Coffee Gold Mine
YESAB Project Proposal
Appendix 18-B Human Health Risk Assessment
Technical Report

VOLUME IV

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EXECUTIVE SUMMARY

The proposed Coffee Gold Mine (Project) is a proposed gold development project in west-central Yukon, approximately 130 kilometres (km) south of the town of the City of Dawson (Dawson). The Project, as proposed, includes an 18-month construction period, followed by a 12-year mine life, followed by closure. The Project is located on Crown Land within the traditional territory of Tr'ondëk Hwëch'in (TH) and the asserted area of White River First Nation. It is important to consider how the Project, through construction, operations, closure and post-closure could influence the health and well-being of people who use the land and frequent areas in the vicinity of the proposed Mine Site or Northern Access Route (NAR). This Human Health Risk Assessment (HHRA) examines how changes in environmental quality (e.g., air quality, environmental noise, water quality, soil quality) could influence human health.

The HHRA considers three major aspects of possible changes in environmental quality as a result of the project:

(i) increased noise during construction and operation
(ii) air emissions and dust generation / dust fall during the peak operational period (which will create greater potential for changes in air quality than the other project phases), and
(iii) changes in the concentrations of deposited contaminants in soils, plants, and their consumers especially in the context of gathering and consuming traditional and non-traditional food substances from reclaimed waste rock deposits during the post closure period at the mine or areas around the mine that might be affected by dust fall.

Various aspects of Project-related changes in the physical and biophysical environment could interact with community health and wellbeing. Given the historical and ongoing importance to First Nations and Yukoners in general of lands, water, and resources in the Project area and along the Northern Access Route (NAR), Project-related changes in surface geology and soils, groundwater, surface hydrology, water quality, air quality, and noise merit close evaluation with regard to community health and well-being.

Human exposures to noise can result in increased stress and annoyance, sleep disturbance, or a decreased ability to communicate, focus on important tasks, and learn, depending on the specific circumstances associated with noise exposures. Project-related changes in the environmental quality of groundwater and surface water, sediment and soil, or air quality could affect humans that interact with the local environment through direct exposure pathways, including the ingestion of water, incidental soil and sediment ingestion (e.g., as occurs when people get soil on their hands and then place them in their mouths when eating), inhalation, and uptake across the skin (dermal uptake). Project-related changes in the environmental quality of the physical environment can also increase contaminant exposures in people through indirect pathways, to the extent that contaminants can be taken up into edible food substances such as plants or edible fish and wildlife. Such indirect human exposure pathways are an important part of this HHRA in light of the importance of local plants and animal resources to both aboriginal and non-aboriginal people in the region.

The spatial scope of this HHRA includes any area where Project-related changes in noise, air quality or dust fall could be distinguished from background conditions. The scope also encompasses any people who may harvest and / or consume country foods from areas that could potentially be influenced by mine activities. The spatial boundaries for the HHRA are largely aligned with the spatial boundaries for the noise assessment (**Section 10.0**) and air quality assessment (**Section 9.0**) since these are the main drivers of interest with regard to Project-related changes in environmental quality which, if adversely affected, can in turn affect human health.

Adverse influences of the proposed Project on human health are of interest during the construction phase, operations phase, through decommissioning and closure, and post closure. For each of the three major HHRA components (noise health effects, air quality health effects, country food safety), health risks are expected to be different for different Project phases. For noise and air quality health effects, sources of noise and air emissions are expected during construction, operation, and decommission / closure. Dust generation and dust fall in the post closure period are expected to be minimal, and any contaminant exposures from the breathing in of suspended particulates are likely to be very small in comparison to the construction, operation or decommissioning phases. No mining related sources of noise or combustion emissions are expected in the post closure phase, so noise and air quality are not assessed in the HHRA for the post closure conditions due to a lack of any viable exposure pathways. Contaminant exposures associated with the gathering and ingestion of country foods were assessed for the post-closure period, since the changes in soil quality either at the mine site based on waste rock deposition or in the local assessment area in general based on mining-related dust fall will be cumulative through construction, operation, and decommissioning, with the extent of contamination, if any, theoretically greatest at the end of the closure phase.

E-1 NOISE HRRA

The primary objective of noise HHRA was to evaluate whether noise arising from Project activities could have an adverse effect on human health. Modelled predictions of noise levels were completed for both Project construction and operation. The noise HHRA was based especially on noise predictions as presented in **Section 10.0** of the Project Proposal, and in particular on continuous noise metrics such as $L_{EQ,}$ over the daytime period, night-time period, or over a 24-hour day-night period, for those aspects of construction and mining that are expected to result in ongoing noise generation over extended durations, such as ore crushing, haul truck operations, or diesel power generation. Outdoor noise levels were predicted as part of the noise assessment for areas within or near the mine camp, and fifteen focal areas of interest beyond the Mine Site, such as the adjacent Yukon River and camping sites used by outfitters and others, the mouth of Coffee Creek, confluence of Coffee and Latte Creeks, and the upper slopes of mountains immediately across the river from the proposed Mine Site.

Predicted outdoor noise levels during peak activity periods were evaluated based firstly on whether the magnitude of change relative to the current existing wilderness-type noise levels is expected to be greater than three decibels as A-weighted sound (dBA). Predicted outdoor noise levels were further compared to a threshold of health effects based on an increase by 6 percentage points in the percent of people experiencing that noise level who feel highly annoyed (and stressed). Additional health effects thresholds used in the HHRA were associated with potential for sleep disturbance (outdoor night time noise greater than 40 to 55 dBA), and the potential for speech interference, youth learning, or ability to focus on various tasks (daytime outdoor noise greater than 55 dBA).

