APPENDIX W
ECOLOGICAL AND HUMAN HEALTH RISK ASSESSMENT

CÔTÉ GOLD PROJECT
DRAFT ENVIRONMENTAL ASSESSMENT REPORT
HUMAN AND ECOLOGICAL HEALTH RISK ASSESSMENT

Version 4

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GLOSSARY AND ABBREVIATIONS

AAQC	Ambient Air Quality Criteria
ACGIH	American Conference of Industrial Hygienists
ATSDR	Agency for Toxic Substances and Disease Registry
BMC	Benchmark Concentration
Cal EPA	California Environmental Protection Agency
CARB	California Air Resource Board
CCME	Canadian Council of Ministers of the Environment
CEAA	Canadian Environmental Assessment Act
CEPA	Canadian Environmental Protection Act
CICADS	Concise International Chemical Assessment Documents
cm	Centimetres
CNAAQO	Canadian National Ambient Air Quality Objective
COHb	Carboxyhemoglobin
CNS	Central Nervous System
CM	Conceptual Model
CWQG	Canadian Water Quality Guideline
CWS	Canada Wide Standard
DEQ	Michigan Department of Environmental Quality
DQRA	Detailed Quantitative Risk Assessment
EA	Environmental Assessment
EEG	Electroencephalography
EHC	Environmental Health Criteria
EHRA	Environmental Health Risk Assessment
EIS	Environmental Impact Statement
EPC	Exposure Point Concentration
ER	Exposure Ratio
ESOD	Erythrocyte Superoxide Dismutase
g	Gram
$g/m^2/year$	Grams per square metre per year
HHRA	Human Health Risk Assessment
HQ	Hazard Quotient
hr	Hour
IARC	International Agency for Research on Cancer
ICSC	International Chemical Safety Cards
ILCR	Incremental Lifetime Cancer Risk
IPCS	International Programme on Chemical Safety
ITSL	Initial Threshold Screening Level
JECFA	Joint Expert Committee on Food Additives
LOAEL	Lowest Observed Adverse Effect Level
m	Metre
m^3/day	Cubic metre per day

MAC	Maximum Acceptable Concentration
mg/kg	Milligram per kilogram
mg/L	Milligram per litre
MOE	Ontario Ministry of the Environment
MRL	Minimal Risk Level
NIOSH	National Institute of Occupational Safety and Health
NOAEC	No Observed Adverse Effect Concentration
NOAEL	No Observed Adverse Effect Level
OEHHA	California Office of Environmental Health Hazards Assessment
$PM_{2.5/10}$	Particulate Matter (particles) less than 2.5 or 10 micrometres in diameter
ppm	Parts Per Million
PQRA	Preliminary Quantitative Risk Assessment
PWQG	Provincial Water Quality Guideline
PWQO	Provincial Water Quality Objectives for the protection of aquatic life
REL	Reference Exposure Level
RIVM	Dutch National Institute for Public Health and Environment
SCOEL	European Scientific Committee on Occupational Exposure Levels
TAC	Tolerable Concentration in Air
TLV	Threshold Limit Value
ToR	Terms of Reference
TRV	Toxicity Reference Value
TSD	Technical Support Document
µg/day	Micrograms (one-millionth of a gram) per day
µg/dL	Micrograms (one-millionth of a gram) per decilitre
$\mu g/m^3$	Micrograms (one-millionth of a gram) per cubic metre
µm	Micrometre
US EPA	United States Environmental Protection Agency
US EPA IRIS	United States Environmental Protection Agency Integrated Risk Information System
US NAAQGS	United States National Ambient Air Quality Guideline Standards
WHO	World Health Organization
WQ/AQOG	Federal Provincial Advisory Committee Working Group on Water/Air Quality Objectives and Guidelines

EXECUTIVE SUMMARY

IAMGOLD Corporation (IAMGOLD) proposes to construct, operate and eventually rehabilitate a new open pit gold mine in the Chester and Neville Townships, District of Sudbury, in northeastern Ontario, approximately 20 kilometres (km) southwest of Gogama, 130 km southwest of Timmins, and 200 km northwest of Sudbury. Currently, the Project is required to complete a Federal Environmental Assessment (EA) as per the Canadian Environmental Assessment Act, 2012. As well, IAMGOLD entered into a Voluntary Agreement to conduct an Individual Provincial EA for the Côté Gold Project. Therefore, to support the environmental assessment of the Project as outlined in the Terms of Reference (ToR) approved by the Ontario Minister of the Environment and in the Environmental Impact Statement (EIS) Guidelines issued by the Canadian Environmental Assessment Agency (the Agency) in July 2013, a Human and Ecological Health Risk Assessment has been completed to better understand the potential risks to human and ecological receptors associated with the Project.

This technical support document (TSD) (Human and Ecological Health Risk Assessment) has been prepared by AMEC Environment & Infrastructure, and is one in a series of technical reports to support the environmental assessment (EA) of the Project as outlined in the Terms of Reference (ToR) approved by the Ontario Minister of the Environment in January 2014, and in the Environmental Impact Statement (EIS) Guidelines issued by the Canadian Environmental Assessment Agency (the Agency) in July 2013.

To determine the potential effects that the various Project phases may have on air, water and soil quality as a result of air dispersion and deposition, and effluent discharge/runoff, predictive air dispersion and deposition modelling (see Appendix F) and water quality modelling (see Appendix J) were conducted. While the results of this modelling are presented as separate reports and are being used in support of obtaining the required permits, results have also been used as inputs to the human and ecological health risk assessment which were undertaken to better understand risks to human and ecological receptors.

1.0 RISK ASSESSMENT FRAMEWORK AND GENERAL APPROACH

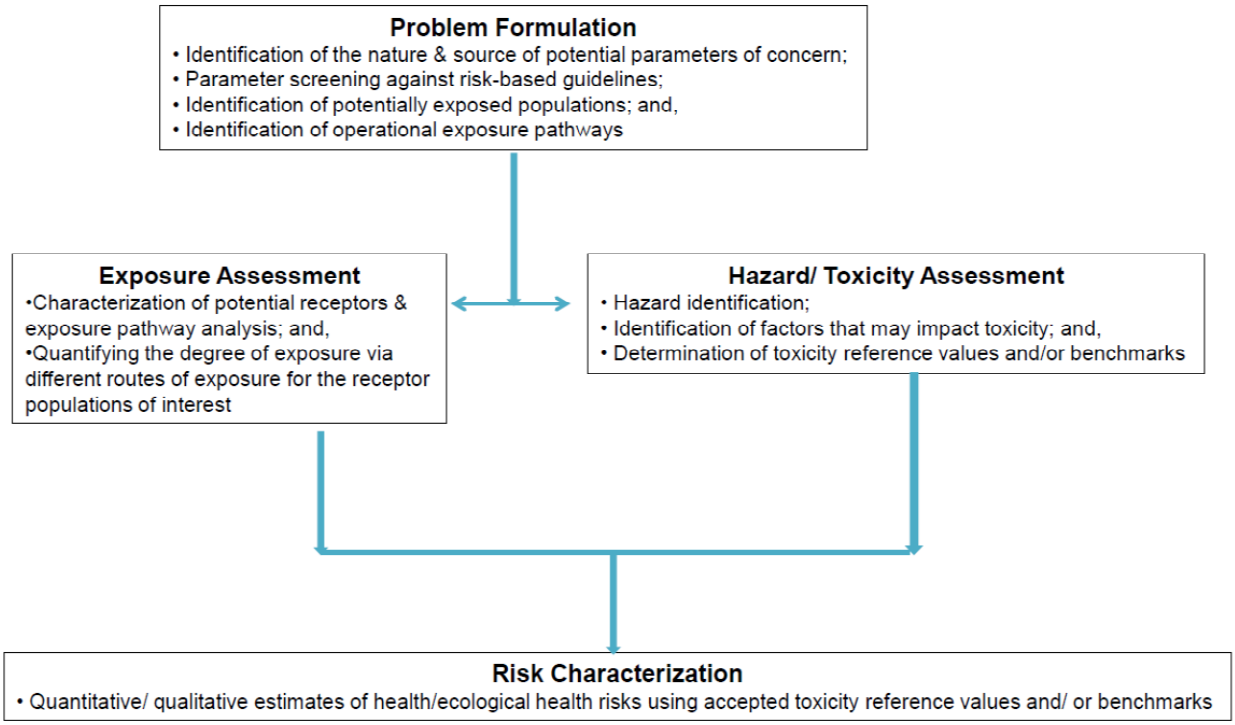
A risk assessment is a process used to assess the potential risks to human and ecological receptors resulting from one or more environmental stressors. In doing so, the risk assessment takes into account the chemicals to be evaluated, their toxicity and the manner in which receptors may be exposed. As risk assessments are considered “forward looking”, they predict what could happen under a certain set of circumstances. They are based on assumptions concerning how much of a chemical might be present, and how ecological and human receptors may be exposed to that chemical.

Risk assessments typically employ assumptions that result in estimates of exposure that overestimate the potential for human health and ecological risks. These are often referred to as “worst case” exposure conditions. This does not mean that actual conditions are expected to reflect these worst case assumptions; rather, it means that the exposure assumptions used in the assessment are meant to represent conditions that overestimate the extent of exposure and risk. Worst case exposure assumptions are used to focus on those chemicals and exposure conditions that may represent a risk and screen out those that do not. If potential risks are within acceptable limits using “worst case” assumptions, then it can be concluded that risks will also be within acceptable limits when using assumptions more in line with exposure conditions likely to be experienced in the potentially affected area surrounding the Project. In contrast, if the potential for unacceptable risks are identified using worst case assumptions, then it is important to examine the assumptions used in the assessment to better understand the sources of those risks and whether mitigative measures are warranted under the circumstances.

The human health risk assessment (HHRA) has followed the general approach recommended by *Health Canada Part I and V: Guidance on Human Health Preliminary/Detailed Quantitative Risk Assessment* (PQRA/DQRA; Version 2.0; 2010 and 2012). The ecological health risk assessment (EHRA) has followed the guidance established under the Federal Contaminated Sites Action Plan (Azimuth, 2012) and supplementary guidance provided by Environment Canada (i.e., a Framework for Ecological Risk Assessment: General Guidance) (CCME, 1996, 1997). In addition, guidance from the Ontario Ministry of the Environment (MOE, 2011) *Rationale for the Development of Soil, and Ground Water Standards for Use at Contaminated Sites in Ontario* has also been relied upon where relevant.

Based on this guidance, the principal elements of the human and ecological health risk assessments followed the broad steps summarized in Graphic 1.

Graphic 1: Risk Assessment Paradigm



This approach has been applied to both the human and ecological health risk assessments as discussed in the following sections.

2.0 HUMAN HEALTH RISK ASSESSMENT

The problem formulation provides the framework and methodology for the Human Health Risk Assessment (HHRA), and consists of identifying the relevant components of the HHRA. These components include reviewing relevant Project site information, identifying and screening the parameters of concern for human health, identifying and characterizing human receptors present in the study area and identifying the potential exposure pathways that are operational. Section 2.1 describes in more detail the problem formulation for the current HHRA.

2.1 PROBLEM FORMULATION

2.1.1 Study Objectives and Methodology

The human health risk assessment (HHRA) has followed the general approach recommended by *Health Canada Part I and V: Guidance on Human Health Preliminary/Detailed Quantitative Risk Assessment* (PQRA/DQRA, Version 2.0; 2010 and 2012).

The objectives of the Human Health Risk Assessment (HHRA) are to qualitatively and quantitatively evaluate the potential for adverse health effects to human receptors resulting from the emissions and discharges related to Project activities.

2.1.2 Study Area and Potential Exposure Pathways

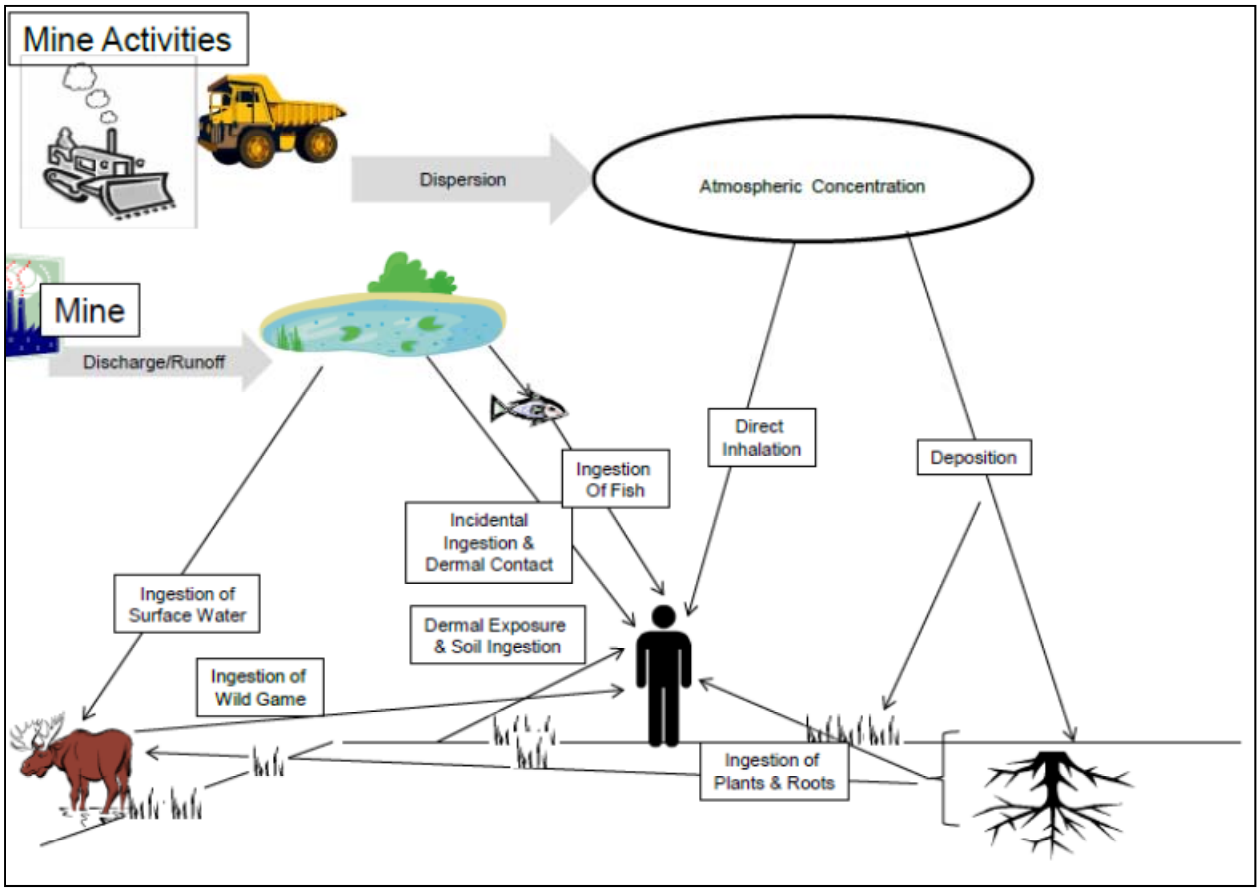
The local study area as defined in the Air Quality Technical Support Document (Appendix F) consists of natural areas with few access restrictions other than those in place at the Project site. In close vicinity to the Project site are areas that are used for recreational and/or traditional uses including hunting, trapping, fishing, camping and canoeing. Within the vicinity of the Project site there are also various cottages and hunter/trapper cabins that are occupied seasonally. Therefore, in the vicinity of the Project site, hunter/anglers or seasonal cottagers who visit the area for recreational activities such as fishing, hunting, camping, etc. are expected to be present. In addition a member of a First Nations group may visit the surrounding Project site area for the purpose of hunting, fishing and gathering of traditional vegetation for subsistence.

These receptors could come into contact with emissions and discharges originating from the Project site through various pathways including the following:

- direct inhalation of airborne emissions;
- deposition of emissions to soil with subsequent direct contact (e.g., dermal contact, incidental ingestion and inhalation of re-entrained dust);
- direct ingestion of surface water;
- incidental ingestion and dermal contact of surface water;
- ingestion of fish and wild game; and
- ingestion of plants (e.g., berries, below-ground and above-ground plants).

