21	0.21	0.21	0.21	0.23	0.24	No
Sodium (Na)	NV	NV	NV	0.01	0.01	2.51	4.46	2.27	268	519	No
Strontium (Sr)	NV	9400	84	76	76	76	76	76	77	78	No
Thallium (Tl)	1	NV	0.26	0.2	0.2	0.2	0.2	0.2	0.3	0.3	No
Tin (Sn)	50	--	0.61	0.6	0.6	0.6	0.6	0.6	0.8	1.1	No
Titanium (Ti)	NV	28000	627	570	570	571	572	571	656	736	No
Uranium (U)	23	--	21	19	19	19	19	19	20	20	No
Vanadium (V)	130 [c]	--	28	26	26	26	26	26	27	29	No
Yttrium (Y)	NV	NV	NV	0.00002	0.00003	0.01	0.01	0.01	0.70	1.3	No
Zinc (Zn)	200 [c]	--	56	51	51	51	51	51	53	54	No
Dioxins and Furans											
Total Dioxins and Furans	4.0E-09	--	NV	3.5E-13	5.7E-13	3.3E-12	4.8E-12	6.6E-12	1.9E-11	1.2E-10	No
Polycyclic Aromatic Hydrocarbons											
2-Methylnaphthalene	NV	NV	NV	6.1E-06	9.9E-06	2.0E-04	2.5E-04	1.7E-04	3.0E-03	1.1E-02	No
Acenapthene	NV	68	1.1E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	No
Acenapthylene	NV	NV	1.1E-03	1.9E-03	1.1E-03	1.0E-03	1.1E-03	1.1E-03	2.0E-03	2.5E-03	No
Anthracene	NV	340	1.1E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	No
Benzo(a)anthracene	NV	0.15	6.5E-02	5.9E-02	5.9E-02	5.9E-02	5.9E-02	5.9E-02	5.9E-02	5.9E-02	No
Benzo(a)pyrene	NV	0.015	2.1E+00	**1.9E+00**	**1.9E+00**	**1.9E+00**	**1.9E+00**	**1.9E+00**	**1.9E+00**	**1.9E+00**	**Yes**
Benzo(b+k)fluoranthene	NV	0.15 [d]	1.3E-01	1.1E-01	1.1E-01	1.1E-01	1.1E-01	1.1E-01	1.1E-01	1.1E-01	No
Benzo(g,h,i)perylene	NV	NV	3.4E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02	3.1E-02	No
Chrysene	NV	15	4.4E-02	4.0E-02	4.0E-02	4.0E-02	4.0E-02	4.0E-02	4.0E-02	4.0E-02	No
Dibenzo(a,h)anthracene	NV	0.015	2.1E-02	**1.9E-02**	**1.9E-02**	**1.9E-02**	**1.9E-02**	**1.9E-02**	**1.9E-02**	**1.9E-02**	**Yes**
Fluoranthene	NV	46	3.0E-01	2.8E-01	2.8E-01	2.8E-01	2.8E-01	2.8E-01	2.8E-01	2.8E-01	No
Fluorene	NV	46	3.0E-02	2.7E-02	2.7E-02	2.7E-02	2.7E-02	2.7E-02	2.7E-02	2.7E-02	No
Indeno(1,2,3-c,d)pyrene	NV	0.15	4.1E-02	3.7E-02	3.7E-02	3.7E-02	3.7E-02	3.7E-02	3.7E-02	3.7E-02	No
Naphthalene	0.6	--	1.3E-02	1.1E-02	1.1E-02	1.1E-02	1.1E-02	1.1E-02	1.1E-02	1.2E-02	No
Phenanthrene	NV	34	2.0E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	No
Pyrene	NV	34	1.1E-03	1.1E-03	1.0E-03	1.0E-03	1.0E-03	1.0E-03	1.1E-03	1.2E-03	No
B[a]P TPE	5.3	--	2.12	1.93	1.93	1.93	1.93	1.93	1.93	1.93	No
IACR	1.0	--	6.73	**6.12**	**6.12**	**6.12**	**6.12**	**6.12**	**6.12**	**6.12**	**Yes**

Notes

3.5 Outline and bold font indicates value >baseline+10% and >guideline.

NV = No Value

(a) CCME Soil Quality Guidelines for the Protection of Environmental and Human Health - Residential/Parkland

(b) US EPA Regional Screening Levels were obtained where no CCME value is available; adjusted to an HQ of 0.2.

(c) SQG_{ECO} residential/parkland

(d) US EPA RSL for benzo(b)fluoranthene was more conservative

(e) B[a]P TPE = Benzo(a)pyrene Total Potency Equivalents (to be used for soil contaminated with coal tar or creosote mixtures) (CCME 2010)

(f) IACR = Index of Additive Cancer Risk (to assess potential impacts to potable groundwater quality for leaching of carcinogenic mixtures from soil) (CCME 2010)

(g) Mean background concentration used for metals; maximum background concentration used for PAH

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