For all areas adjacent to the proposed Mine Site (e.g. as represented by focal areas of interest), noise levels associated with construction or operational activities are expected to be lower than the existing measured background noise levels and thus will not be easily distinguishable from the background wilderness-type noise environment. In addition, noise at all modelled locations will be far lower than authoritative health effects thresholds relating to sleep disturbance or speech interference. The highest predicted continuous sound level offsite was 28 dBA during peak operations at a location near the centre of the Yukon River, approximately 10 km downriver from the mouth of Coffee Creek. It is conceivable that people will be able to faintly detect noise from mine operations when the ambient noise levels are very low (infrequent periods when background sound levels are less than 25 dBA; for example, during low wind conditions and in the absence of wildlife or personal sounds), especially given the different frequency and tonal characteristics than natural sounds. Nonetheless, the predicted Project-related noise levels are far lower than levels associated with high annoyance (and stress), or with sleep disturbance or speech interference.

Along the mine access road, daytime vehicle transits will generally not exceed eight vehicles per day, and no nighttime traffic is proposed. The resulting infrequent and transient noise levels adjacent to the access road will be sufficiently low that no human health effects are predicted.

Overall, the Project is not predicted to result in any noise-related human health risks.

E-2 AIR QUALITY HRRA

The primary objective of air quality HHRA was to evaluate whether contaminants associated with air emissions from Project activities could have an effect on human health. A detailed evaluation of emissions sources based on the Project Description for each of construction and operations phases indicates that the potential for both dust generation / dust fall and emissions from internal combustion engines (or the incinerator) is far greater for the peak operational year than construction year. The HHRA, therefore, is based on air quality predictions (**Section 9.0**), based on source emissions and dispersion modelling for the peak operational year (Year 6).

Contaminants of potential concern based on the types of emissions expected from the Project include criterion air contaminants (CACs) such as fine particulate matter, sulphur dioxide, and nitrogen dioxide. Predictions concentrations of CACs were compared to the Yukon Ambient Air Quality Objectives (Yukon AAQO) as relevant health based threshold for both shorter duration exposures (1-hour, 8-hours, 24-hours depending on the CAC and chronic exposures (based on annual arithmetic or geometric mean concentrations). The predicted concentrations of CACs were lower than their respective Yukon AAQO at the focal areas of interest, and no health risks are predicted in association with exposures to CACs.

Various types of diesel and gasoline powered equipment also emit other types of especially volatile organic contaminants such a formaldehyde and benzene, as well as limited volatility compounds such as polycyclic aromatic hydrocarbons. The air dispersion modelling provided predicted concentrations during the peak operational year, at the focal areas of interest of total volatile organic compounds (TVOC) and total polycyclic aromatic hydrocarbons (TPAH). The predicted exposures from breathing to formaldehyde, acetaldehyde, benzene, acrolein, 1,3-butadiene, naphthalene, and benzo[a]pyrene were estimated from the TVOC and TPAH predictions for this risk assessment based on literature-based summaries of the composition of these compounds as a fraction of total organic matter in diesel engine exhaust. The estimated concentrations of these individual compounds was compared to human threshold of health effects values developed by the World Health Agency, United States Environmental Protection Agency, United States Agency for Toxic Substances and Disease Registry, California Office of Environmental Health Hazard Assessment, and the Texas Commission on Environmental Quality. Exposures to any of the individual volatile organic contaminants at the focal areas of interest are not predicted to exceed health-based exposure thresholds, either for acute or chronic exposure scenarios. Cancer risks from benzo[a]pyrene inhalation in association with mine operational emissions sources are also predicted to be acceptably low. Overall, no health risks are predicted in association with breathing in contaminants arising from Project emissions.

E-3 CONTAMINANT HEALTH RISKS FROM COUNTRY FOOD GATHERING AND CONSUMPTION

The purpose of the country food safety HHRA was to provide a good understanding of the potential health risks associated with the influence of the Coffee mine on desired human ingestion of plants, birds and mammals that ingest the affected plants, and of fish. This is particularly important in light of the traditional and ongoing importance of local and regional country food and medicinal plant resources to First Nations people and others. Changes in soil quality from mining-related dust deposition - and the associated direct deposition of dust to plant surfaces - could result in increased human exposures to those trace elements that occur at much higher concentrations in mining-related dust than in the existing forested area surface soils in general. In addition, areas with altered soil quality or water quality could result in altered trace metal uptake into edible forest and stream resources. While the focus herein is on the evaluation of potential for

contaminant uptake via dietary intake, the HHRA also assessed the exposures and risks for people while hunting and gathering in mining affected areas through direct exposures from incidental soil ingestion.