The conceptual exposure model for the human health risks assessment is provided in Graphic 2.

Graphic 2: Human Health Conceptual Exposure Model



2.1.3 Parameters of Potential Concern

2.1.3.1 Air

Air emissions will be generated as a result of activities occurring from all phases of the Project. Some of the main sources of emissions will originate as a result of: blasting; material handling in the open pit; crushing; road traffic; managing mine rock, ore and overburden; and, exhaust from back-up power generation. A detailed assessment of the air emissions arising from the Project can be found in Appendix F. For each of those substances that were expected to be emitted in appreciable quantities, dispersion modelling was conducted providing predicted airborne concentrations at the maximum point of impingement (defined as being outside of the Project site) as well as the location of different receptors in the study area for different averaging times (1-hour, 24-hours and annual). Modelling also includes an evaluation of deposition to understand potential impacts to soil quality resulting from the deposition of contaminants of concern to soil.

Predicted maximum ground level concentrations for the substances expected to be emitted from the Project are provided in Appendix F. These substances include criteria air contaminants as well as various inorganic chemicals. In addition, a number of sensitive receptor locations (i.e., cottages) were identified and maximum ground level concentrations for the substances expected to be emitted from the Project at these locations were modeled and are provided in Appendix F. Modelled parameters were compared to ambient air quality criteria in addition to being used as inputs to a direct evaluation of human health risk. Tables 1, 2 and 3 provide a summary of the parameters and their expected concentrations at the maximum point of impingement as well as different receptor locations of interest.

Table 1: Predicted Air Emissions at the Maximum Point of Impingement

Parameter	Modelled Concentration at the Maximum Point of Impingement ($\mu g/m^3$)	Averaging Period
Particulate Matter (<10 µm) (PM_{10})	113	24-hr
Particulate Matter (<2.5 µm) ($PM_{2.5}$)	30.4	24-hr
	3.84	annual
Nitrogen Dioxide	304	1-hr
	101	24-hr
Carbon Monoxide	2640	1-hr
	1680	8-hr
Sulphur Dioxide	36.4	24-hr
	165	1-hr
	4.68	annual
Arsenic	0.000849	24-hr
	0.000092	annual
Calcium Oxide	39.3	1-hr
	8.67	24-hr
Chromium	0.0375	24-hr
Copper Sulphate	11.2	1-hr
	2.48	24-hr
Hydrogen Cyanide	7.61	24-hr
Mercury	0.00001975	24-hr
Magnesium	5.92	24-hr
Manganese (in $PM_{2.5}$)	0.0287	24-hr
Nickel (in Total Suspended Particulates)	0.0112	24-hr
Nickel (in Total Suspended Particulates)	0.00122	annual
Lead	0.000258	30-day
	0.00118	24-hr
Titanium	1.06	24-hr
Zinc	0.0161	24-hr

Table 2: Predicted Air Emissions at the Sensitive Receptor Location - Criteria Air Contaminants

Parameter	Modelled Concentration of the Criteria Air Contaminants at the Sensitive Receptor Location (µg/m^3)								Averaging Period (hr - unless noted otherwise)
	POR01	POR02	POR03	POR04	POR05	POR06	POR07	POR08	
Particulate Matter (PM_{10})	22.3	**23.8**	16.1	5.73	7.81	11	17	23.1	24-hr
Particulate Matter ($PM_{2.5}$)	7.08	**11.4**	6.23	2.24	3.10	4.20	7.64	7.14	24-hr
	0.297	0.459	0.161	0.107	0.192	0.347	0.67	**0.755**	annual
Nitrogen Dioxide	128	**149**	128	108	104	133	147	**149**	1-hr
	28.4	27.8	19.6	10.8	24.9	25.5	29.3	**31.6**	24-hr
Carbon Monoxide	734	899	818	752	579	729	895	**914**	1-hr
	204	220	184	96.5	152	186	**251**	224	8-hr
Sulphur Dioxide	76.6	**80.7**	51.3	45.9	34.2	48.2	66.8	61	1-hr
	6.78	**7.82**	5.43	2.08	3.22	5.03	7.36	6.41	24-hr
	0.317	0.475	0.157	0.106	0.194	0.329	0.504	**0.514**	annual

Bold- indicates maximum value

Table 3: Maximum Predicted Air Emissions at the Sensitive Receptor Location – Inorganics

Parameters	Receptor ID	Maximum Modelled Concentration at the Sensitive Receptor Location ($\mu g/m^3$)	Averaging Period
Arsenic	POR08	0.000142	24-hr
Calcium Oxide	POR02	1.83	24-hr
Chromium	POR08	0.00629	24-hr
Copper Sulphate	POR02	0.524	24-hr
Hydrogen Cyanide	POR07	2.23	24-hr
Mercury	POR08	0.00000331	24-hr
Magnesium	POR08	0.992	24-hr
Manganese (in $PM_{2.5}$)	POR07	0.0108	24-hr
Nickel (in Total Suspended Particulates)	POR08	0.00189	24-hr
Nickel (in Total Suspended Particulates)	POR08	0.000221	annual
Lead	POR07	0.000309	30-day
	POR08	0.000198	24-hr
Titanium	POR08	0.177	24-hr
Zinc	POR08	0.0027	24-hr

2.1.3.2 Soil

Air emissions resulting from the Project and Project-related activities may deposit as particulates to the soil. The presence of these substances in soil could then be available for uptake by human receptors via various pathways including consumption of traditional vegetation (e.g., berries) grown in the soil, consumption of wild game (e.g., moose) that has consumed vegetation grown in the soil and/or soil organisms or mammals/birds present in the area, and via direct contact and inhalation of soil particles.

Therefore, to determine what the predicted concentrations of inorganics in soil are as a result of air deposition from the Project, depositional modelling was conducted at the maximum point of impingement and at the receptor locations deemed “sensitive” as they are occupied by seasonal cottagers and campers.

To determine the concentration of these parameters in soil resulting from deposition, the following equation from US EPA (2005) was utilized:

$$C_{s=100} \times \frac{Dyd+Dyw}{Z_s \times BD} \times tD$$

Where:

C_s= Predicted soil concentration over exposure duration (mg of the parameter of interest/kg soil);
100 = Units conversion factor (mg-m^2/kg-cm^2);
Dyd = Yearly dry deposition rate of pollutant (g/m^2-yr);
Dyw = Yearly wet deposition rate of pollutant (g/m^2-yr);
Z_s = Soil mixing zone depth (cm) (assume 1 cm mixing for direct ingestion of soil;
BD = Soil bulk density (g soil/cm^3 soil) (assume 1.5 g soil/cm^3); and,
tD = Time period over which deposition occurs (time period of combustion) (assume 15 yrs).

Predicted concentrations of inorganics in soil at both locations were compared to background soil concentrations (Table 4) obtained from the Ontario Ministry of the Environment. These are considered protective of human health and ecological receptors for all pathways of exposure (MOE, 2011).

Table 4: Predicted Soil Concentrations Resulting from Atmospheric Deposition- Maximum Point of Impingement and Sensitive Receptor Location

Parameter	MOE (2011) Background Soil Concentration (mg/kg)	Maximum Point of Impingement Deposition (g/m^2/year) (Dry & Wet Deposition)	Maximum Point of Impingement Soil Concentration Resulting from Deposition (mg/kg)	Maximum Point of Impingement Soil Concentration Above MOE (2011) Background Soil Concentration?	Maximum Sensitive Receptor Location Deposition (g/m^2/year) (Dry & Wet Deposition)	Maximum Sensitive Receptor Location Soil Concentration Resulting from Deposition (mg/kg)	Maximum Sensitive Receptor Location Soil Concentration Above MOE (2011) Background Soil Concentration?
Arsenic	18	0.000164	0.16	No	0.0000139	0.01	No
Chromium	70	0.00726	7.26	No	0.000616	0.62	No
Magnesium	15000	1.15	1146	No	0.0972	97.2	No
Manganese	1400	0.0361	36.09	No	0.00306	3.06	No
Mercury	0.27	0.00000382	0.0038	No	0.000000324	0.00032	No
Nickel	82	0.00218	2.18	No	0.000185	0.18	No
Lead	120	0.000229	0.23	No	0.0000194	0.02	No
Zinc	290	0.00311	3.11	No	0.000264	0.26	No

2.1.3.3 Surface Water

Surface water quality modelling was conducted to predict changes that may occur as a result of the Project (see the Water Quality Technical Support Document, Appendix J). The maximum predicted concentrations of major ions, nutrients and metals occurring during each of the Project phases were compared to the Ontario Drinking Water Standard and Canadian Water Quality Guidelines which are protective of human health. In cases where the human health benchmark is based on an aesthetic objective or an operational guideline which does not impact human health, and the predicted concentrations in surface water exceeded these guidelines, then the most recent Provincial Water Quality Objective (OMOEE PWQO) or the Canadian Water Quality Guidelines (CCME WQG), or the British Columbia Ministry of the Environment Water Quality Guideline (BC MOE WQG) was used or if not available the 95th percentile baseline concentration was used for comparison (see Table 5).

Table 5: Comparison of Predicted Surface Water Concentrations of Various Parameters to Human Health Benchmarks

Parameter	95th Percentile Baseline Concentration (mg/L)	Human Health Benchmark (mg/L)*	Rationale for Selection of Benchmark for Comparison	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Aluminum	0.1182	0.1182	Drinking water guideline is based on an operational value (<0.1[a]). Therefore, screening value has been set at the 95th percentile baseline concentration of 0.1182 mg/L.	0.08	0.11	0.08	No
Ammonia (Total)	0.21	6.89	No drinking water guideline is available. Therefore, screening value is based on the CCME Water Quality Guideline of 6.89 mg/L.	0.44	0.42	0.48	No
Ammonia (Unionized)	0.00049	0.019	No drinking water guideline is available. Therefore, screening value is based on the CCME Water Quality Guideline of 0.019 mg/L.	0.002	0.002	0.003	No
Antimony	<0.006	0.006[b]	Screening value is based on an interim MAC.	0.001	0.001	0.002	No
Arsenic	<0.003	0.025[b]	Screening value is based on an interim MAC.	0.005	0.005	0.006	No
Barium	0.007	1[c]	Screening value is based on an established MAC.	0.01	0.01	0.01	No
Boron	<0.01	5[b]	Screening value is based on an interim MAC.	0.01	0.01	0.01	No
Cadmium	0.00005	0.005[c]	Screening value is based on an established MAC.	0.00004	0.00005	0.00003	No
Calcium	10.465	10.465	No drinking water guideline is available. Therefore, screening value has been set at the 95th percentile baseline concentration of 10.465 mg/L.	29.04	27.22	41	Yes

Parameter	95th Percentile Baseline Concentration (mg/L)	Human Health Benchmark (mg/L)*	Rationale for Selection of Benchmark for Comparison	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Chloride	1.2	120	Screening value is based on an aesthetic objective for drinking water (≤250[d]). Therefore, screening value is based on the CCME Water Quality Guideline of 120 mg/L.	1.63	1.99	2.1	No
Cobalt	0.00025	0.0025	No drinking water guideline is available. Therefore, screening value is based on the CCME Water Quality Guideline of 0.0025 mg/L.	0.0005	0.0006	0.0005	No
Copper	0.001	0.005	Screening value is based on an aesthetic objective for drinking water (≤1[d]). Therefore, screening value is based on the OMOEE PWQO of 0.005 mg/L.	0.003	0.003	0.004	No
Cyanide (Free)	--	0.2[c]	Screening value is based on an established MAC.	0.01	0.01	0.01	No
Iron	0.369	0.369	Drinking water guideline is based on an aesthetic objective (≤0.3[d]). Therefore, screening value has been set at the 95th percentile baseline concentration of 0.369 mg/L.	0.30	0.39	0.28	Yes
Lead	0.0005	0.01[c,e]	Drinking water guideline is based on a MAC at the point of consumption.	0.0001	0.0001	0.0001	No
Magnesium	2.003	NV	No drinking water guideline is available. Therefore, screening value has been set at the 95th percentile 2.003 mg/L.	2.40	3.42	2.42	Yes

Parameter	95th Percentile Baseline Concentration (mg/L)	Human Health Benchmark (mg/L)*	Rationale for Selection of Benchmark for Comparison	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Manganese	0.0878	0.7	Screening value is based on an aesthetic objective for drinking water (≤0.05[d]). Therefore, screening value is based on the BCMOE Water Quality Guideline of 0.7 mg/L.	0.11	0.14	0.10	No
Molybdenum	<0.002	0.073	No drinking water guideline is available. Therefore, screening value is based on the CCME Water Quality Guideline of 0.073 mg/L.	0.003	0.003	0.004	No
Nickel	0.0015	0.025	No drinking water guideline is available. Therefore, screening value is based on the OMOEE PWQO of 0.025 mg/L.	0.003	0.004	0.003	No
Nitrate	0.13	10[e,f]	Drinking water guideline should not exceed 10 mg/L for both nitrate and nitrite and is based on the point of consumption.	1.36	0.26	1.7	No
Phosphorus (total)	0.035	0.035	No drinking water guideline is available. Therefore, screening value has been set at the 95th percentile baseline concentration of 0.035 mg/L.	0.05	0.06	0.06	Yes
Potassium	0.49	373	No drinking water guideline is available. Therefore, screening value is based on the BC MOE water quality guideline of 373 mg/L as no CCME or OMOEE PWQO are available for potassium.	1.75	1.8	2.5	No

Parameter	95th Percentile Baseline Concentration (mg/L)	Human Health Benchmark (mg/L)*	Rationale for Selection of Benchmark for Comparison	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Sodium	1.3365	1.3365	Screening value is based on an aesthetic objective for drinking water (≤200[d]). Therefore, screening value has been set at the 95th percentile baseline concentration of 1.3365 mg/L.	2.51	3.79	2.60	Yes
Strontium	0.026	0.026	No drinking water guideline is available. Therefore, screening value has been set at the 95th percentile baseline concentration of 0.026 mg/L.	0.05	0.05	0.07	Yes
Sulphate	4.092	≤500[d,g]	Drinking water guideline is based on an aesthetic objective. However, effects may occur in some individuals if concentrations exceed 500 mg/L.	7.1	10.7	7.34	No
Uranium	<0.002	0.02*	Drinking water guideline is based on a MAC	0.004	0.004	0.01	No
Vanadium	<0.002	0.006	No drinking water guideline is available. Therefore, screening value is based on the OMOEE PWQO of 0.006 mg/L.	0.002	0.003	0.003	No
Zinc	0.032	0.032	Screening value is based on an aesthetic objective for drinking water (≤5[d]). Therefore, screening value has been set at the 95th percentile baseline concentration of 0.032 mg/L.	0.02	0.02	0.02	No

NV- No Value

* Based on the Ontario Drinking Water Standard and Canadian Water Quality Guidelines.

a. operational guideline

b. Interim Maximum Acceptable Concentration- insufficient toxicological data to establish a Maximum Acceptable Concentration with reasonable certainty, or not feasible for practical reasons to establish a MAC at the desired level.
c. Maximum Acceptable Concentration- The length and time the MAC can be exceeded without health effects will depend on the nature and concentration of the parameter.
d. Aesthetic Objective
e. Applies to point of consumption
f. Nitrate + Nitrogen should not exceed 10 mg/L.
g. When sulphate levels exceed 500 mg/L, water may have a laxative effect on some people.

Note, the maximum concentrations do not take into account the data at the point of discharge as these concentrations are not expected to be representative concentrations of parameters that individuals would potentially be exposed to in the Project area. However, the closest area located downstream to the point of discharge (approximately 500 m away) where individuals may come into contact with surface water has been considered.

2.2 EXPOSURE ASSESSMENT

The exposure assessment component of the HHRA is intended to estimate potential exposures for the receptors that could be expected to come into contact with parameters of potential concern in the local study area. It describes the receptors and the exposure pathways that could contribute to exposure and uses this information to estimate the potential exposure for each type of receptor. The exposure assessment also provides an indication of the relative contribution that each exposure pathway makes to the total daily exposure experienced by each type of receptor. The exposure assessment can be summarized by these three basic components:

- Characterization of Parameters of Potential Concern;
- Characterization of Potential Receptors and Exposure Pathway Analysis; and
- Exposure Estimates.

Each of the three components of the exposure assessment is detailed below.

2.2.1 Characterization of Parameters of Potential Concern

Exposure point concentrations (EPCs) are concentrations in each relevant media to which receptors were assumed to be exposed. Consistent with the Health Canada (2012) guidance, the maximum concentrations have been used in the exposure assessment.

For air, maximum concentrations were identified in the following tables:

- Table 1 – Predicted air emissions at the maximum point of impingement; and
- Tables 2 & 3 – Predicted air emissions at sensitive receptor locations.

With respect to air emissions all of the modelled parameters were considered “of potential concern” and were assessed in the HHRA. Although comparison could be made to the Ambient Air Quality Criteria, which are protective of human health, they are not necessarily all based on human health risk thresholds. Therefore, the following parameters in air were assessed in the HHRA: Criteria Air Contaminants (PM_{10}, $PM_{2.5}$, nitrogen dioxide, carbon monoxide, sulphur dioxide); and, Inorganics (hydrogen cyanide, calcium oxide, copper sulphate, arsenic, chromium, mercury, manganese, nickel, lead, titanium, and zinc).

With respect to soil, no parameters of potential concern were identified as predicted soil concentrations of inorganics resulting from atmospheric deposition were less than soil concentrations considered “background” in the province of Ontario. As such, no adverse exposure could be attributed to emissions from the Project via soil contact pathways(see Table 4).

For surface water, maximum concentrations were identified in Table 5. Predicted concentrations were compared to drinking water guidelines that are protective of human health. In cases where

drinking water guidelines were not available, concentrations were compared to aquatic health benchmarks which are more conservative than drinking water guidelines. From the screening, parameters of potential concern in surface water include: calcium, iron, magnesium, total phosphorus, sodium and strontium.

2.2.2 Characterization of Potential Receptors

2.2.2.1 Resident – Aboriginal

In this assessment, a resident receptor encompasses all life stages (i.e., infant, toddler child, teen and adult) and has been assumed to reside in the study area all year-round. In the HHRA it is assumed that the resident engages in traditional activities, consumes locally grown/harvested foods and is exposed outdoors for 24 hours per day, 7 days per week, 52 weeks per year, for the full duration of the Project. For the purpose of amortizing exposure, the receptor is assumed to have a life expectancy of 80 years per Health Canada recommendations (2012).

2.2.2.2 Recreational – Angler/Hunter

In the study area and vicinity there are various cottages, camp sites and outfitters which accommodate recreational visitors to the area who will engage in activities such as fishing, hunting, camping, swimming, boating, etc. In this assessment, the recreational angler/hunter will include a receptor that encompasses all life stages. The cottages in the study area are not residential homes but are instead used for seasonal purposes (see Appendix O and T). Other receptor characteristics were the same as those used for the Aboriginal resident receptor Health Canada (2012).

2.2.3 Exposure Pathway Analysis

2.2.3.1 Inhalation of Exposure to Ambient Air

Exposure point concentrations were modelled for the receptors located at the maximum point of impingement (i.e., outside of the Project site) and at the sensitive receptor locations. For the purposes of modelling, it has been assumed that the modelled concentrations of each of the compounds in outdoor air are equal to that of indoor air. Therefore, exposure to the emitted compounds has been assumed to occur continuously (i.e. 24-hours per day).

Potential exposure to operations-related emissions is based on the results of air dispersion modelling which relies on an understanding of the emission rates of different contaminants from various sources and the dispersion characteristics of those contaminants under different atmospheric and physical settings. The exposure point concentrations used in the risk calculations are estimates and represent the maximum estimate of emissions. Depending on the parameter of concern, exposures were modelled for 1-hour, 24-hour and annual averaging times. The emission estimates and dispersion modelling used to develop the exposure point concentrations are described in Appendix F. For the purpose of evaluating exposure via direct inhalation, it was assumed a receptor would be exposed to the maximum predicted concentration on a continuous basis for the duration of the Project.

2.2.3.2 Direct and Indirect Exposure to Soil

As indicated above, airborne emissions resulting from the Project and Project-related activities have the potential to deposit to soil ultimately affecting soil quality and the health of any organisms that inhabit the soil, or consume plants that grow in the soil. To understand potential risk associated with this exposure pathway, deposition modelling was undertaken to provide a maximum deposition rate in terms of grams per square metre per year. Assuming incidental mixing within the first 1 cm of soil, this was used to develop an understanding of the incremental change in background soil quality over the 15-year operational phase of the facility. As there was no appreciable change to background soil quality resulting from deposition, it was concluded that exposure via this pathway would not result in "unacceptable" risk and therefore it was not considered further (see Table 4).

2.2.3.3 Direct and Indirect Exposure to Surface Water

Surface water quality modelling, which examined loadings and flow conditions in the surrounding water bodies, was used to predict concentrations of major ions, nutrients and metals occurring during each of the phases of the Project. Maximum predicted concentrations were compared to Human Health and Aquatic Health Benchmarks. Human Health Benchmarks for surface water are considered protective of all exposure pathways relevant to surface water including direct ingestion, dermal contact during swimming and indirect ingestion of fish. As the concentrations of those substances attributable to the Project in surface water are predicted to be below Human Health Based Benchmarks, it was concluded that there are no unacceptable health risks associated with discharge to surface water.

In terms of human health, the essential elements calcium, iron, magnesium, phosphorus and sodium did not have applicable Human Health Benchmarks. However, these are essential elements and are not predicted to be present at concentrations that would pose a risk to human health. With respect to strontium, potential risks were evaluated by assuming that surface water would be used as a source of drinking water with a consumption rate of 1.5 litres per day, consistent with Health Canada recommendations (Health Canada, 2012). Under such assumptions, strontium is not predicted to be present at concentrations that would present an unacceptable risk to human health.

As mercury is not expected to be present in process elements in appreciable quantities, exposure to this contaminant was not evaluated. It is noted however, that the construction of the watercourse realignments will result in the flooding of former terrestrial lands. While the areas to be inundated are prone to flooding within the baseline condition, it is possible that the decay of terrestrial vegetation will result in the production of methyl mercury that will be taken up by resident fish. The removal of vegetation prior to flooding will reduce the potential for methyl mercury production and will be undertaken prior to construction. As there are currently fish consumption advisories for mercury in lakes within the study area, the potential to affect exposure to mercury is considered minor.

2.3 HAZARD/TOXICITY ASSESSMENT

The Toxicity Assessment describes the nature of the potential adverse health effects associated with exposure to each of the identified parameters of concern. It also provides recommended toxicity reference values (TRVs) used for evaluating the relationship between predicted levels of exposure and the potential health effects associated with that exposure.

2.3.1 Toxicity Reference Values for the Protection of Human Receptors

To quantitatively address the potential health effects associated with exposure, TRVs from regulatory agencies with well-documented and reviewed sources have been used in this assessment. These include:

- Health Canada;
- US EPA IRIS (United States Environmental Protection Agency Integrated Risk Information System);
- Ontario MOE (Ministry of the Environment);
- ATSDR (The Agency for Toxic Substances and Disease Registry);
- Cal EPA (California Environmental Protection Agency); and
- WHO (World Health Organization).

TRVs for parameters of concern in the assessment were generally available from more than one source. To select the most appropriate acute and chronic TRVs, information presented from each of the regulatory agencies for each of the parameters of concern was reviewed using a weight of evidence approach. For several parameters of concern, the Ontario Ministry of the Environment recently completed comprehensive jurisdictional reviews of available criteria in support of the development of air standards and ambient air quality criteria. Where available, these were generally relied upon for the selection of TRVs as they represent the MOE's current thinking regarding the toxicity of a particular compound.

In general, TRVs can consider both acute (short term) and chronic (long term) exposure. Acute TRVs are generally threshold concentrations that can be tolerated without evidence of adverse effects based on a short duration of exposure (minutes to hours). For the purpose of this assessment, acute exposures are evaluated for 1-hour and 24-hour exposure periods where such TRVs exist. In contrast, chronic TRVs define the daily dose or exposure that can be tolerated over an extended period (years to a lifetime), that is without adverse effect. Chronic exposures are generally assessed using annual average concentrations.

In addition to TRVs for specific parameters of concern, existing air quality objectives and guidelines for criteria air contaminants have been relied upon, on the understanding that these represent desirable or acceptable levels of air quality in a community. These are assessed using the appropriate averaging period. Table 6 lists the TRVs used in this assessment.

Table 6: Summary of Acute and Chronic (1-hour, 24-hour and annual averaging times) Toxicity Reference Values for Parameters of Potential Concern Assessed in the Human Health Assessment

Parameter	Averaging Time	Toxicity Reference Value (µg/m^3)	Critical Effect	Additional Notes	Agency
Criteria Air Contaminants					
PM_{10}	24-hr	25	Health effects	—	CNAAQO, 1998
$PM_{2.5}$	24-hr	27	Health effects	Currently, the Canada Wide Standard for $PM_{2.5}$ (24-hour averaging time) is 30 µg/m^3. This value is based on the 24-hour 98th percentile annual ambient measurement averaged over three consecutive years (Health Canada, 2006). However, for 2020 a new 24-hour averaging time for $PM_{2.5}$ of 27 µg/m^3 has been proposed (CCME, 2012).	CCME, 2012
	annual	8.8	Health effects	Canada Wide Standard (2020). The 3-year average of the annual average concentrations.	CCME, 2012
Nitrogen Dioxide	1-hr	400	Health effects	Maximum acceptable level; Maximum tolerable level = 1000 µg/m^3	CNAAQO, 1989
	24-hr	200	Health effects	Maximum acceptable level; Maximum tolerable level = 300 µg/m^3	CNAAQO, 1989

Parameter	Averaging Time	Toxicity Reference Value (µg/m^3)	Critical Effect	Additional Notes	Agency
Carbon Monoxide	1-hr	15,000	Carboxyhemoglobin blood level less than 1%	Maximum desirable level; Maximum acceptable level = 35000 µg/m^3	CNAAQO, 1996
	8-hr	6,000	Carboxyhemoglobin blood level less than 1%	Maximum desirable level; Maximum acceptable level = 15000 µg/m^3; Maximum tolerable level = 20000 µg/m^3	CNAAQO, 1996
Sulphur Dioxide	24-hr	150	Health effects	Maximum desirable level; Maximum acceptable level = 300 µg/m^3; Note: MOE AAQC = 275 µg/m^3 (health and vegetation)	CNAAQO, 1989
	1-hr	450	Health effects	Maximum desirable level; Maximum acceptable level = 900 µg/m^3; Note: MOE AAQC = 690 µg/m^3 (health and vegetation)	CNAAQO, 1989
	annual	30	Health effects	Maximum desirable level; Maximum acceptable level (annual arithmetic mean) = 60 µg/m^3; Note: MOE AAQC = 55 µg/m^3 (health and vegetation)	CNAAQO, 1989

Parameter	Averaging Time	Toxicity Reference Value ($\mu g/m^3$)	Critical Effect	Additional Notes	Agency
Inorganics					
Arsenic	24-hr	0.2	Decreased fetal weight in mice	Acute value adopted	Cal EPA, 2008
	Annual	0.015	Decrease in intellectual function, adverse effects on neurobehavioral development	For assessing non-carcinogenicity	Cal EPA, 2008
	Annual	0.0033 $(\mu g/m^3)^{-1}$	Lung tumours	For assessing carcinogenicity	Cal EPA, 2009
Calcium Oxide	1-hr	100	Sensory irritation	Sensory irritation is based on an acute study (Cain et al., 2004) & no relevant respiratory effects noted among kiln workers (Toren et al., 1996). Value of 1 mg/m^3 is supported from a study whereby workers were exposed to cement dust containing a similar alkalinity. Safety factor of 10 added to account for sensitive members of the population.	Cain et al., 2004
	24-hr		Sensory irritation (acute) & decrease of lung function (long-term)		
Chromium (II and III)	24-hr	0.5	Macroscopic and microscopic inflammatory responses in the respiratory tract associated with exposure to Cr(III)	Hexavalent chromium has not been addressed as it has not been identified as a source to be emitted from the Project and Project-related activities.	MOE, 2009
Copper Sulphate	1-hr	100	Respiratory effects	—	Cal EPA, 2008
	24-hr	35	Gastrointestinal effects	Based on an intermediate oral MRL; converted using a breathing rate of 20 m^3/day and a body weight of 70 kg.	ATSDR, 2004

Parameter	Averaging Time	Toxicity Reference Value (µg/m^3)	Critical Effect	Additional Notes	Agency
Hydrogen Cyanide	24-hr	9	Thyroid enlargement and altered iodide uptake	Based on the chronic reference exposure level. MOE AAQC of hydrogen cyanide is based on the same principal study and effects and incorporates a similar uncertainty factor as utilized by Cal EPA.	Cal EPA, 2000
Magnesium	24-hr	100	Based on 1% of the ACGIH TLV of 10 mg/m^3 which is protective of adverse health effects which include: irritation of the eyes and nose and symptoms of metal fume fever	ITSL applicable for magnesium oxide and magnesium hydroxide; ITSL derived for an 8-hour averaging time = 100 µg/m^3 and adopted as the 24-hour value.	Michigan DEQ, 1994
Manganese in Particulate Matter less than 2.5 microns	24-hr	1.00E-01	Neurological effect: impairment in the eye-hand coordination	Point of departure of 84 µg/m^3 is based on the derivation by ATSDR (2008); adjusted to 30 µg/m^3 for continuous exposure	ATSDR, 2008
Mercury	24-hr	0.03	Neurotoxicity as measured by: intention tremor; memory and sleep disturbances; decreased performance on neurobehavioral tests; decreased EEG activity	—	Cal EPA, 2008
Nickel in Total Suspended Particulates	24-hr	0.2	Critical target for adverse effects from inhalation is the respiratory system; non-cancer respiratory effect is lung fibrosis	—	MOE, 2009

Parameter	Averaging Time	Toxicity Reference Value (µg/m^3)	Critical Effect	Additional Notes	Agency
Nickel in Total Suspended Particulates	annual	0.04	Critical target for adverse effects from inhalation is the respiratory system; non-cancer respiratory effect is lung fibrosis	For assessing non-carcinogenicity	MOE, 2009
Nickel	annual	5E-05 (µg/m^3)$^{-1}$	Lung cancer	For assessing carcinogenicity	MOE, 2009
Lead	30-day	0.2	Neurological effects in children	Used the Cal EPA (2001) model which determines the air lead concentration associated with a 5% probability of children's blood lead level in a reference population exceeding a pre-determined blood lead level. MOE value is updated with more relevant parameter info data	MOE, 2007
	24-hr	0.5			
Titanium	24-hr	34	Health	Health-based standard for titanium oxide	MOE, 2012
Zinc	24-hr	18	Decrease in erythrocyte superoxidase dismutase activity and serum ferritin levels	Converted from an intermediate oral MRL to an inhalation MRL using a breathing rate of 16.6 m^3/day and a body weight of 70.7 kg for an adult as per Health Canada	ATSDR, 2005

2.3.1.1 Summary of Health Effects of Parameters of Potential Concern

The following section summarizes key toxicological information for the parameters of potential concern.

Criteria Air Contaminants

Particulate Matter (less than 10 µm) and Particulate Matter (less than 2.5 µm)

Particulate matter (PM) is a complex mixture of small solid and liquid particles (excluding water) that are airborne and microscopic in size (CEPA/FPAC, 1998; US EPA, 2011). PM can consist of various components including elemental and organic carbon, oxides of silicon, alumina and iron, trace metals, sulphates, nitrates and ammonia. PM_{10} consists of particles that are less than 10 µm in diameter (mean aerodynamic diameter) or smaller. $PM_{2.5}$ consists of particles that are less than 2.5 µm in diameter. Both PM_{10} and $PM_{2.5}$ can be directly emitted to the atmosphere or formed in the atmosphere through various chemical and physical properties. Particles that are greater than 2 µm in diameter are typically related to mechanical processes such as wind erosion, road dust and construction activities. These particles tend to remain in the atmosphere for a short duration (e.g., few hours to a few days) before settling. Particles that are between 0.1-2 µm result from the coagulation of particles in the nuclei mode and from the condensation of vapours onto existing particles which may arise from combustion sources. Particles of this size can remain in the atmosphere considerably longer (e.g., days to weeks) before they are removed *via* dry or wet deposition. Anthropogenic sources of PM include road dust, and dust from construction sites and fossil fuel combustion. However, the release of dust, tends to release PM that is considered the coarse fraction (>2.5 µm). This fraction tends to contain particles that are derived from the soil/earth's crust and may be elemental in nature. The combustion of fossil fuels leads to the generation of smaller particles (<2.5 µm) which tend to consist of sulphate, nitrate, ammonium, inorganic and organic compounds and heavy metals (CEPA/FPAC, 1998; US EPA, 2011; WHO, 2005).