As discussed in **Section 24.0 Land and Resource Use**, commonly used edible plants in the Coffee Creek area and along the NAR include raspberries, blueberries, blackberries, salmonberries, highbush cranberries, lowbush cranberries, blueberries, kinnikinnik (bearberry), crowberry, Bear root, and Labrador tea. Morel mushrooms are routinely harvested in the spring in coniferous forest areas that experienced a summer wildfire in the preceding year. Historical or current hunting efforts have focussed in the fortymile woodland caribou, moose, wolves, bears, thinhorn sheep, porcupine, beaver, muskrat, rabbits, ptarmigan, grouse, ducks, and geese. In addition, trapping provides not just furs but also meat resources. The animals that are often trapped include lynx, fox, wolverine, mink, marten, and snowshoe hare. The ridges surrounding Coffee Creek and the northern bank of the Yukon River across from Coffee Creek have been important trapping areas, and some of the earliest registered traplines are documents in that area. Coffee Creek is well known for being an important fishing location which was used in the past. TH, Selkirk First Nation (SFN), and White River First Nation (WRFN) would travel to Coffee Creek and fish. Several fish are fished in the Coffee Creek area including Chinook and Chum salmon, whitefish and grayling.

Based on detailed evaluation of the chemical composition of representative samples of ore, host rock (which will be deposited as waste rock) and candidate borrow sources along the NAR, it was concluded that only arsenic in ore or waste rock exhibits an average concentration greater than either background soil concentrations based on the documented upper crustal abundances of trace elements or generic soil screening levels derived to be protective of human health based on direct exposure scenarios. Arsenic was selected as a contaminant of potential concern (COPC) therefore, while other trace elements were discounted as being of concern.

Based on the chemical composition of candidate borrow source samples, use in road construction and top-dressing, or mobilization as dust followed by dust fall in adjacent areas will not appreciably alter the chemical composition of forest soils or plants. Therefore, no health risks from dust fall associated with transportation activities along the NAR are expected.

For dust fall arising from the blasting, hauling and deposition of waste rock and ore, or the crushing and conveyance of ore, the predicted concentrations of arsenic based on cumulative loading to soils over the entire operational mine life amounted to only 0.7% of the average observed arsenic concentrations in native soils pre-mining, and ≤ 0.01% of observed arsenic concentrations in native soils in any focal area of interest beyond the mine-site proper. As such, there will be no detectable changes in arsenic levels in forest soils and biota as a result of dust fall. There is no potential for increased human exposures or risks, therefore.

Finally, human exposures to arsenic were estimated in the post-closure scenario for people who may spend time at and harvest plants and animals from land areas covered with waste rock, assuming (i) no cover on the deposits with clean material, and (ii) the presence over the longer term of plants and other food substances on waste rock deposits for collection. Human health exposure and risks were quantified in terms of daily and lifetime-average contaminant intake doses (milligrams or kilograms per day (mg/kg/day)) for the routes of exposure deemed to be potentially viable, including incidental soil or rock dust ingestion and inhalation, dermal exposure, and uptake into country foods via uptake from soil into plants. The total exposures from all pathways were predicted for each of granitic, gneiss and schist waste rock, based on 50th percentile concentration estimates for arsenic. This was to determine whether different risk management strategies may be necessary for the different rock types. Screening level calculations suggest that waste rock arsenic concentrations could potentially lead to marginally higher exposure levels than acceptable based on cancer risk potential based on the observed arsenic concentrations in granite waste rock but not the other two rock types. Risk management of granitic waste rock may be required, based on placement during operations, or at decommissioning. No other possible health risks were identified.

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ACRONYMS AND ABBREVIATIONS

Acronym / Abbreviation	Definition
LMAX	maximum noise levels
LN	night-time noise level from 10:00 p.m. to 6:00 a.m.
MAC	maximum acceptable concentration
MOE	Ministry of Environment
MPC	maximum permissible concentration
MSAT	mobile source air toxin
NAR	Northern Access Route
NNG	nighttime noise guidelines
NO2	nitrogen dioxide
OEHHA	Office of Environmental Health Hazard Assessment
PAH	polycyclic aromatic hydrocarbon
PM2.5, PM10	fine particle matter
ppb	parts per billion
ppm	parts per million
Project	Coffee Gold Mine
Proponent	Kaminak Gold Corporation, a wholly owned subsidiary of Goldcorp Inc.
RAA	Reginal Assessment Area
RQ	risk quotient
Sb	antimony
SFN	Selkirk First Nation
SO2	sulphur dioxide
TCEQ	Texas Commission on Environmental Quality
TDI	tolerable daily intake
TH	Tr'ondëk Hwëch'in
TPAH	total polycyclic organic hydrocarbons
TSP	total suspended particulates
TVOC	total volatile organic compounds
US EPA	Unites States Environmental Protection Agency
VOC	volatile organic compound
WHO	World Health Organization
WRFN	White River First Nation
YESAA	Yukon Environmental and Socio-Economic Assessment Act
YESAB	Yukon Environmental and Socio-economic Assessment Board

SYMBOLS AND UNITS OF MEASURE

Abbreviation	Measurement
µ/m^3	micrograms per cubic metre
µ/L	micrograms per litre
%	percent
dB	decibel
dBA	A-weighted decibel
km	kilometre
mg/kg/day	milligrams per kilogram per day
mg/L	milligrams per litre

1.0 INTRODUCTION

The proposed Coffee Gold Mine (Project) is a proposed gold development project in west-central Yukon, approximately 130 kilometres (km) south of Dawson City. The Project is proposed by Kaminak Gold Corp., a wholly owned subsidiary of Goldcorp Inc. (Proponent or Goldcorp). The Project is located on Crown Land within the traditional territory of Tr'ondëk Hwëch'in and the asserted area of WRFN. A portion of Goldcorp's claim block is located in SFN's traditional territory. The Project is scoped as an open pit gold mine using a cyanide heap leach process to extract ore. It would consist of an 18-month construction period, followed by a 12-year mine life with an average operation rate of five million tonnes per annum of heap leach feed.