The fraction size of particulate matter will influence its toxicity (CEPA/FPAC, 1998; US EPA, 2011). Typically, particles that are less than 10 µm are inhalable and can reach the respiratory tract area, however, particles that are smaller than 2-3 µm are able to reach the alveoli and can potentially be absorbed into the body. The smaller particles also have a greater surface to volume ratio and can absorb larger numbers of particles such as metals, thereby influencing the overall toxicity (CEPA/FPAC, 1998; US EPA, 2011). Health effects noted from epidemiologic studies include: increased mortality due to cardio-respiratory diseases (e.g., pre-existing heart and lung disease); increased hospitalizations; decreased lung function; increased respiratory-related activity restrictions (e.g., due to irritation of the airways, coughing, difficulty breathing, etc.); decreased lung function and capacity and increased development of chronic asthma and bronchitis (CEPA/FPAC, 1998; US EPA, 2011; WHO, 2005).

The Canadian Council of Ministers of the Environment (CCME) established a Canada Wide Standard for $PM_{2.5}$ of 30 $\mu g/m^3$ based on the adverse effects on human health and the environment (MOE, 2011). Compliance with the CWS is based on the 98th percentile annual

ambient measurement averaged over three consecutive years. Provinces were required to meet the CWS for $PM_{2.5}$ by 2010 and to begin reporting by 2011. While it is recognized that health effects may occur at exposures less than 30 µg/m^3, CCME rationalized the CWS on the basis that it represents a concentration that is achievable, taking into consideration natural as well as transboundary sources. According to the CCME, the CWS represents "*...a balance between the desire to achieve the best health and environmental protection possible in the relative near-term, and the feasibility and costs of reducing the pollutant emissions that contribute to elevated levels of PM and ozone in ambient air*." The CWS is currently used by the MOE to assess air quality impacts associated with $PM_{2.5}$ (MOE, 2011). However, new standards as set out in the *Canadian Environmental Protection Act* (2013) have set a 24-hour averaging time of 28 µg/m^3 for 2015 and 27 µg/m^3 for 2020 for $PM_{2.5}$.

With respect to PM_{10}, the Federal Provincial Advisory Committee Working Group on Air Quality Objectives and Guidelines (WGAQOG) established under CEPA developed a reference level of 25 µg/m^3 for PM_{10} based on the observed relationships between observations of adverse health outcomes and concentrations of PM_{10} in ambient air. More recent studies have focussed on the contribution of the $PM_{2.5}$ fraction to the health outcomes observed. As such, CCME has concluded that owing to the uncertainty in the causality of PM_{10} and the effects observed, there is insufficient knowledge on which to base a Canada Wide Standard for this contaminant. The US EPA has taken a similar view by revoking the PM_{10} standard due to a lack of evidence linking health problems to long-term exposure to coarse particle pollution. The WHO has established a 24-hour air quality guideline for PM_{10} µg/m^3 of 50 µg/m^3 based on typical relationships between $PM_{2.5}$ and PM_{10} concentrations in ambient air.

For the purpose of this assessment, the CWS of 27 µg/m^3 (24-hour average, 2020 standard) and 8.8 µg/m^3 (annual average) are used for assessing air quality impacts associated with $PM_{2.5}$. While it is acknowledged that there is considerable uncertainty in the quantitative relationship between exposure to PM10 and adverse health outcomes and the basis of the reference level developed by the Federal Provincial Advisory Committee Working Group on Air Quality Objectives and Guidelines, the value of 25 µg/m^3 has been used for assessing air quality impacts (24-hour average) associated with PM_{10}.

Nitrogen Dioxide

NO_x primarily consists of NO and NO_2 both of which are emitted into the atmosphere by various combustion sources (US EPA, 2008). NO_x also contains other oxides of nitrogenThe rate at which NO_x is converted to NO_2 in the atmosphere will largely depend on air dispersion which can vary seasonally (US EPA, 2008). For example, in the summer months, NO_x conversion to NO_2 may only take a few hours, whereas in the winter, it may take a full day (US EPA, 2008).

The US EPA (2008) extensively reviewed the available epidemiological, human clinical and animal toxicology data on exposure on NO_2. Some of the key findings in regards to health effects included the following:

- Short-term exposure to NO_2 has shown an increase in impairment of host defence systems in individuals which increases their risk shortly thereafter, of experiencing bacterial and viral infections.
- In human clinical tests, at NO_2 concentrations <2 ppm (3762 µg/m^3), airway inflammation has been shown to increase. In healthy individuals, the onset of airway inflammation has been observed at concentrations of 100-200 ppm/min (188,139 to 376,278 µg/m^3 per minute).
- Exposure to NO_2 has been shown to enhance the sensitivity of an individual to allergen-induced airway inflammation. Concentrations as low as 0.26 ppm (489 µg/m3) for 30 minutes have been shown to elicit an airway inflammation response. In healthy individuals, lung function has been observed as being altered after exposure to 1.5 - 2 ppm (2822 - 3762 µg/m^3) for 3 hours.
- Exposures to 34-37 ppb (63.9-69.6 µg/m^3) for 24-hour exposures have shown a clear association between respiratory symptoms and increased use of medication.

In Canada, the Canadian National Ambient Air Quality Objectives were developed in 1989 to evaluate air quality impacts associated with NO_2 emissions. The values derived were as follows: 400 µg/m^3; maximum acceptable level for 1-hour exposure; 200 µg/m^3; maximum acceptable level for 24-hour exposure; and, 60 µg/m^3; annual average level (Health Canada, 2006).

The Ontario Ministry of the Environment has adopted the 1-hour and 24-hour National Ambient Air Quality Objectives for use as health-based Ambient Air Quality Criteria in Ontario. These are used as benchmarks for assessing air quality and the potential for health effects in Ontario communities (MOE, 2011).

For the current assessment, the Canadian National Ambient Air Quality Objectives have been used for assessing potential health effects associated with exposure to NO_2. These are equivalent to the health-based Ambient Air Quality Criteria used by the MOE for assessing air quality in the province.

Carbon Monoxide

Carbon monoxide is a colorless, non-irritating, odourless and tasteless gas (ATSDR, 2009). The largest anthropogenic source of carbon monoxide to the environment is from vehicle traffic through the incomplete combustion of fuel. Once released to the atmosphere, carbon monoxide can remain in the air for approximately two months and it can undergo reactions with other compounds to form carbon dioxide (ATSDR, 2009).

Regulatory values for carbon monoxide in the atmosphere are largely based on cardio-respiratory effects seen in sensitive populations, in particular, people with exercise-induced myocardial ischemia (CEPA/FPAC, 1994). In addition, other respiratory effects include exacerbation of asthma, increased risk of heart failure, ischemic heart disease, myocardial infarction and stroke. Neurobehavioral and developmental effects are also seen to occur with increased exposure to carbon monoxide. These effects can be characterized by neurobehavioral and cognitive changes, and neurological impairment (ATSDR, 2009).

Exposure to carbon monoxide is typically characterized by measuring carbon monoxide levels in the blood as carboxyhemoglobin (COHb) (ATSDR, 2009). For example, exposure to 0.1 ppm (114.5 µg/m^3) CO is associated with a steady-state blood COHb of 0.25% and 5 ppm (5,725 µg/m^3) of CO is associated with 1% COHb (ATSDR, 2009). Cardio-respiratory effects have been observed at levels below 6% COHb, at approximately 40 ppm (45,807 µg/m^3 CO; CEPA/FPAC, 1994). Contrary, neurobehavioral and developmental effects have not been seen in COHb levels below 5%. However, in individuals with ischemic heart disease (susceptible sub-population), cardio-respiratory effects were noted where COHb levels measured as low as 2.9% (CEPA/FPAC, 1994).

Canadian air quality guidelines for carbon monoxide were derived to be protective of sensitive sub-populations, but also to provide for an additional margin of safety for the general population (CEPA/FPAC, 1994). Therefore, per the CNAAQO, a value of 15,000 µg/m^3 for 1-hour acute exposure, with a critical effect of a COHb less than 1% was derived. In addition, an 8-hour exposure period value of 6,000 µg/m^3 was derived and adopted for the 24-hour value.

The WHO (2000) has derived less conservative guideline values for carbon monoxide based on a COHb level of 2.5% (not to be exceeded) to protect non-smoking, middle-aged and elderly populations with documented or latent coronary artery disease from acute ischemic heart attacks and to protect foetuses of non-smoking pregnant women from hypoxic effects. The WHO recommends limits of 10,000 µg/m^3 for an 8-hour averaging period; 30,000 µg/m^3 for a 1-hour averaging period; and short-term values of 60,000 µg/m^3 and 100,000 µg/m^3 for 15 minute and 30 minute exposures respectively.

In the current assessment, the Canadian guideline values which represent the maximum desirable levels were selected to assess potential health risks from carbon monoxide. These are consistent with the health-based ambient air quality criteria used by the MOE when assessing air quality in Ontario communities.

Sulphur Dioxide

Sulphur dioxide is a colorless gas which has a pungent odour (ATSDR, 1998; Cal EPA, 2008). It has a high vapour pressure; therefore, it remains primarily in the gas phase in the atmosphere. Sulphur dioxide released to the environment occurs primarily from the burning of fossil fuels. Acute exposure to sulphur dioxide commonly leads to respiratory effects such as decreased lung function (e.g., increase in airway resistance, decrease in forced expiratory volume) and

constriction of the bronchia. The occurrence of these respiratory effects is more prevalent in asthmatics and other susceptible sub-populations with cardiovascular diseases (ATSDR, 1998; Cal EPA, 2008).

Epidemiological studies whereby individuals were exposed chronically to sulphur dioxide, health effects included mortality, morbidity and decreased lung function (WHO, 2005). However, no threshold for effects has been determined, and it is not clear whether sulphur dioxide is the primary pollutant responsible for causing the adverse effects, or if it is merely a surrogate for ultrafine particulates (WHO, 2005).

For the purpose of the current assessment, the CNAAQO (1989) for 1-hour acute exposure (450 µg/m^3), 24-hour exposure (150 µg/m^3) and annual exposure (30 µg/m^3) were selected to assess sulphur dioxide exposure. Values for all averaging times are maximum desirable levels.

Inorganics

Arsenic

Arsenic has both metal and non-metal properties capable of complexing with carbon and hydrogen or oxygen, chlorine and sulphur. While arsenic can be released to the environment through anthropogenic sources such as mining and smelting of lead and copper ores and coal-fired power plants and incinerators, the principal source of arsenic exposure to consumers is through dietary items such as seafood, poultry, rice and mushrooms. Daily intake is estimated to be on the order of 50 µg/day with only a minority of this (3.5 µg/day) being of inorganic arsenic (ATSDR, 2007).

In adults, the principal health effects from chronic oral and inhalation exposure to arsenic include skin effects and disease such as hyperpigmentation and keratosis; vascular disease; respiratory effects and bladder and lung and liver cancer. In children, health effects due to chronic exposure to arsenic include skin lesions, neurodevelopmental effects, lung disease, and reproductive effects including decreased birth weight, spontaneous abortion and neonatal death (Cal EPA, 2008).

IARC (1987) has listed arsenic and arsenic compounds as Class 1; carcinogenic to humans based on sufficient evidence of carcinogenicity in humans and limited evidence of carcinogenicity in animals. The US EPA IRIS (1998) has classified inorganic arsenic into Group A; a human carcinogen based on sufficient evidence from human data. Increased lung cancer mortality, and increased mortality from multiple internal organ cancers and increased incidence of skin cancer was observed in populations exposed to arsenic *via* inhalation or via drinking water. Cal EPA has listed arsenic and compounds as a chemical known to cause cancer (Cal EPA, 2010).

For the current assessment, the acute value of 0.2 µg/m^3 derived by Cal EPA (2008) was selected to assess risks resulting from the 24 hour exposure duration to arsenic. This value is based on decreased fetal weights, as seen in mice. The chronic inhalation TRV derived by Cal EPA (2008) of 0.015 µg/m^3 is based on a decrease in intellectual function and adverse effects on neurobehavioral development (Wasserman et al., 2004, and Tsai et al., 2003), and was selected to assess non-carcinogenic risks from inhalation. Cal EPA derived the TRV based on a LOAEL of 2.27 µg/L or 0.23 µg/m^3 for critical effects noted in children (n=201) of approximately 10 years old who were exposed to arsenic in drinking water for a period between 9.5 to 10.5 years. An uncertainty factor of 30 was applied to the LOAEL (3x use of a LOAEL; 10x intraspecies extrapolation) to derive the final TRV (Cal EPA, 2008). Carcinogenicity was assessed using the inhalation unit risk of 3.3E-03 (µg/m^3)$^{-1}$ derived by Cal EPA (2009) which was based on an occupational study and observations of lung cancer.

Calcium Oxide

Calcium oxide is a white hygroscopic, crystalline powder (ICSC, 1997). It is odourless and non-combustible. As per JECFA (1975), calcium oxide is classified as a food additive and has three functional uses as an additive including: use as an alkali, component of yeast food and dough conditioner. Exposure to calcium oxide may occur through inhalation and/or ingestion (ICSC, 1997). Exposure to high concentrations of calcium oxide via the dermal exposure pathway, may cause dry skin, redness, and burning sensation. Prolonged skin contact may cause dermatitis. Exposure to high concentrations of calcium oxide via ingestion may cause a burning sensation and abdominal pain, cramps, vomiting and diarrhoea. Eye contact with high concentrations of calcium oxide will cause redness, pain, blurred vision and possible severe deep burns. Inhalation exposure to calcium oxide may also cause a burning sensation, cough, shortness of breath and sore throat. However, prolonged exposure may cause a nasal ulceration (ICSC, 1997). At low level exposures, sensory irritation and decreased lung function has been noted as critical effects (SCOEL, 2008).

From the Cain et al. (2004) acute study, a threshold for sensory irritation of 1 mg/m^3 was observed. From the studies reviewed by SCOEL (European Scientific Committee on Occupational Exposure Limits, 2008; Toren et al., 1996), no additional respiratory effects could be noted at concentrations of 1 mg/m^3 as a result of long-term exposure (occupational study amongst kiln workers).

Therefore, to assess health effects resulting from the inhalation exposure of calcium oxide, the threshold of 1,000 µg/m^3 was utilized with an additional safety factor of 10 to address sensitive members of the public.

Chromium (II and III)

Chromium is a metallic element which exists in various oxidation states (MOE, 2009). The most common forms are metallic chromium (Cr(0)), divalent chromium (Cr(II)), trivalent chromium (Cr(III)) and hexavalent chromium (Cr(VI)). The divalent state of chromium is relatively unstable in the environment and readily oxidizes to the trivalent state. In general, due to the unstable

nature of divalent chromium, its toxicity is assumed to be similar to that of the trivalent form. Effects to the respiratory tract and depressed body weights have been noted as a result of exposure to Cr(III) via inhalation. Common anthropogenic sources of chromium to the environment include industrial sources such as combustion and ore processing (MOE, 2009).

Chromium (III) and metallic chromium compounds have been classified into Group 3- not classifiable as to their carcinogenicity in humans (IARC, 1990). The rationale for this classification is that there is inadequate evidence in humans for the carcinogenicity of metallic chromium and chromium (III) compounds; and there is inadequate evidence in experimental animals for the carcinogenicity of metallic chromium, barium chromate and chromium (III) compounds (IARC, 1990).

For the current assessment, the 24-hour average ambient air quality criterion of 0.5 µg/m^3 derived by the MOE (2009) was adopted to assess chronic exposure to chromium. This value is based on a rat inhalation study (Derelanko et al., 1999) whereby rats were exposed to chromium sulphate for a 13 week period (5 days/week, 6 hours/day). A human equivalence of 0.809 mg/m^3 for chromium sulphate was derived, adjusted specifically to account for chromium (III) which was equal to 0.138 mg/m^3. Application of an uncertainty factor of 300 (10x human variability; 3x extrapolation from animals to humans and 10x use of a subchronic study) equals a value of approximately 0.5 µg/m^3.

Copper Sulphate

Copper sulphate is readily soluble in water where it will dissociate into cupric ion and sulphate. While copper is an essential trace element, adverse health outcomes associated with ingesting relatively high concentrations are well documented. Excessive ingestion will lead to abdominal pain, headache, nausea, dizziness and vomiting with a number of well documented studies where accidental ingestion of copper present in cocktails, juices, teas and water have resulted in gastrointestinal symptoms (reviewed in ATSDR, 2004). Owing to the ability of copper to induce vomiting, soluble copper sulphate is used clinically as an emetic. The recommended dietary allowance for elemental copper in adults is 0.9 mg/day (Health Canada, 2010).

In terms of exposure via inhalation, copper can be acutely toxic when copper-containing dusts are inhaled or accidentally aspirated into the lungs. Acute inhalation exposure to copper dust can result in metal fume fever with mucous membrane irritation, inflammation, sweet metallic taste, dryness of the mouth and throat, chills, fever, and muscle aches. Reports of death by acute bronchopneumonia and pulmonary edema have been reported in subjects who have accidentally inhaled powders containing high concentrations (e.g. 70%) of copper (see Gosselin et al. Clinical Toxicology of Commercial Products, 5th Ed. Baltimore: Williams & Wilkins, 1984). Chronic exposure in an occupation setting has led to observations of “Vineyard Sprayers Lung” in workers exposed to copper sulphate sprays used to control mildew in vineyards. The condition manifests as the development of lesions, scarring, and nodules in the lung which are accompanied by weakness, loss of appetite, decreased body weight, shortness of breath, and in some cases cough. Deposition of copper in the lung and liver were observed on autopsy while sputum samples

identified the presence of macrophages containing copper granules. Similar pathology can be observed in animals exposed to copper sulphate containing dusts.

Toxicological reference values for copper are limited. The US EPA has not developed TRVs for elemental copper. ATSDR lists acute oral and sub-chronic oral minimal risk levels of 0.01 mg/kg/day based on gastrointestinal effects. On the basis that the available data on the toxicity of inhaled copper were considered inadequate, ATSDR has not developed minimal risk levels for inhalation. California's Office of Environmental Health Hazards Assessment (OEHHA) has established an acute reference exposure level (REL) for copper of 100 µg/m^3. The acute REL is based on the occupational exposure limit (TLV) of 1 mg/m^3 for copper dust developed by ACGIH. The TLV is based on an unpublished report that short duration exposure to 1-3 mg/m^3 copper fume resulted in a "sweet taste in the mouth" and that exposure to 0.02-0.4 mg/m^3 did not result in any symptoms. OEHHA treated 1 mg/m^3 as an NOAEL and applied an uncertainty factor of 10 to account for variability within the population. Owing to a lack of quantitative information on chronic exposure via inhalation, OEHHA has not developed chronic REL for copper (OEHHA, 2013).

For the current assessment, a value of 100 µg/m^3 based on OEHHA was used as the 1-hour averaging time. A value of 35 µg/m^3 was used for assessing exposure to copper sulphate through the 24-hour averaging time. This value was derived using the intermediate oral MRL of 0.01 mg/kg/day from ATSDR (2004) which is based on gastrointestinal effects seen in a population of males and females exposure to copper sulphate in drinking water for a 2 month period. Using a body weight of 70 kg and a breathing rate of 20 m^3/day, a value of 35 µg/m^3 was derived and adopted.

Hydrogen Cyanide

Hydrogen cyanide is a colorless or pale blue liquid or gas with a faint-bitter almond like odour (US EPA, 2010). There are various uses and sources of hydrogen cyanide to the environment. In particular, releases of hydrogen cyanide to the air include; from the syntheses of various chemicals including pharmaceuticals and chelating agents, through manufacturing activities including electroplating, metal mining, metallurgy, and metal cleaning processes, as well as via insecticides/fungicides and through cigarette smoke (Cal EPA, 2000; US EPA, 2010). Natural sources of hydrogen cyanide to the environment include via biomass burning, volcanoes and natural biogenic processes from higher plants, bacteria and fungi (US EPA, 2010). In non-urban areas, hydrogen cyanide has been found to range between 180 and 190 ng/m^3 which equates to an exposure of approximately 3.8 µg/day based on an inhalation rate of 20 m^3/day (CICADS, 2004).

Hydrogen cyanide can be rapidly absorbed via the oral, dermal and inhalation routes of exposure (US EPA, 2010). Once absorbed it is widely distributed and converted to the less acutely toxic compound thiocyanate. Excretion of exposed hydrogen cyanide occurs as thiocyanate in the urine. Chronic exposure to low concentrations of hydrogen cyanide has been seen to cause neurological, respiratory, cardiovascular and thyroid effects in occupational

studies. Due to inadequate information the carcinogenic potential of hydrogen cyanide cannot be assessed and therefore, hydrogen cyanide has not been classified as to its carcinogenicity (US EPA, 2010).

To assess the potential health effects resulting from the inhalation exposure for hydrogen cyanide the inhalation reference concentration Cal EPA (2000) of 9 µg/m^3 for chronic exposure was selected. This value is based on the study by El Ghawabi et al. (1975) whereby workers in three different electroplating factories were exposed to hydrogen cyanide for a period between 5 and 10 years. Critical effects included altered rates of iodide uptake by the thyroid, thyroid enlargement and CNS symptoms (headaches, weakness and sensory changes). From the primary study, a LOAEL of 6.4 ppm (7.07 mg/m^3) was extracted. Adjustment of the LOAEL to account for the difference between occupational exposure versus continuous ambient exposure led to an adjusted LOAEL of 2.5 mg/m^{3,} which was used as the point of departure. An uncertainty factor of 300 (10x extrapolation of a LOAEL to a NOAEL; 10x intraspecies variability; and, 3x extrapolation from subchronic to chronic exposure) was applied to the point of departure, and a reference concentration of 9 µg/m^3 was derived (Cal EPA, 2000). Recently, the US EPA (2010) derived a new inhalation reference concentration of 0.8 µg/m^3 for hydrogen cyanide using the same study, but with an additional safety factor of 10 for database deficiencies. Although this is a more recent value, this value was not adopted for use in the assessment as the MOE AAQC value incorporates the same study and level of uncertainty as the Cal EPA utilized for the derivation of the chronic REL. The need for an additional uncertainty factor to address database uncertainties was not considered warranted.

Magnesium

Magnesium is the 8th most abundant element on earth and the fourth most common mineral in the human body (Health Canada, 1987). In humans, magnesium is essential as it is needed in over 300 enzymatic reactions. In the human body, approximately 67% of total magnesium in the body (approximately 25 mg) is found in the bone. The remaining amount is found in soft tissues (30%) and fluids (1%). A deficiency in magnesium can contribute to adverse effects on the cardiovascular, neuromuscular and renal systems (Health Canada, 1987). Individuals who have impaired renal systems, and who consume high levels of magnesium may be at risk of developing hypermagnesemia (Michigian DEQ, 1994). However, individuals who are exposed to magnesium (i.e., magnesium oxide) via inhalation through fumes generated from combustion sources may be at risk of developing metal fume fever (Michigan DEQ, 1994).

In the current assessment, individuals may come into contact with magnesium via inhalation of dust. As magnesium is considered an essential element there is limited toxicological data and therefore, a limited availability of TRVs. However, occupational exposure values have been derived for magnesium, specifically magnesium oxide. ACGIH has derived a value of 10 mg/m^3 for exposure to magnesium oxide, which is protective of adverse health effects including: irritation of the eyes and nose and symptoms of metal fume fever (i.e., metallic taste in mouth, headache, fever, chills, aches, chest tightness and cough) (New Jersey Department of Health and Senior Services, 2007). Michigan DEQ (1994) derived an initial threshold screening level

(ITSL) of 100 µg/m^3 based on 1% of the ACGIH occupational value of 10 mg/m^3 which is based on an 8-hour averaging time and was adopted as the TRV (24-hr averaging time).

Manganese

Manganese is an essential trace element (IPCS, 1981). The largest source of manganese in humans is food-products which contribute to an average intake ranging from 10 to 50 µg/day. Anthropogenic sources of manganese to the environment include the production of manganese-oxide and dry-cell batteries, production and use of fertilizers and fungicides, mining operations and the manufacture of alloys, steel and iron products (IPCS, 1981).

In general, inhalation exposure to large quantities of manganese has been associated with neurological effects referred to as “manganism” (ATSDR, 2008). This condition begins with mild symptoms but evolves progressively to symptoms such as altered-gait, fine tremors, and possible psychiatric disturbances. Some of these symptoms also occur with Parkinson’s disease; therefore, manganism is also referred to as “Parkinsonism-like” disease or “manganese-induced Parkinson’s”. Inhalation exposure to large amounts of manganese have also been linked to reproductive effects in men such as decreased sperm count. Oral exposure to manganese has been observed to cause neurological, reproductive and developmental effects in humans; however, the evidence is limited in these studies (ATSDR, 2008).

IARC and the US EPA have concluded that there is insufficient data to assess the carcinogenicity of manganese and its compounds (IPCS, 1981; US EPA IRIS, 1996). Therefore, on this basis, IARC has not classified manganese and its compounds, and the US EPA has classified manganese into Group D; not classifiable as to human carcinogenicity.

For the purpose of this assessment, an inhalation TRV for a 24-hour exposure duration of 1.00E-01 µg/m^3 based on neurological effects (i.e., impairment in the eye-hand coordination) derived by MOE (2011) was selected to assess manganese oxide in the respirable fraction as defined by particulate matter less than 2.5 µm. This value is based on the occupational study by Roels et al. (1992) whereby individuals were exposed for an average period of 5.3 years. A point of departure based on a BMCL (5% increase in risk) of 84 µg/m^3 was derived by ATSDR (2008) which was adjusted from occupational to continuous exposure to equal a value of 30 µg/m^3. Application of an uncertainty factor of 300 (10x human variability; 3x database limitations; 3x vulnerability of the developing nervous system; and 3x extrapolation from subchronic to chronic exposure) resulted in a TRV of 1.00E-01 µg/m^3 (MOE, 2011).

Mercury

Mercury is present in the environment in different forms including metallic mercury (elemental mercury), inorganic mercury, and organic mercury (ATSDR, 1999). Metallic mercury is a shiny, silver-white liquid at room temperature. It is not combined with any other elements but may evaporate at room temperature with vapours that are colorless and odourless. Inorganic mercury is combined with other elements including chlorine, sulphur and oxygen and is

commonly referred to as a mercury salt. Environmentally relevant forms of mercury include: metallic mercury, mercuric sulphide, mercuric chloride and methyl mercury. Nevertheless, 80% of the mercury released to the environment is elemental mercury, released from sources such as fossil fuel combustion, mining and smelting and solid waste incineration. Although exposure to mercury may occur through inhalation, the dermal and oral pathways are significant exposure pathways depending on the circumstance (ATSDR, 1999).

The major target organs of metallic mercury intoxication are the kidneys (likely due to the high accumulation of mercury in the kidneys) and the central nervous system (CNS) for both inhalation and oral exposure (ATSDR, 1999). The CNS is the most sensitive target of mercury intoxication and the effects on the CNS and elicited responses will be the same for acute, sub-chronic and chronic exposures. In particular, decreased cognition and sensory perception will be observed as well as disturbances on the motor system. Signs of mercury intoxication include: tremors, irritability, nervousness, loss of confidence, insomnia, memory loss, neuromuscular changes, headaches, and polyneuropathy.

IARC (1993) has classified metallic mercury and inorganic mercury compounds into Group 3; not classifiable with respect to their carcinogenicity to humans. There is inadequate evidence in humans for the carcinogenicity of mercury and mercury compounds and limited evidence in experimental animals for the carcinogenicity of mercuric chloride. The US EPA concurred with the IARC and has classified elemental mercury as Group D indicating that it is not classifiable for human carcinogenicity (US EPA IRIS, 1995).

To assess the inhalation risks from inhalation of mercury, the chronic TRV of 0.03 µg/m^3 from Cal EPA (2008) for neurotoxicity as measured by intention tremor, memory and sleep disturbances, decreased performance on neurobehavioral tests (e.g., finger tapping, visual scan, visuomotor coordination and visual memory) and decreased EEG activity was selected. The TRV was derived using a time-adjusted point of departure of 9 µg/m^3 which is based on the LOAEL of 25 µg/m^3. The LOAEL is based on an occupational exposure duration of 8 hours per day (10 m^3/workday), 5 days per week.

Nickel

Elemental nickel is a hard, silvery-white metal that has no characteristic odour or taste (ATSDR, 2005; MOE, 2009). Anthropogenic sources of nickel include combustion (heavy residual and fuel oil), municipal incineration, cement manufacturing, nickel primary production operations and high temperature metallurgical operations. The source of nickel will influence the speciation of nickel that is released. Typically, nickel is found in the environment in combination with oxygen or sulphur as oxides or sulphides. Exposure to nickel may occur *via* all pathways including oral, dermal and inhalation (ATSDR, 2005, MOE, 2009).

The primary target of nickel for adverse effects in the human body is the respiratory system (MOE, 2009). Exposure to nickel will cause non-cancer effects in the respiratory tract, kidneys, immune system, endocrine system, and dermal system (EHC, 1991; MOE, 2009). Effects

include, but are not limited to: the development of asthma, Loffler's syndrome, pathological changes in the nasopharynx and decreased pulmonary residual capacity (EHC, 1991). Nickel can also act as a primary skin irritant and a skin sensitizer (EHC, 1991).

The US EPA did not evaluate soluble salts of nickel for potential human carcinogenicity (US EPA IRIS, 1996). Nickel refinery dust and specific nickel compounds (e.g., nickel carbonyl and nickel subsulphide) have been evaluated for carcinogenicity by US EPA IRIS, and IARC. US EPA concluded that nickel carbonyl is a probable human carcinogen (Group B2) due to sufficient experimental animal carcinogenicity data and inadequate human carcinogenicity data (US EPA IRIS, 1991). Nickel refinery dust and nickel subsulphide were categorized as human carcinogens (Group A) based on sufficient human carcinogenicity data (epidemiological data showing lung and nasal tumours) (US EPA IRIS, 1991). IARC (1990) has classified nickel compounds as human carcinogens (Class 1) and metallic nickel as possibly a human carcinogen (Class 2B).

The chronic inhalation TRV used was 0.04 µg/m^3 for nickel and nickel compounds in the total suspended particulate fraction based on the value derived by the Ontario MOE (2009). The potential for carcinogenicity *via* inhalation was assessed using an inhalation unit risk derived by the Ontario MOE (2009) of 5 x 10^{-5} (µg/m^3)$^{-1}$.

Lead

In the environment, lead is widespread, however, it is rarely ever found as a metal and usually found in the +2 valence state and in combination with two or more other elements (ATSDR, 2007). Lead is metal that is resistant to corrosion, has a low density, is ductile and has a low melting point. These characteristics make it a preferred metal for combining with other metals to form alloys that can be used in pipes, solder, weights, storage batteries, and shot/ammunition, etc. Lead can also be used in pigments in paint, dyes, ceramic glazes and in caulking. Individuals may be exposed to increased concentrations of lead in the environment that result from anthropogenic sources such as smelters, refineries and other industrial processes (ATSDR, 2007).

Gastrointestinal absorption of lead is dependent on various physiological and physicochemical properties including: nutritional status (i.e., fasting increases absorption; deficits in iron and calcium stores leads to increased absorption); age (i.e., absorption decreases in adults compared to children); particle size and solubility (Health Canada, 2011). However, once absorbed, the lead will rapidly circulate in the blood and distribute to the bone or to the soft tissues, or it will be excreted. Under steady-state conditions, 96 to 99% of lead is bound to protein and therefore, will not be able to cross into tissues or organisms. In general, up to 90% of the absorbed lead in adults is distributed to the bone, while 70% of lead in children is distributed to the bone and 8% to the soft tissues and less than 1% is circulated in the blood. There tends to be a continual exchange of lead that is present in the blood, soft tissue and bone. Under certain circumstances, lead in the bone can be mobilized and released back to the blood stream. The half life of lead in blood is approximately 30 days compared to 10 to 30 years

for lead in the bone. Excretion of lead occurs via the sweat, saliva, hair, nails and breast milk but to a greater extent it is excreted via the urine and feces. Increased exposure to lead may result in developmental neurotoxic, neurodegenerative, cardiovascular, renal and reproductive effects (Health Canada, 2011).