It is important to consider how the Project, through construction, operations, closure and post-closure could influence the health and well-being of people who use the land and frequent areas in the vicinity of the proposed Mine Site or NAR. The purpose of this technical report is to document the scope, methods, results and conclusion of a quantitative HHRA for the proposed Project.

A major reason for undertaking an HHRA is to identify those circumstances where risks to the health of individuals and larger groups of people cannot be confidently discounted, and to develop appropriate risk management approaches for the purpose of preventing adverse health outcomes. Conclusions about risk that arise from this HHRA are an important step for identifying any potential need for risk management actions, or mitigations, against any adverse health outcomes.

1.1 HUMAN HEALTH RISK ASSESSMENT VERSUS HEALTH IMPACT ASSESSMENT

Human health risk assessment is a tool that is useful for examining how changes in environmental quality (e.g., air quality, environmental noise, water quality, soil quality) could influence human health. The underlying premise is that changes in environmental quality can drive changes in the characteristics and magnitude of human exposures to stressors such as noise, or chemicals such as those trace elements present at atypically high concentrations in mine wastes or ore.

An HHRA is based on toxicological dose-response relationships that have been defined through epidemiological and laboratory-based scientific studies. The analysis assumes that the degree of human exposures to stressors or substances of interest can be measured or predicted. The analysis also assumes that a threshold for exposure can be identified with adequate confidence below which any associated risks of adverse health outcomes are highly unlikely. By its nature, HHRA is narrowly focussed on those questions that can be addressed through quantitative approaches to defining for any stressor or substance of interest: i.e. - *How much is too much from the perspective of protecting the health of humans?* and *Will an activity or situation result in exposures that could exceed that amount?*

The World Health Organization, Health Canada, and a large proportion of health authorities, organizations, researchers, and health care providers define human health and well-being far more broadly than the physiological health status associated with toxicological effects, as can be addressed using HHRA. The overall health and well-being of people and communities is influenced by a complex series of interacting health determinants, including various social determinants of health. Those linkages and relevant health determinants that can't be adequately captured in simple dose-response relationships are the major subject of HIA. An HHRA and HIA, therefore, are complimentary approaches for developing an adequate understanding about adverse effects on health.

1.2 RELEVANT INFORMATION

This HHRA was completed in part through use of the following information:

- Our understanding of the Project, including mine construction, operation, closure, and post-closure activities and conditions, as discussed in **Section 2.0 Project Description** of the Project Proposal being prepared for the socio-economic and environmental assessment required by the YESAA.
- The current environmental conditions, prior to any potential drivers of change in environmental quality, especially with regard to the acoustic environment and air quality (**Sections 9.0 and 10.0; Appendix 9-A),** soil quality (**Section 11.0; Appendix 11-A**), and water and sediment quality (**Section 12.0; Appendix 12-A**)
- Land and resource use, and locations of residences or various activities close to the NAR or proposed Mine Site for the Tr'ondëk Hwëch'in, White River First Nation, Na-cho Nyäk Dun, Selkirk First Nation, and various other interested parties, affected communities, and the public (**Section 3.0 Consultation** and **Section 4.0 Project Setting**; **Sections 18.0 Introduction to the Human Environment** and **Section 24.0 Land and Resource Use Assessment**)
- Locations within the proposed Mine Site where occupational workers could be exposed to airborne contaminants or noise over extended durations (**Section 2.0 Project Description**)
- Air quality and dust fall predictions (**Section 9.0; Appendix 9-B**)
- Noise predictions for the NAR and mine (**Section 10.0; Appendix 10-A**).

1.3 STUDY OBJECTIVES

The purpose of this study was to evaluate the potential for adverse effects on human health as a result of changes in environmental quality that are attributable to the Project. The study components, major objectives, and a brief overview of the study are provided in **Table 1-1** and discussed below.

Table 1-1 Human Health Risk Assessment Study Components and Major Objectives

Component	Major Objective(s)	Brief Overview
Health Assessment for Existing Conditions	• Describe existing conditions with respect to health within the region affected by changes in environmental quality from the Project • Compare health status indicators for the Project region to health status indicators for Canada in general.	Identify and describe biomedical health indicators affected by changes, especially in air quality and noise, including asthma, lung cancer and respiratory disease.
Noise	• Characterize potential health risks associated with noise from the Project	Identify noise sources and types that could produce adverse health effects to people located within the range where the noise is perceptible relative to background noise. Quantify near worst-case exposures during construction and during the peak operational year. Compare the predicted exposures to scientifically defensible effects thresholds for stress and annoyance, sleep disturbance, or communications / learning interference.
Air Quality	• Characterize potential health risks associated with direct inhalation exposure to airborne contaminants	Identify chemicals in Project emissions that could produce adverse health effects following inhalation. Identify receptor locations for near worst-case exposure potential. Quantify exposures based on existing air quality conditions and predicted air quality during the peak operational year (Year 6). Compare predicted air concentrations to health-based, acute and chronic inhalation exposure limits, for both occupationally exposed individuals and members of the public.
Country Food Safety	• Characterize potential health risks associated with indirect exposure to Project emissions and dust fall following wet and dry deposition onto forest soil and plant surfaces, followed by human ingestion of plants or of birds and mammals that ingest affected plants	Identify chemicals for which adverse health effects could occur through chronic exposure affected by deposition of Project emissions or dust fall. Define expected chemical composition of dust fall, near the Mine Site and along the NAR. Quantify chemical concentrations in soil and plants as a result of direct deposition of airborne chemicals or dust fall. Quantify chemical concentrations in wildlife as a result of soil and plant ingestion. Compare estimated exposures via use of country foods with health-based exposure limits.

2.0 HUMAN HEALTH RISK ASSESSMENT SCOPE

Human health risks from a proposed project or activity are only plausible if there is a **source** of stressors or substances released to the environment, humans (as "**receptors**" of the exposure) that are adequately close to the area where an environmental change has occurred or will occur, and **exposure pathways** or mechanisms that connect the receptor to the source. If any of these three elements is absent then the possibility of health risk can be qualitatively ruled out, without further quantitative analysis.

As summarized in **Appendix 5-A Project Interaction Matrix)**, many aspects of Project-related changes in the physical and biophysical environment could interact with community health and wellbeing. Given the historical and ongoing importance to First Nations and Yukoners in general of lands, water, and resources in the Project area and along the NAR, Project-related changes in surface geology and soils, groundwater, surface hydrology, water quality, air quality, and noise merit close evaluation with regard to community health and well-being.

Human exposures to noise can result in increased stress and annoyance, sleep disturbance, or a decreased ability to communicate, focus on important tasks, and learn, depending on the specific circumstances associated with noise exposures. This is discussed in greater detail in **subsection 3.1** herein. Beyond the possibility that noise can result in direct physiological (or somatic[1]) responses in humans, the loss of tranquility in previously undeveloped areas of wilderness can influence well-being in ways not well captured by HHRA methods, and therefore this is addressed in the complimentary HIA. This analysis examines changes in noise holistically through identifying both the areas were the Project could result in a perceptible change in noise relative to background conditions and the areas where Project-related noise could result in specific, negative physiological and functional responses in people present.

Project-related changes in the environmental quality of groundwater and surface water, sediment and soil, or air quality could affect humans that interact with the local environment through direct exposure pathways, including the ingestion of water, incidental soil and sediment ingestion (for example, as occurs when people get soil on their hands and then place them in their mouth when eating), inhalation, and uptake across the skin (dermal uptake).

Project-related changes in the environmental quality of the physical environment can also increase contaminant exposures in people through indirect pathways, to the extent that contaminants can be taken up into edible food substances such as plants or edible fish and wildlife. Such indirect human exposure pathways are an important part of this HHRA in light of the importance of local plants and animal resources to both aboriginal and non-aboriginal people in the region.

[1] Somatic: of or relating to the body, especially as distinct from the mind.

2.1 SPATIAL BOUNDARIES

The spatial scope of the HHRA includes any area where Project-related changes in noise, air quality or dust fall could be distinguished from background conditions. The spatial scope also encompasses any people who may harvest and / or consume country foods from areas that could potentially be influenced by mine activities.

The spatial boundaries for the HHRA are largely aligned with the spatial boundaries for the noise assessment (**Section 10.0**) and air quality assessment (**Section 9.0**) since these are the main drivers of interest with regard to Project-related changes in environmental quality.

The spatial boundaries for the HHRA also include:

- Mine Site areas where mine wastes will be deposited and remain at the land surface in an accessible manner post-closure (**Section 11.0**), since these are areas where there could be altered accumulation of some trace elements in plants and wildlife.
- Surface waters where the Project could alter sediment and water quality (**Section 12.0**), in light of the possibility of altering the concentrations of some trace elements in fish tissues that might be consumed by people.

2.1.1 NOISE HRRA SPATIAL BOUNDARIES

- Local Assessment Area (LAA): Per **Section 10.0** of the Project Proposal, the LAA for noise includes an area that extends 1.5 km outward in all directions from the Mine Site area and laterally on either side of the NAR. The rationale for this distance is that Project-related noise, either during construction or operation, would generally not be discernible beyond the 1.5 km boundary. Regardless of the articulated boundaries, the LAA includes all areas where there is a possibility of a perceptible change in noise.
- Regional Assessment Area (RAA): The RAA encompasses the LAA and extends farther outward, encompassing an area the boundaries for which extend five kilometres outward in all directions from the Mine Site area and laterally on either side of the NAR. The purpose of the RAA is to place Project-related changes within the LAA in context based on the background conditions that are reasonably expected there both currently and in the future. This is also the area in which any residual changes associated with the Project could potentially interact with the residual effects of other past, present, or future projects or activities to result in a cumulative change or changes.

2.1.2 AIR QUALITY AND DUST FALL SPATIAL BOUNDARIES

- LAA: Per **Section 9.0** of the Project Proposal, the LAA for air quality and dust fall around the mine site is a grid extending 44 km in the east-west direction and 29 km in the north-south direction, approximately centred around the mine open pits. The LAA for the NAR includes an area extending two kilometres laterally from the road on either side.
- RAA: The RAA encompasses the LAA and extends farther outward, and is intended to encompass an area in which any residual changes associated with the Project could potentially interact with the residual effects of other past, present, or future projects or activities to result in a cumulative change or changes.