The evidence for the carcinogenicity of lead in humans is inconclusive, because of the limited number of studies, the use of small cohorts leading to a lack of statistical power and a lack of consideration of confounding variables. An association has been shown in animals between the ingestion of lead salts at high doses, and renal tumours. Based on the results of these studies, the US EPA has classified lead as B3 (possibly carcinogenic to humans) and considers there to be inadequate data in humans, and limited evidence in animals to develop estimates of the carcinogenic potency for ingestion of lead (US EPA IRIS, 1993). IARC (2006) has classified inorganic lead into Group 2A as probably carcinogenic to humans. This is based on limited evidence in humans for the carcinogenicity of inorganic lead compounds and sufficient evidence in experimental animals for the carcinogenicity of inorganic lead compounds (IARC, 2006).

To assess health impacts resulting from the inhalation of lead in air, values of 0.2 µg/m^3 and 0.5 µg/m^3 for 30 day and 24-hour averaging times were selected as derived by the MOE (2007). These values are based on the Cal EPA (2001) model which determines air lead concentrations associated with a 5% probability of children's blood lead level in a reference population exceeding a pre-determined blood lead level. The five parameters in the Cal EPA model include: geometric mean blood lead level for children (MOE value = 1.70 µg/dL); geometric standard deviation about the geometric mean blood lead level (MOE value = 1.6); lowest observed adverse effect level (MOE value = 5 µg/dL); air-to-blood slope factor (MOE value = 4.2 µg/dL per µg/m^3); and, probability for children exceeding the LOAEL (MOE value = 5%).

Titanium

Titanium is the ninth most abundant element on the earth's crust where the average concentration is approximately 4,400 mg/kg (WHO, 1982). The most common oxidation state for titanium is the +4 state and it is rarely ever found by itself in nature as it has a high affinity for oxygen and other elements. Anthropogenic sources of titanium to the environment include the combustion of fossil fuels and incineration of titanium containing wastes. In urban air, concentrations of titanium are generally less than 0.1 µg/m^3, however, in industrialized areas, concentrations can reach approximately 1 µg/m^3. Titanium is not an essential element. Generally, the principle source of exposure of titanium to the general population is through food. Intake via inhalation is generally less than 1% of the total intake. However, in occupational settings, where the presence of titanium is significantly greater, then inhalation is the greater source of exposure (WHO, 1982).

Titanium has not been ranked for its carcinogenicity, but titanium dioxide has been categorized into Group 2B as possibly carcinogenic to humans due to sufficient evidence of carcinogenicity in experimental animals and inadequate evidence of carcinogenicity in humans (IARC, 2010).

For the purpose of the current assessment, and in the absence of TRVs from other sources, a value of 34 µg/m^3 has been selected to assess exposure to titanium in air. This value is adopted from the MOE's health-based standard for titanium oxide.

Zinc

Zinc is an essential element in living organisms and comprises 0.004% of the earth's crust and is one of the most common elements present (ATSDR, 2005). In its elemental form, zinc is a bluish-white, shiny metal. Overall, taking into consideration all possible routes of exposure to zinc, the dietary intake route contributes 99% of the overall exposure (ATSDR, 2005). Zinc is an essential nutrient in the human body which allows for the functioning of various metalloenzymes. A deficiency in zinc may lead to the following health effects: dermatitis, anorexia, growth retardation, poor wound healing, hypogonadism with impaired reproductive capacity, impaired immune function, depressed metabolic function, and increased incidence of congenital malformations in infants (ATSDR, 2005). Excess levels of zinc may lead to decreased hematocrit, decrease in leukocyte number and function, decrease in high density lipoprotein and decreased iron stores. Exposure to zinc compounds such as zinc oxide via inhalation may lead to metal fume fever which can be characterized with impaired pulmonary function as well as nausea and leukocytosis (ATSDR, 2005).

US EPA (2005) has indicated that there is inadequate evidence to assess the carcinogenicity of zinc to humans. Therefore, it has not been classified as per the carcinogenic groupings. IARC has not classified zinc as per its carcinogenicity.

In the current assessment, a value of 18 µg/m^3 was selected to assess inhalation exposure (24-hours) to zinc. This value is based on a route-to-route extrapolation from an intermediate oral minimal risk level of 0.3 mg/kg/day derived by ATSDR (2005) using a standard breathing rate of 16.6 m^3/day and a body weight of 70.7 kg for an adult as per Health Canada (2011). The original derivation of the oral MRL is based on a study by Yadrick et al. (1989) whereby eighteen women ranging between the ages of 25 and 40 years old were given supplements of zinc gluconate twice daily (0.83 mg/kg/day) for a 10 week period. Results indicated a decrease in erythrocyte superoxide dismutase (ESOD) activity which is a sensitive indicator of copper status and a decrease in serum ferritin levels. The decrease in the ESOD activity was not considered a toxic effect but rather a precursor to more severe symptoms seen with zinc induced copper deficiency and the serum ferritin levels were still above the level that would represent a depletion of iron stores. Therefore, the dose of 0.83 mg/kg/day was considered to be a NOAEL. Using an uncertainty factor of 3 to account for human variability, an intermediate oral MRL of 0.3 mg/kg/day was derived (ATSDR, 2005).

2.4 RISK CHARACTERIZATION

The risk characterization of the risk assessment compares time-adjusted exposures to the appropriate toxicological reference values. For chemicals that operate via a threshold-type of dose response, the comparison most often used is termed a hazard quotient (HQ) or exposure

ratio (ER), which is simply the ratio between the estimated exposure divided by the TRV as shown in the following equation.

$$HQ\ or\ ER = \frac{Predicted\ Concentration\ or\ Dose}{TRV\ or\ Health\ based\ benchmark}$$

To account for potential exposure to the same chemical from multiple sources, the Ontario Ministry of the Environment, as well as several other regulatory agencies (e.g., Health Canada), use an HQ of 0.2 to define an acceptable level of exposure. This accounts for the potential for exposure via other sources (e.g., consumer products, drinking water, food, etc.). For criteria air contaminants where the only source of exposure is ambient air, and the point of comparison is an air quality guideline, the appropriate exposure ratio is 1.0.

For parameters where the mechanism of action has a threshold (i.e., No Observed Adverse Effect Level) and when predicted levels of exposure are less than the allowable limit (i.e., HQ is less than 0.2 or ER is less than 1.0), no adverse health outcomes would be expected. However, the converse is not automatically true. That is, when levels of exposure exceed the allowable exposure limit, adverse health outcomes are not necessarily expected. Rather, considering the uncertainties that are inherent in the assessment and the safety/uncertainty factors often employed, there is an erosion in the margin of safety between the estimated level of exposure that is known to cause adverse effects. Under such a situation, it is prudent to re-examine the basis of all the assumptions used to generate the estimates of risk and exposure.

For carcinogens that are assumed to operate via a non-threshold mechanism of action, the risk characterization identifies the incremental lifetime cancer risk (ILCR) associated with a particular exposure pathway per the following:

$$ILCR = Predicted\ Concentration\ or\ Dose \times Fraction\ of\ Lifetime\ Exposed \times Carcinogenic\ TRV$$

Incremental lifetime cancer risks are unitless values that express the probability of developing cancer for a specified level of exposure average over a lifetime (assumed to be approximately 80 years). Health Canada considers incremental lifetime cancer risks of one in a hundred thousand (10-5) or less as *de minimus*, which means they are below a level that would be of concern. The Ontario Ministry of the Environment consider incremental lifetime cancer risks of one in a million (10-6) or less as *de minimus*.

The characterization for risk at the maximum point of impingement is presented in Table 7 and for the nearest sensitive receptor in Table 8.

Table 7: Non-Carcinogenic and Carcinogenic Risks for Parameters Modelled at the Maximum Point of Impingement

Parameter	Modelled Concentrations at the Maximum Point of Impingement ($\mu g/m^3$)	Averaging Period (hr - unless noted otherwise)	Non-Carcinogenic TRV ($\mu g/m^3$)	ER or HQ	Inhalation Slope Factor $(\mu g/m^3)^{-1}$	ILCR
Criteria Air Contaminants						
PM_{10}	113	24-hr	25	4.52	—	—
$PM_{2.5}$	30.4	24-hr	27	1.13	—	—
	3.84	annual	8.8	0.44	—	—
Nitrogen Dioxide	304	1-hr	400	0.76	—	—
	101	24-hr	200	0.51	—	—
Carbon Monoxide	2640	1-hr	15000	0.18	—	—
	1680	8-hr	6000	0.28	—	—
Sulphur Dioxide	36.4	24-hr	150	0.24	—	—
	165	1-hr	450	0.37	—	—
	4.68	annual	30	0.16	—	—
Inorganics						
Arsenic	0.000849	24-hr	0.2	0.004	—	—
	0.000092	annual	0.015	0.01	0.0033	3.04E-07
Calcium Oxide	39.3	1-hr	100	0.4	—	—
	8.67	24-hr		0.01	—	—
Chromium (II & III)	0.0375	24-hr	0.5	0.08	—	—
Copper Sulphate	11.2	1-hr	100	0.11	—	—
	2.48	24-hr	35	0.07	—	—
Hydrogen Cyanide	7.61	24-hr	9	0.85	—	—
Mercury	0.0000197	24-hr	0.03	0.001	—	—
Magnesium	5.92	24-hr	100	0.06	—	—
Manganese (in $PM_{2.5}$)	0.0287	24-hr	0.1	0.29	—	—
Nickel (in TSP)	0.0112	24-hr	0.2	0.06	—	—
Nickel (in TSP)	0.00122	annual	0.04	0.03	0.00005	6.10E-08

Parameter	Modelled Concentrations at the Maximum Point of Impingement ($\mu g/m^3$)	Averaging Period (hr - unless noted otherwise)	Non-Carcinogenic TRV ($\mu g/m^3$)	ER or HQ	Inhalation Slope Factor $(\mu g/m^3)^{-1}$	ILCR
Lead	0.000258	30-day	0.2	0.001	—	—
	0.00118	24-hr	0.5	0.002	—	—
Titanium	1.06	24-hr	34	0.03	—	—
Zinc	0.0161	24-hr	18	0.0009	—	—

Table 8: Non-Carcinogenic and Carcinogenic Risks for Parameters Modelled at the Nearest Sensitive Receptor Location

Parameter	Maximum Modelled Concentration at the Nearest Sensitive Receptor ($\mu g/m^3$)	Averaging Period (hr - unless noted otherwise)	Non-Carcinogenic TRV ($\mu g/m^3$)	ER or HQ	Inhalation Slope Factor $(\mu g/m^3)^{-1}$	ILCR
Criteria Air Contaminants						
PM_{10}	23.8	24-hr	25	0.95	—	—
$PM_{2.5}$	11.4	24-hr	27	0.42	—	—
	0.755	annual	8.8	0.09	—	—
Nitrogen Dioxide	149	1-hr	400	0.37	—	—
	31.6	24-hr	200	0.16	—	—
Carbon Monoxide	914	1-hr	15000	0.06	—	—
	251	8-hr	6000	0.04	—	—
Sulphur Dioxide	7.82	24-hr	150	0.05	—	—
	80.7	1-hr	450	0.18	—	—
	0.51	annual	30	0.02	—	—
Inorganics						
Arsenic	0.000142	24-hr	0.2	0.001	—	—
Calcium Oxide	1.83	24-hr	100	0.02	—	—
Chromium (II & III)	0.00629	24-hr	0.5	0.01	—	—
Copper Sulphate	0.524	24-hr	10	0.05	—	—

Parameter	Maximum Modelled Concentration at the Nearest Sensitive Receptor ($\mu g/m^3$)	Averaging Period (hr - unless noted otherwise)	Non-Carcinogenic TRV ($\mu g/m^3$)	ER or HQ	Inhalation Slope Factor $(\mu g/m^3)^{-1}$	ILCR
Hydrogen Cyanide	2.23	24-hr	9	0.24	—	—
Mercury	0.00000331	24-hr	0.03	0.0001	—	—
Magnesium	0.992	24-hr	100	0.01	—	—
Manganese (in PM2.5)	0.0108	24-hr	0.1	0.11	—	—
Nickel (in TSP)	0.00189	24-hr	0.2	0.01	—	—
Nickel (in TSP)	0.000221	annual	0.04	0.01	5.0E-05	1.11E-08
Lead	0.000309	30-day	0.2	0.002	—	—
	0.000198	24-hr	34	5.8E-06	—	—
Titanium	0.177	24-hr	120	0.001	—	—
Zinc	0.0027	24-hr	18	0.0002	—	—

At the point of impingement, which represents the maximal theoretical exposure, maximum predicted concentrations of particulate matter (both PM_{10} and $PM_{2.5}$) have ERs greater than 1.0 (4.5 and 1.1 for PM_{10} and $PM_{2.5}$ respectively for 24-hour exposure periods). The ERs are based on concentrations predicted using the worst case emissions coupled with the worst case dispersion conditions using five years of historical meteorological data. In terms of understanding the potential for health risk, it is illustrative to examine the frequency with which the concentration of particulate is predicted to be greater than the risk-based threshold. As such, periods when concentrations of particulate result in an ER of greater than 1.0 are expected to be infrequent and transitory and not indicative of an unacceptable health risk.

For calcium oxide, a hazard quotient greater than 0.2 (HQ = 0.4) has been noted at the maximum point of impingement. HQs at the sensitive receptor location were less than 0.2. The increased HQ is a slight erosion in the margin of safety, however, adverse effects are not expected. Similar to the criteria air contaminants in which an exposure ratio of 1.0 is used to evaluate health risk rather than a HQ of 0.2, calcium oxide is a sensory irritant and not a systemic toxicant. In addition, adverse effects are not expected as an HQ greater than 0.2 is expected to be infrequent and transitory.

With respect to manganese, the maximum predicted concentration for manganese at the maximum point of impingement results in a hazard quotient greater than 0.2 (HQ of 0.3 for manganese) but is well below 0.2 at the nearest sensitive receptor location. As manganese is a

relatively abundant crustal element, its source is the result of fugitive emissions associated with material handling. Adverse health effects associated with exposure to this element is considered unlikely based on the conservative nature of the assumptions that have been made regarding dispersion conditions and exposure. In addition, it is important to note that concentrations at the sensitive receptor locations are well below the Ambient Air Quality Criterion that is permitted under Ontario Regulation 419/05 (see Appendix F).

Predicted hazard quotients for hydrogen cyanide based on maximum predicted concentrations are greater than 0.2 (HQ = 0.85) for the receptor located at the maximum point of impingement. As discussed above, acceptable health risks for non-carcinogens are based on a HQ of 0.2 on the assumption that exposure to a particular substance of concern can originate from multiple sources. In using a HQ of 0.2 to define acceptable risk, 80% of exposure is “reserved” for other potential exposure pathways. In the case of hydrogen cyanide, the principal source of exposure in a non-occupational setting is cigarette smoke, which can contribute 200 - 8,000 µg per day for an average smoker. In developing an ambient air standard for hydrogen cyanide for use under Ontario Regulation 419/05, the Ontario Ministry of the Environment used a hazard quotient of 1.0 to define an acceptable level of risk for hydrogen cyanide (MOE, 2005). Notwithstanding potential exposure via other sources such a cigarette smoke, considering the conservatism inherent in developing an acceptable ambient air standard for hydrogen cyanide, the MOE deemed a HQ of 1.0 suitably conservative for this compound. On a similar basis, considering the conservative nature of the dispersion modelling and the transient manner in which people may be exposed, adverse health effects associated with exposure to hydrogen cyanide are considered unlikely.

2.5 HUMAN HEALTH RISK ASSESSMENT UNCERTAINTIES

With any assessment that is reliant on making predictions on what may happen in the future, there are uncertainties that affect the confidence that can be placed on the assessment and the conclusions drawn. Understanding the uncertainty and its many sources helps to ensure that any management decisions that derive from the assessment are made with a full appreciation of the inherent uncertainties in the analysis.