2.1.3 COUNTRY FOOD SAFETY SPATIAL BOUNDARIES

Plants and wildlife could become contaminated to the extent that airborne contaminants from combustion sources and dust fall are deposited on local soils and vegetation. The LAA for examining health risks associated with country food consumption, therefore, is linked to the LAA for air quality and dust fall as discussed above.

In addition, plants growing on surface accessible mine waste and geological disturbances theoretically could alter the quality of food substances consumed by people in the region. The areas where this is possible are found within the air quality and dust fall LAA.

Finally, fish inhabiting surface waters could be affected by water quality if adversely affected by the project and in turn the fish could be consumed by humans. The LAA for country food safety, therefore, also includes fish-bearing sections of Coffee Creek / Latte Creek, Halfway Creek, and other small watersheds that are potentially affected by the Project. This includes the LAA for air quality and dust fall around the Mine Site.

The RAA for the purpose of evaluating health risk potential associated with country food consumption includes the west central Yukon in general, based on the broader resource use activities of the TH, WRFN, Na-cho Nyäk Dun (FNNND), SFN, and various other interested parties, affected communities, and the public. The RAA provides a regional context for the HHRA and encompasses the area for which cumulative effects on country food quality are evaluated.

2.1.4 HRRA SPATIAL BOUNDARIES – SUMMARY

The overall spatial boundaries for this HHRA are as summarized in **Table 2-1.**

Table 2-1 Summary of Spatial Boundaries for the Human Health Risk Assessment

HHRA Component	LAA	RAA
Noise	1.5 km outward from Mine Site and NAR	5 km outward from Mine Site and NAR
Air Quality and Dust fall	Mine Site: gridded area 44 km (E – W) x 29 km (N – S) Access route: 2 km on either side of access road	Approx. 100 km radius of mine and access road
Country Food Safety	The cumulative area associated with: (i) areas where mining related dust fall or deposition of other airborne contaminants could deposit to soils or plants surfaces, generally comprising the LAA for the air quality and dust fall assessment (immediately above); (ii) areas where surface soil quality will be altered post closure as a result of mine waste deposits or mining physical disturbances; and (iii) any area where surface water quality could change as a result of the project, either within habitat that supports edible freshwater resources upstream.	Regional area that supports aboriginal and non-aboriginal resource use (country foods and medicinal plants), generally within 100 km radius of the mine and access road.

2.2 TEMPORAL BOUNDARIES

Positive and negative influences of the proposed Project on human health are of interest during the construction phase (18 months), operations phase (estimated to be 10 years based on currently proven reserves), through decommissioning and closure (approximately 10 years), and post closure. For each of the three major HHRA components (noise health effects, air quality health effects, country food safety), health risks are expected to be different for different Project phases.

For noise and air quality health effects, sources of noise and air emissions are expected during construction, operation, and decommission / closure. Dust generation and dust fall in the post closure period are expected to be minimal, and any contaminant exposures from the breathing in of suspended particulates are likely to be very small in comparison to the construction, operation or decommissioning phases. No mining related sources of noise or combustion emissions are expected in the post closure phase, so noise and air quality are not assessed in the HHRA for the post closure conditions due to a lack of any viable exposure pathways.

This HHRA defines human contaminant (and noise) exposure potential, and the associated health risk potential, based on near worst-case scenarios for noise, air quality, and dust fall, through construction, mine operation, decommissioning, and post-closure. For noise, the near-worst case conditions have been modelled for both construction (Year –1) and mine operation (peak operational year: Year 6), as described in **Section 10.0 and Appendix 10-A.** Based on the reasonably foreseeable types of activities, these years represent years of peak activities that are expected to generate noise **(Noise Emissions Inventory Report**).

Similarly, for air quality, near-worst case conditions with regard to human inhalation exposures have been modelled for the peak operational year (Year 6). For most criterion air contaminants [CACs, including carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), fine particulate matter ($PM_{2.5}$, PM_{10}) and total suspended particulates (TSP)], the air quality assessment (**Section 9.0 and Appendix 9-B**) assumes that peak operation phase emissions will result in greater flux to the lower atmosphere than for all simultaneously occurring construction phase activities that could emit contaminants to the airshed.

The HHRA for country food safety addresses possible human exposures to plants, fish and wildlife, that could be potentially affected by altered soil and water quality during operations and decommissioning, and particularly in the post-closure period. During the analysis, it is important to consider how the characteristics of surface accessible mine wastes (especially post closure) might alter human exposures to various trace elements, through incidental ingestion of soil and uptake into edible food and medicinal resources. The post-closure exposure scenario is considered to be a near-worst case scenario relative to the operating and decommissioning phase in light of the expected spatial extent of waste rock deposits and a potentially greater use of the mine disturbance area by people who access food resources. The HHRA is based on a

worst case scenario for altered soil quality and country food consumption, assuming limited to no covering of waste rock deposits at closure with salvaged or other soils.

The temporal scope for the various HHRA components is summarized in **Table 2-2**.