This assessment, uncertainty derives from a number of sources that relate to understanding the potential for exposure as well as toxicity. To the extent possible, assessments are undertaken to obtain a realistic and accurate evaluation of risk based upon the available data. Where uncertainty exists, assumptions are typically made with an understanding that is preferable to err on the side of caution, thereby overestimating the degree of risk as opposed to underestimating the potential health risks.

Key factors that affect the uncertainty in this assessment, and their consequences are discussed below.

2.5.1 Toxicity Reference Values

Toxicity reference values were selected from a number of regulatory agencies after a careful review. TRVs are typically developed to be protective of sensitive individuals within a population (e.g., young children, the elderly and individuals with compromised health) and incorporate considerable conservatism when extrapolating from high doses, where overt effects can be observed, to low doses typical of environmental exposures.

While the majority of TRVs used in this assessment are conservative, those based on the National Ambient Air Quality Objectives and Canada Wide Standards may not be equally conservative as these represent recommended guidelines for ambient air in areas of the country with multiple sources of these pollutants. For pollutants such as $PM_{2.5}$, where adverse effects have been observed in cities with ambient concentrations less than Canada Wide Standard, the Canadian Council of Ministers of the Environment cautions that the CWS should not be interpreted as a threshold since there is minimal evidence to suggest a threshold exists below which no adverse effects would be expected.

2.5.2 Exposure Modelling

Exposure modelling was based on identifying the highest exposed receptor, using what would be considered worst-case emission characteristics coupled with worst case dispersion conditions, based on five years of meteorology. Although the use of conservative assumptions (i.e., maximum modelling scenarios used) over predicts ground level concentrations and exposure, they help identify the parameters that have the greatest contribution to risk.

2.6 HUMAN HEALTH RISK ASSESSMENT CONCLUSIONS

In conclusion, unacceptable health risks to human health receptors are not expected to occur as a result of the Project based on the following:

- Air dispersion modelling was completed to evaluate potential exposure at the maximum point of impingement and at the nearest sensitive receptor locations. Results of the modelling indicate exposure ratios greater than 1.0 for PM_{10}, $PM_{2.5}$ and hazard quotients greater than 0.2 for, calcium oxide, manganese and hydrogen cyanide at the maximum point of impingement. However, the periods when this may occur during the Project life will be infrequent and will be localized to the immediate vicinity of the Project site. At the nearest sensitive receptor locations, exposure rations and hazard quotients for all parameters were less than 1.0 and 0.2 respectively. Considering the inherent conservatism associated with the dispersion modelling used to develop exposure estimates and the toxicological reference values, adverse health outcomes associated with Project-related emissions are not anticipated.

- Project-related emissions that subsequently deposit to soil were predicted not to alter soil concentrations at the maximum point if impingement above values representative of background for Ontario soils. As such, indirect exposure of project related emissions that would result from consumption of local vegetation and/or game that consume such vegetation is not predicted to result in unacceptable health risk.
- Potential health risks associated with discharges to surface water was evaluated through an examination of changes to water quality in the receiving environment under different flow conditions. Resulting water quality, when compared to health-based benchmarks was not found to result in unacceptable health risks to users or consumers of such surface water. While it is understood that watercourse realignments will result in the flooding of former terrestrial lands, the areas to be inundated are already prone to flooding. Nevertheless there is the possibility that the decay of terrestrial vegetation will result in the production of methyl mercury that will be taken up by resident fish. The removal of vegetation prior to flooding will reduce the potential for methyl mercury production and will be undertaken prior to construction. As there are currently fish consumption advisories for mercury in lakes within the study area, it is considered unlikely that Project-related activities will have the potential to increase exposure to mercury for anglers in the area.

3.0 ECOLOGICAL HEALTH RISK ASSESSMENT

3.1 PROBLEM FORMULATION

Ecological Health Risk Assessments (EHRAs) are typically conducted using an iterative approach involving increasingly stringent tiers of evaluation. The level of detail of a risk assessment adopted for a particular situation should be equal to the degree and extent of potential effects to ecological receptors. Where evidence indicates that adverse effects may occur, a more detailed assessment may be required.

Conceptually, the EHRA consists of the following steps:

- Problem Formulation: The Problem Formulation step of the EHRA defines the issues at the Site as they relate to ecological receptors. In this step, parameters of potential concern are identified, and an ecological conceptual model (CM) is developed that describes basic assumptions regarding fate and transport of parameters of potential concern, ecological receptors and exposure pathways.
- Receptor Characterization: The Receptor Characterization is designed to characterize potential ecological receptors, identify potential exposure pathways by which ecological receptors may be exposed, and to determine the appropriate assessment and measurement endpoints.
- Exposure Assessment: In the Exposure Assessment, exposure pathways identified in the ecological CM are described, and chemical exposures are estimated by considering major exposure pathways.
- Hazard Assessment: Reference values for ecological receptors are determined based on a review of information provided by regulatory agencies, and the primary ecotoxicology literature, as necessary. Ecological effects that could potentially result from exposure to the parameters are also identified.
- Risk Characterization: In this step, potential ecological risks are determined by either a quantitative assessment (i.e., comparing the estimated rates of exposure from the Exposure Assessment to the acceptable exposure levels from the Hazard Assessment for each of the parameters of concern) or a qualitative assessment.

3.1.1 Study Objectives

The EHRA has followed the guidance established under the Federal Contaminated Sites Action Plan (Azimuth, 2012) and supplementary guidance provided by Environment Canada (i.e., a Framework for Ecological Risk Assessment: General Guidance; CCME, 1996, 1997).

The objectives of the current EHRA are to qualitatively and quantitatively evaluate the potential for adverse health effects to ecological receptors resulting from the Project activities.

3.1.2 Parameters of Potential Concern- Ecological Health

3.1.2.1 Air

Air emissions will be generated as a result of activities occurring from all phases of the Project. Some of the main sources of emissions will originate as a result of: blasting; material handling in the open pit; crushing; road traffic; managing mine rock, ore and overburden; and, exhaust from back-up power generation. A detailed assessment of the air emissions arising from the Project can be found in Appendix F. For each of those substances that were expected to be emitted in appreciable quantities, dispersion modelling was conducted providing predicted airborne concentrations at the maximum point of impingement (off the Property site) for different averaging times (1-hour, 24-hours and annual) considered sensitive. Modelling also includes deposition modelling to understand potential impacts to soil quality resulting from the deposition of contaminants of concern to soil.

Predicted maximum ground level concentrations for the substances expected to be emitted from the Project activities at the point of impingement are provided in Appendix F. These substances include criteria air contaminants as well as various inorganic chemicals. In addition, a number of sensitive receptor locations (i.e., cottages) were identified and maximum ground level concentrations for the substances expected to be emitted from the Project at these locations were modeled and are provided in Appendix F. Modeled parameters were compared to ambient air quality criteria. Table 9 provides a summary of the parameters and their expected concentrations at the maximum point of impingements.

Table 9: Predicted Air Emissions at the Maximum Point of Impingement

Parameter	Modelled Concentrations at the Maximum Point of Impingement (µg/m^3)	Averaging Period (hr - unless noted otherwise)
Particulate Matter (<10 µm) (PM_{10})	113	24-hr
Particulate Matter (<2.5 µm) ($PM_{2.5}$)	30.4	24-hr
	3.84	annual
Nitrogen Dioxide	304	1-hr
	101	24-hr
Carbon Monoxide	2640	1-hr
	1680	8-hr
Sulphur Dioxide	36.4	24-hr
	165	1-hr
	4.68	annual
Arsenic	0.000849	24-hr
	0.000092	annual
Calcium Oxide	39.3	1-hr
	8.67	24-hr
Chromium	0.0375	24-hr

Parameter	Modelled Concentrations at the Maximum Point of Impingement (µg/m^3)	Averaging Period (hr - unless noted otherwise)
Copper Sulphate	2.48	24-hr
Hydrogen Cyanide	7.61	24-hr
Mercury	0.00001975	24-hr
Magnesium	5.92	24-hr
Manganese (in $PM_{2.5}$)	0.0287	24-hr
Nickel (in Total Suspended Particulates)	0.0112	24-hr
Nickel (in Total Suspended Particulates)	0.00122	Annual
Lead	0.000258	30-day
	0.00118	24-hr
Titanium	1.06	24-hr
Zinc	0.0161	24-hr

3.1.2.2 Soil

Air emissions resulting from the Project and Project-related activities, may deposit as particulates to the soil. The presence of these substances in soil could then be available for uptake by ecological receptors via various pathways including:

- Soil contact
  - Soil nutrient and energy cycling (i.e., decomposition, respiration and organic matter cycles);
  - Dermal contact; and
  - Inhalation of re-entrained dust.
- Soil and food ingestion
  - Incidental ingestion of soil;
  - Herbivores (i.e., ingestion of vegetation grown in the soil); and
  - Secondary and tertiary consumers (i.e., consumption of organisms that are present in the area and that have consumed vegetation and/or organisms in the area).
- Soil to Ground Water (and eventually surface water) (i.e., migration of particulates in soil leaching to ground water and eventually discharging to surface water).

Therefore, to determine what the predicted concentrations of inorganics in soil are as a result of air deposition from the Project, depositional modelling was conducted at the maximum point of impingement.

To determine the concentration of these parameters in soil resulting from deposition, the following equation from US EPA (2005) was utilized:

$$C_{s=100 \times \frac{Dyd+Dyw}{Z_s \times BD} \times tD}$$

Where:

C_s = Predicted soil concentration over exposure duration (mg of the parameter of interest/kg soil);
100 = Units conversion factor (mg-m^2/kg-cm^2);
Dyd = Yearly dry deposition rate of pollutant (g/m^2-yr);
Dyw = Yearly wet deposition rate of pollutant (g/m^2-yr);
Z_s = Soil mixing zone depth (cm) (assume 1 cm mixing for direct ingestion of soil;
BD = Soil bulk density (g soil/cm^3 soil) (assume 1.5 g soil/cm^3); and,
tD = Time period over which deposition occurs (time period of combustion) (assume 15 yrs).

Predicted concentrations of inorganics in soil at the maximum point of impingement were compared to background soil concentrations (Table 10) obtained from the Ontario Ministry of the Environment. These background values are considered protective of ecological health exposure pathways.

Table 10: Predicted Soil Concentrations Resulting from Atmospheric Deposition- Maximum Point of Impingement

Parameter	MOE (2011) Background Soil Concentration (mg/kg)	Maximum Point of Impingement Deposition (g/m^2/year) (Dry & Wet Deposition)	Maximum Point of Impingement Soil Concentration Resulting from Deposition (mg/kg)	Maximum Point of Impingement Soil Concentration Above MOE (2011) Background Soil Concentration?
Arsenic	18	0.000164	0.16	No
Chromium	70	0.00726	7.26	No
Magnesium	15000	1.15	1146	No
Manganese	1400	0.0361	36.09	No
Mercury	0.27	0.00000382	0.0038	No
Nickel	82	0.00218	2.18	No
Lead	120	0.000229	0.23	No
Zinc	290	0.00311	3.11	No

3.1.2.3 Surface Water

Surface water quality modelling was conducted to predict changes that may occur as a result of the Project (see Appendix J). The maximum predicted concentrations of major ions, nutrients and metals occurring during each of the Project phases were compared to Aquatic Health Benchmarks. The Aquatic Health Benchmarks have been set to the most recent Provincial Water Quality Guideline (PWQG) or Canadian Water Quality Guideline (CWQG), or the British Columbia Ministry of the Environment (BCMOE) Guideline for parameters with no PWQO or CWQG. Where no guideline exists then the baseline concentration (upper 95th percentile) was used as the Aquatic Health Benchmark. See Table 11 for further details.

Table 11: Comparison of Maximum Predicted Surface Water Concentrations of Various Parameters to Aquatic Health Benchmarks

Parameter	95th Percentile Baseline Concentration (mg/L)	Aquatic Health Benchmark* (mg/L)	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Aluminum	0.1182	0.1182**	0.08	0.11	0.08	No
Ammonia (Total)	0.21	6.89	0.44	0.42	0.48	No
Ammonia (Unionized)	0.00049	0.019	0.002	0.002	0.003	No
Antimony	<0.006	0.02	0.001	0.001	0.002	No
Arsenic	<0.003	0.005	0.005	0.005	0.006	Yes
Barium	0.007	1	0.01	0.01	0.01	No
Boron	<0.01	1.5	0.01	0.01	0.01	No
Cadmium	0.00005	0.000058	0.00004	0.00005	0.00003	No
Calcium	10.465	10.465***	29.04	27.22	41	Yes
Chloride	1.2	120	1.63	1.99	2.1	No
Cobalt	0.00025	0.0025	0.0005	0.0006	0.0005	No
Copper	0.001	0.005	0.003	0.003	0.004	No
Cyanide (Free)	--	0.009784	0.005	0.008	0.005	No
Iron	0.369	0.369**	0.30	0.39	0.28	Yes
Lead	0.0005	0.001	0.0001	0.0001	0.0001	No
Magnesium	2.003	2.003***	2.40	3.42	2.42	Yes
Manganese	0.0878	0.7	0.11	0.14	0.10	No
Molybdenum	<0.002	0.073	0.003	0.003	0.004	No
Nickel	0.0015	0.025	0.003	0.004	0.003	No
Nitrate	0.13	13	1.36	0.26	1.7	No
Phosphorus (total)	0.035	0.035**	0.05	0.06	0.06	Yes
Potassium	0.49	373	1.75	1.8	2.5	No

Parameter	95th Percentile Baseline Concentration (mg/L)	Aquatic Health Benchmark* (mg/L)	Maximum Concentration (mg/L)- Average Conditions	Maximum Concentration (mg/L)- Dry Conditions	Maximum Concentration (mg/L)- Wet Conditions	Further Assessment?
Sodium	1.3365	1.3365***	2.51	3.79	2.60	Yes
Strontium	0.026	0.026***	0.05	0.05	0.07	Yes
Sulphate	4.092	218	7.1	10.7	7.34	No
Uranium	<0.002	0.015	0.004	0.004	0.01	No
Vanadium	<0.002	0.006	0.002	0.003	0.003	No
Zinc	0.032	0.032**	0.02	0.02	0.02	No

*The most recent Canadian Water Quality Guideline or Provincial Water Quality Objective for the protection of aquatic life was used. If there was no federal or provincial guideline, the most recent guideline from another Canadian jurisdiction (British Columbia Ministry of the Environment) was used.
** Upper limit of baseline is greater than the water quality guideline. Therefore, the Upper limit of baseline was adopted for use as the selected benchmark to evaluate aquatic health.
*** Aquatic health benchmark is the upper limit of baseline.

Note, the maximum concentrations do not take into account the data at the point of discharge as potential effects related to these concentrations have been discussed in Appendix J.

3.1.3 Ecological Conceptual Model

The local study area as defined in Appendix F is inhabited by various terrestrial ecological receptors including soil invertebrates (e.g., earthworms), terrestrial plants (e.g., trees such as balsam fir, red maple, black ash, etc., small trees, shrubs and woody vines such as bunchberry, Labrador tea, choke cherry and sweet blueberry, ferns and allies, graminoids, forbs, mosses and lichens), mammals (e.g., beaver, black bear, fisher, moose, red fox, white-tailed deer, etc.) and birds (e.g., American bittern, American robin, barred owl, blue-wing teal, Canada goose, etc.), detailed in Chapter 6, Description of the Environment and Appendices K to M. With respect to aquatic receptors, the water bodies at, and around the Project site are inhabited with aquatic vegetation (submergent and emergent), benthic communities and higher trophic level receptors including a variety of fish such as: blacknose shiner, spottail shiner and the Iowa darter which are small-bodied fish and larger sport fish including: northern pike, yellow perch, walleye, whitefish and smallmouth bass (see Chapter 6 and Appendix N).

Ecological receptors may come into contact with emissions and discharges originating from the Project site through various pathways including the following:

- direct inhalation of airborne emissions;
- deposition to soil with subsequent direct contact (e.g., dermal contact, incidental ingestion and inhalation of re-entrained dust);
- direct ingestion of surface water;
- incidental ingestion and dermal contact of surface water;
- consumption of fish, soil/aquatic invertebrates and mammals; and
- consumption of plants (e.g., berries, below-ground and above-ground plants and aquatic plants).