Table 2-2 Summary of Temporal Scope for Human Health Risk Assessment Components

HHRA Component	Project Phase			
	Construction	Operation	Decommissioning / Closure	Post-Closure
Noise	Year -1 noise modelled as near-worst case	Year 6 modelled as near-worst case	Assessed indirectly based on expected similarity to or lesser noise-generating activity than during construction or peak operations	Not assessed: No mining related noise sources anticipated
Air Quality		Year 6 modelled as near-worst case	Assessed indirectly based on expected similarity to or lesser emissions than during construction or peak operations	Not assessed: No mining related noise sources anticipated
Country Food Safety	Not assessed: Changes in soil quality expected to be much smaller than for subsequent Project phases	Assessed indirectly based on expectation that any magnitude of change in soil quality from dust fall or direct disturbance and mine water deposition will be lower than for the cumulative influence of mining as reflected in post closure conditions	As for the operation phase	Focus of assessment as being near-worst case scenario. Assessed based on predicted chemistry of surface accessible waste deposits following closure as well as cumulative mass inputs of trace elements as a result of dust fall

2.3 ADMINISTRATIVE BOUNDARIES

The administrative / management boundaries that inform the execution of this HHRA are as discussed in **Section 4.0 Project Setting.** The Project, including the mine site and the northern access road, is located entirely on Crown land. The mine site is within the traditional territory of TH and the asserted territory of WRFN. A portion of Kaminak's claim block is located in the shared traditional territory of SFN. The road alignment is located within the traditional territory of TH, portions of which are located within the shared traditional territories of SFN and FNNND and the asserted area of WRFN.

The Project footprint does not overlap with any First Nation Category A or B Settlement Lands as defined in the Umbrella Final Agreement between the Government of Canada, The Council for Yukon First Nations, and The Government of the Yukon. There is a Category B camp site near the mouth of Coffee Creek, however.

The major portion of the Mine Site and most of the northern access road is located within the Dawson Land Use Planning Region. The Project footprint is not located within any Special Management Area or Habitat Protection Area as enabled under the *Wildlife Management Act.*

Of importance to the HHRA is that two trapline concession areas overlap with the Mine Site and seven trapline concession areas overlap with the northern access road. Also of particular relevance to this HHRA is the existing and anticipated future land and resource use, as discussed in **Section 24.0** and **Appendix 24-A** of the Project Proposal.

2.4 TECHNICAL BOUNDARIES

The degree of confidence in conclusions about human health risk potential that result from a HHRA are related to the following:

(i) Predictive accuracy of changes in environmental quality that could influence human exposures to contaminants or stressors (i.e., as provided in the noise and air quality assessments)

(ii) How well the modelled exposure scenarios capture the true interactions between humans and their environment, through interactions with soil, breathing, food and medicinal plant consumption, listening / hearing, and other experiential modes

(iii) Adequacy of the available thresholds of effects estimates used in the assessment to protect the health of humans, including those individuals and groups that may be more sensitive to contaminants / stressors than the population in general (for example, developing children, the elderly, pregnant mothers, or those suffering from other diseases).

It is common practice in HHRA to define key areas of uncertainty and discuss their possible influence on conclusions about health risks; accordingly, this HHRA for each of noise, air quality and country food consumption includes a discussion about uncertainties.

There is some degree of uncertainty in any analysis. For HHRA, this is addressed by conservatively assuming conditions that would tend to over-estimate levels of exposures relative to the true case and to use lower thresholds of effects levels than specifically supported by epidemiological and toxicological studies in the face of uncertainty. HHRA, by design, is biased towards over-predicting health risks if there is any uncertainty about key inputs into the risk characterization. As such, a finding that health risks would be acceptably low generally provides confidence that the health of the people of interest will not be compromised by the issue of interest, while a finding that health risks may be unacceptably high may suggest either that (i) some type of active risk management or risk reduction may be needed, or (ii) a more detailed analysis is required, accompanied by efforts to decrease key sources of uncertainty.

3.0 NOISE HEALTH RISK ASSESSMENT

This section provides the technical details of the HHRA for noise. The primary objective of HHRA is to evaluate whether noise arising from Project activities could have an effect on human health. Noise predicted during both Project construction and operation are considered.

3.1 NOISE CHARACTERISTICS AND METRICS

Noise may be defined as unwanted sound. The noise level at any given location is rarely steady from one moment to the next, even in quiet situations, and will vary over a range depending on the characteristics of local noise sources. Close to a busy highway, for example, the noise level may vary over a narrow range of only 5 decibels (dB), as the near continual flow of traffic provides a persistent stream of overlapping noise events. In more remote areas near roadways, this variability may increase to 15-25 dB since the traffic-related noise sources are less frequent, transient, and imposed on a much lower background noise level. Furthermore, in most locations, the range of night-time noise levels will often be smaller and the levels significantly reduced compared to daytime levels, as activity rates tend to be lower at night. When considering environmental noise, it is necessary to consider how to quantify these variable noise conditions to account for moment to moment, and longer term variations. Consequently, the science of acoustics has developed a range of different noise metrics that produce more easily interpreted single figure values that are intended to describe how people experience noise exposures and may be affected by them.

This noise HHRA, relies on background noise measurements and interpretation, as documented in **Appendix 9-A**, as well as predicted noise characteristics at and near the proposed mine and along the NAR, as discussed in **Section 10.0** and **Appendix 10-A**.