3.1.4 Receptor Characterization

A receptor is defined as an organism or group of organisms that have the potential to be affected by a chemical or other stressors. Receptors selected for assessment typically represent ecological receptors, resources or environmental features that have economic and/or social value, or intrinsic ecological significance. The relevant ecological receptors have local, regional, provincial, national, and/or international profiles, and serve as a baseline from which the impacts of the Project activities can be evaluated, including changes in management or regulatory policies.

3.1.5 Terrestrial Receptors

3.1.5.1 Terrestrial Plants

Consistent with CCME (1996; 1997) guidance, plants are typically assessed as a group rather than as separate species. Plants are potentially exposed to parameters of concern in soil via root uptake and, in some cases, foliar uptake from aerial deposition. As no parameters of concern have been identified in soil, then root uptake is considered an incomplete pathway.

With respect to foliar uptake from aerial deposition, this is not expected to be a significant pathway on the basis of wash-off due to precipitation.

3.1.5.2 Soil Invertebrates

In terms of sensitivity to toxic parameters, earthworms are considered to be one of the most sensitive receptors for parameters in soil. Earthworms are in near-constant direct dermal contact with soil and are important in ensuring soil fertility. Their feeding and burrowing activities breakdown organic matter and release nutrients and improve aeration, drainage and aggregation of soil. Earthworms are also important components of the diets of many birds and mammals.

3.1.5.3 Birds

The local and regional study areas, as defined in Appendix F, provide suitable habitat for a number of avian species including: owls, raptors, waterfowl, upland game birds, songbirds and woodpeckers.

3.1.5.4 Mammals

Within the local and regional study areas there are several mammals that have been observed, or have the possibility to occur due to appropriate habitat conditions. Some of the smaller mammals include: deer mouse; woodland/meadow jumping mouse; meadow/rock vole; southern bog lemming; red squirrel, etc. Larger mammals include: beaver, black bear, gray wolf, red fox, snowshoe hare, etc. Lastly, with respect to ungulates, moose have been observed and there is appropriate habitat for white-tailed deer.

3.1.6 Aquatic Receptors

3.1.6.1 Aquatic Plants

Aquatic plants are an important component of freshwater ecological systems. Aquatic plants take a variety of forms, including submerged, emergent, and free-floating forms. Aquatic plants, including algae, oxygenate water and form the basis of the aquatic food chain. Similar to terrestrial plants, aquatic plants are typically evaluated as a group rather than as individual species.

3.1.6.2 Aquatic Invertebrates

Aquatic invertebrates are an important group of organisms in most freshwater systems. Aquatic invertebrates, as a group, play a critical role in the ecology of aquatic systems, as primary consumers, detritivores, and as prey for higher trophic level organisms. Additionally, aquatic invertebrates as a group tend to be one of the most sensitive to environmental contaminants, so protection of invertebrates also tends to result in protection of other species. Invertebrates are often used as “indicators” of environmental degradation, because of their rapid and predictable response to various environmental contaminants and other stressors.

3.1.6.3 Fish

Fish can be exposed to contaminants in surface water and sediment, but regardless of the source, uptake across the gills occurs via the aqueous pathway. Therefore, for the purposes of this assessment it was assumed that fish are exposed primarily via uptake of aqueous constituents across the gills. It is important to note that unlike some other receptors, fish are mobile and capable of avoiding contaminants; fish in an unconfined water body can ameliorate their exposure to contaminants in surface water by moving to another location.

3.2 EXPOSURE ASSESSMENT

The exposure assessment includes an analysis of the pathways through which receptors may be exposed to parameters of potential concern and an estimate of the concentrations to which they may be exposed. For parameters of concern to have deleterious effects on ecological receptors, they must gain access to the organism or receptor. The route by which this occurs is referred to as an exposure pathway and is dependent on the nature of the chemical and the nature of the receptor. A complete exposure pathway is one that meets the following criteria:

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

3.2.1 Pathway Analysis

Ecological receptors may come into contact with emissions and discharges originating from the Project site through various pathways including the following:

- direct inhalation of airborne emissions;
- deposition to soil with subsequent direct contact (e.g., dermal contact, incidental ingestion and inhalation of re-entrained dust) and root uptake;
- direct ingestion of surface water;
- incidental ingestion and dermal contact of surface water;
- consumption of fish, soil/aquatic invertebrates and mammals; and
- consumption of plants (e.g., berries, below-ground and above-ground plants and aquatic plants).

3.2.1.1 Inhalation Exposure to Ambient Air

Ecological receptors could come into contact with airborne emissions via inhalation or through deposition to foliage and subsequent uptake. However, exposure via these pathways is thought

to be minimal as a comparison of predicted airborne emissions to ambient air standards, which are protective of both human and ecological receptors based on direct inhalation, indicating that only particulate matter (<10 µm and <2.5 µm) had predicted airborne concentrations higher than their respective AAQC. Note, the AAQCs are a conservative screening tool for ecological receptors as the AAQCs for particulate matter are focused on protecting small decrements in human health (i.e., lung function) which would not affect an ecological receptor on a population level. Particulate matter typically consists of a variety of components including elemental and organic carbon, nitrates, ammonia, sulphates and trace metals. US EPA (2003) indicates that these fractions are not typically considered respirable; rather, predominant exposure is typically through the ingestion of dust which is accounted for through the soil ingestion pathways. As there are no expected appreciable changes to background soil quality resulting from deposition from the Project, exposure to parameters of potential concern via soil ingestion within the study area is not expected. As such, this exposure pathway is not considered further in the EHRA.

3.2.1.2 Direct and Indirect Exposure to Soil

Airborne emissions resulting from the Project and Project-related activities have the potential to deposit to soil and affect the soil quality and the health of any organisms that inhabit the soil or consume plants/soil organisms and/or mammals that reside in the potentially impacted area. Specifically, terrestrial receptors may be exposed to parameters of potential concern in soil through:

- root uptake of parameters of potential concern from soil and/or root contact by terrestrial plants;
- direct contact with parameters of potential concern in soil by terrestrial plants, invertebrates, small mammals and birds; and
- incidental ingestion of vegetation and prey items that have accumulated parameters of potential concern from the soil by mammals and birds.

To understand potential risks associated with this exposure pathway, depositional modelling was undertaken to provide a maximum deposition rate of different parameters of concern. Assuming incidental mixing within the first 1 cm of soil, this was used to develop an understanding of the incremental change in background soil quality over the 15-year operational phase of the facility. As there was no incremental change to background soil quality resulting from deposition, it was concluded that exposure via this pathway would not result in “unacceptable” risk to ecological receptors, and therefore it was not considered further.

3.2.1.3 Direct and Indirect Exposure to Surface Water

Terrestrial receptors may be exposed to parameters of potential concern in surface water through ingestion via drinking water and through direct contact. Aquatic receptors may be exposed to parameters of concern in surface water through the ingestion of aquatic vegetation and prey items (see Appendix N, Aquatic Biology TSD, for further details).

3.3 HAZARD/TOXICITY ASSESSMENT

3.3.1 Aquatic Receptors- Toxicological Reference Values

For parameters that exceeded the aquatic benchmarks, toxicological reference values using the most sensitive, relevant aquatic species were developed (TRVs) (see Table 11) which were used to characterize risks to aquatic species.

Table 12: Toxicological Reference Values for Aquatic Receptors

Parameter	TRV (mg/L)	Species Endpoint	Endpoint Type	Rationale	Reference
Arsenic	0.05	Algae (*Scenedesmus obliquus*)	Effect concentration for 14-day growth test	This value represents the 14-day EC50 (growth) to the most sensitive organism to arsenic, the algae *S. obliquus* and thus will be of fish and aquatic life.	Canadian Environmental Quality Guidelines, CCME (1999) updated 2001- Arsenic Fact Sheet
Calcium	423.9	Aquatic invertebrates (*Daphnia magna*)	Lowest observed effect concentration (reproduction 21-day)	Not specified	Baillieul et al. 1993
Iron	1	Water quality guideline used	Not specified	A recent review by BCMOE of iron toxicity and guidelines recommends a guideline for the protection of aquatic life of 1.0 mg/L	Ambient Water Quality Guidelines for Iron, BCMOE, 2008
Magnesium	82	Aquatic invertebrates (*Daphnia magna*)	Lowest chronic value (EC16-reproduction)	Not specified	Biesinger and Christensen, 1972 as cited in Suter II and Tsao, 1996
Sodium	180	fish	Not specified	Lowest reported toxicity value for aquatic life	Mount et al., 1997 as cited in MOE, 2011
Strontium	15 (acute)	Various datasets reviewed	Tier II secondary acute	US EPA Ecotox database value for acute toxicity- short term exposure used to assess maximum values.	Suter II and Tsao, 1996
	1.5 (chronic)	Various datasets reviewed	Tier II secondary chronic	US EPA Ecotox database value for chronic toxicity- long term exposure used to assess median values.	

3.4 RISK CHARACTERIZATION

Characterization of risk to ecological receptors in an EHRA can employ either, or both qualitative or quantitative methods. The Hazard Quotient (HQ) is a simple approach that provides a quantitative estimate of overall risk. The HQ is a unit less value defined as the ratio of the magnitude of exposure to magnitude of a standard effect:

$$Hazard\ Quotient = \frac{Exposure\ Estimate}{TRV}$$

Hazard quotient ratios are interpreted as follows: if the HQ is less than one, no unacceptable risks to ecological receptors would be expected, because concentrations are below levels known to cause adverse effects. Conversely, if the HQ exceeds one, it may be inferred that adverse effects to individuals are possible. Given a certain magnitude and type of effect associated with a particular TRV or assessment endpoint, inferences about potential effects can be made. For example, if the level of exposure exceeds a TRV based on a 25% reduction in a growth-based endpoint (HQ >1), it can be inferred that one possible outcome may be diminished growth of individuals, potentially (but not necessarily) leading to a reduction in population abundance of that receptor. It is important to note that exceeding an HQ of one, does not necessarily mean adverse effects will occur; rather, it suggests that we have less confidence that adverse effects will not occur. For a variety of reasons, adverse effects demonstrated in laboratory studies often fail to manifest in the field as a measurable or meaningful impact. It is also important to recognize that the magnitudes of HQs are not directly associated with the magnitude of potential effects. That is, a large HQ (>10) should not be interpreted as a 10-fold greater risk than an HQ of one.

For those parameters of potential concern with HQs greater than one, potential risks at a population level cannot be ruled out. Evidence from sources other than chemical analysis may be employed to evaluate the potential risks further, including evidence of toxicity at the Site, toxicity of media in laboratory exposures (i.e., bioassays), the absence of species formerly present or commonly found at similar Sites, or diminished populations compared to a reference location.

3.4.1 Aquatic Receptors

As the maximum predicted concentrations of arsenic, calcium, magnesium, sodium, strontium, and iron exceeded their respective aquatic health benchmarks, concentrations of parameters were compared to risk-based aquatic toxicity reference values (see Table 11- also refer to Appendix N).

With respect to phosphorus, although the predicted concentrations yielded hazard quotients greater than 1 when compared to aquatic benchmarks, adverse effects resulting from potential increased phosphorus in surface water are not expected as the model incorporates baseline data that was elevated as a result of the analytical process.

Table 13: Predicted Hazard Quotients for Aquatic Receptors under Various Modelled Scenarios Using Risk Based Aquatic Toxicity Reference Values

Parameter	TRV (mg/L)	Average Conditions		Dry Conditions		Wet Conditions	
		Maximum Concentration (mg/L)	HQ	Maximum Concentration (mg/L)	HQ	Maximum Concentration (mg/L)	HQ
Arsenic	0.05	0.005	0.1	0.005	0.1	0.006	0.12
Calcium	423.9	29.04	0.07	27.22	0.06	34.80	0.08
Iron	1	0.30	0.3	0.39	0.39	0.28	0.28
Magnesium	82	2.40	0.03	3.42	0.04	2.42	0.03
Sodium	180	2.51	0.01	3.79	0.02	2.60	0.01
Strontium	15 (acute) 1.5 (chronic)	0.05	0.003 (acute) 0.03 (chronic)	0.05	0.003 (acute) 0.03 (chronic)	0.06	0.004 (acute) 0.04 (chronic)

3.4.2 Terrestrial Receptors (Exposure to Surface Water via Drinking Water)

Human Health Benchmarks were used to assess potential risk to terrestrial receptors associated with exposure to surface water as these are considered protective of all exposure pathways relevant to surface water including direct ingestion, dermal contact during swimming and indirect ingestion of fish. As the concentration of all of the parameters of concern attributable to the Project in surface water are predicted to be below Human Health Based Benchmarks (see Table 5), it can be concluded that there are no unacceptable health risks associated with discharge to surface water to terrestrial receptors. However, no benchmarks were available for the following: calcium, iron, magnesium, phosphorus and sodium. As these are essential elements, they are not considered to pose adverse health effects to terrestrial receptors. While no ecological benchmarks were available for strontium, it was evaluated in the HHRA and was not found to pose a risk to human health based on direct ingestion as a source of drinking water. As the drinking water pathway for human health is considered to be more sensitive than the drinking water pathway for terrestrial organisms, strontium is not expected to pose a risk to terrestrial receptors.

3.5 ECOLOGICAL HEALTH RISK ASSESSMENT UNCERTAINTIES

Uncertainty in risk assessment is introduced by the necessary use of assumptions concerning various aspects or characteristics of the system that cannot be measured accurately. Incomplete understanding of environmental processes is inherent in any EHRA. Uncertainty is acknowledged, documented, and addressed primarily by the use of conservative assumptions which ensure risk is overestimated rather than underestimated. Various sources of uncertainty associated with the current EHRA are discussed below:

- ***Exposure*** – Uncertainty in the exposure assessment was related primarily to assumptions regarding the presence of ecological receptors at the Site and the concentrations to which they are exposed.
- ***Toxicity Assessment*** – Because of the inherent uncertainty in predicting toxicological responses from literature studies rather than directly measuring toxicity at the Site, there is some uncertainty associated with toxicity reference values. In most cases, TRVs are assumed to be conservative; i.e., no toxicity is anticipated if Site concentrations are below TRVs. This is because most reference values are based on the most sensitive species tested or a similar low effect level (e.g., 10^{th} or 25^{th} percentile of species sensitivity distribution), and toxicity tests upon which they are based are typically conducted under conditions that maximize toxicity (i.e., the use of soluble metal salts).
- ***Risk Characterization*** – For the most part, the Hazard Quotients generated in the risk characterization phase of the EHRA should be considered to be quite conservative; i.e., HQs greater than one do not necessarily mean toxicity is occurring at the Site. Generally, predicted risks are for individual organisms. For the current Project, no toxicity studies have been performed. In many cases, toxicity at a Site is considerably diminished compared to effects predicted from laboratory studies, for a variety of reasons. Generally, in laboratory tests, animals are exposed to higher doses and test animals are observed for overt signs of adverse health effects which would not typically be seen in the environment.

3.6 ECOLOGICAL HEALTH RISK ASSESSMENT CONCLUSIONS

In conclusion, unacceptable health risks to ecological receptors are not expected to occur as a result of the Project based on the following:

- Ecological receptors could come into contact with airborne emissions via inhalation or through deposition to foliage and subsequent uptake. However, exposure via these pathways is minimal. A comparison of predicted airborne emissions to ambient air standards indicates that only particulate matter (<10 µm and <2.5 µm) had predicted airborne concentrations higher than their respective AAQC. However, the AAQCs are a conservative screening tool for ecological receptors as the AAQCs for particulate matter are focused on protecting small decrements in human health (i.e., lung function) which would not affect an ecological receptor on a population level.
- Project-related emissions that deposit to soil are predicted to not alter soil concentrations at the maximum point of impingement above background values for Ontario soils. As such, exposure to ecological receptors via direct and indirect exposure to soil is not expected to result in adverse effects.
- Lastly, maximum predicted surface water quality results were below aquatic health benchmarks except for arsenic, calcium, iron, magnesium, phosphorus (total), sodium and strontium. However, risks to aquatic receptors as a result of exposure to these parameters is not expected as comparison to toxicity based reference values resulted in exposure ratios of less than one indicating that unacceptable risks are unlikely to occur.

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