Noise from a given source can occur over a large range of frequencies, from pitches that are of a frequency too high to be audible to humans to very low frequency noise that may also be sub-audible to humans. The noise characteristics that are discussed herein generally occur within the audible range for human hearing, and have been quantified as A-weighted noise. The range of frequencies that are generally perceptible by humans may be different for various other animals, and this is addressed separately, in the **Wildlife and Wildlife Habitat Assessment** and **Birds and Bird Habitat Assessment** section of the Project Proposal (**Section 16.0** and **Section 17.0**, respectively).

The ideal noise metric for assessing the influence of noise on human perceptions and health would have all of the following attributes:

- Captures the absolute or peak noise level of the noise emission source
- Describes the duration that the noise is audible at a specific location
- Indicates the degree to which the noise exceeds the ambient noise
- Measures how often the noise occurs

- Is associated with the different health effects of Project-related noise that are of interest (for example, in relation to annoyance, sleep and activity disturbance, speech interference, etc.)
- Can be easily measured
- Can be readily modelled and predicted, and
- Is readily understood by non-specialists.

Unfortunately, no single noise metric has yet been developed that can meet all of the above requirements. It is necessary, therefore, to select a primary noise metric that covers as many of the above attributes as possible, as well as supplementary metrics, so that cumulatively, the primary and supplementary metrics serve the objectives of the HHRA. A brief summary of the broader range of metrics used worldwide in contemporary evaluations of noise effects is provided in **Table 3-1**.

This noise HHRA is based in particular on continuous noise metrics such as L_{EQ} for those aspects of construction and mining that are expected to result in ongoing noise generation over extended durations, such as ore crushing, haul truck operations, or diesel power generation. Many of the mining operations and construction activities are currently anticipated to occur around the clock, and the noise modelling as discussed in **Section 10.0** was completed based on the assumption that continuous noise generation will occur at the maximum output magnitude (maximum sound power level) continuously without pause for all equipment or other noise sources operating during either the peak construction or peak mine operational period. For the purpose of the noise HHRA, we divide the 24-h L_{EQ} predictions into their daytime (L_D) and nighttime components (L_N) since the first of these is most relevant to possible interference with communications or learning, while the second is important for sleep disturbance, as discussed in **Section 3.3**.

For more transient types of noise, such as the high energy impulsive noise associated with pit blasts, the primary metric used in this assessment is L_{MAX}.

3.2 FOCAL AREAS OF INTEREST

The noise existing conditions and predictions for construction and operation are presented in **Section 10.0** for the entire LAA and outward into the RAA. It is also useful, however, to develop an understanding about potential project-related changes in noise at locations where people may permanently or temporarily reside, or frequently use for recreational, food-gathering or spiritual purposes, *et cetera*. Based on consultations with TH members, WRFN members, and others, a candidate list of Focal Areas of Interest (FAI) was established, as summarized in **Table 3-2**.

Table 3-1 Summary of Several Commonly Used Noise Metrics

Application	Metric	Brief Description	Unit	Relevant Time Period (h)
Continuous noise exposure over a predefined period such as a 24-h period or night time.	**L_{EQ}**	**Equivalent Noise Measure**: cumulative noise metric based on steady state noise over a defined period. A logarithmic mean over the period of interest.	dBA*	Variable – For example 1 h, 24 h, 16 h Day, 8 h Night
Speech interference Learning disruption in early learning settings	**L_D**	**Daytime Noise Level:** an L_{EQ} (or logarithmic mean) for the daytime period only (6:00 a.m. to 10:00 p.m.)	dBA	16 h Day
Sleep disturbance	**L_N**	**Nighttime Noise Level:** an L_{EQ} (or logarithmic mean) for the nighttime period only (10:00 p.m. to 6:00 a.m.)	dBA	8 h Night
Stress and annoyance	**L_{DN}**	**Day-Night Noise Level:** This is an equivalent noise measurement (L_{EQ}) as described above except that noise occurring in the night-time period (e.g. from 10:00 p.m. to 6:00 a.m.) is adjusted by adding 10 dBA to the noise level before calculating a 24-h logarithmic mean.	dBA	24 h
Speech interference Sleep disturbance	**L_{MAX}**	**Maximum Sound Level**: highest A-weighted sound level during a distinct event, or over a period such as the night-time	dBA	Dependent on event duration
Sleep disturbance	**SEL**	**Sound Exposure Level**: composite metric that captures both the intensity and duration. SEL approximates the net effect of an entire acoustic event, since it estimates on a logarithmic scale the total sound energy transmitted to a recipient during a specified event. Can be used to predict the % likelihood of awakening.	dBA	Dependent on event duration
Supplemental Metrics	**TA**	Time above (TA): The amount of time that noise levels are greater than a given threshold.	Minutes / day	Daily
	NA	Number of Events Above (NA): The number of noise events exceeding a given threshold.	Events / day or Events / night	Daily Nightly

Note: the "A" in dBA denotes that the measured or predicted noise is A-weighted.

Such areas are sometimes also referred to as "sensitive receptor" locations in noise and air quality effects assessments, since they reflect example areas that are both:

- Sufficiently near the air emissions or noise sources of interest that they reflect near worst-case in terms of the human exposure potential, and
- Areas where people are present for extended periods of time, and where people may be sensitized to potential negative effects based on the types of activities that the area supports (for example, culturally important areas, early learning and infant or child care facilities, extended health care facilities, and areas important for recreation and tranquility).

Table 3-2 Representative Focal Areas of Interest for Noise Human Health Risk Assessment

FAI No.	Description	Easting	Northing	Comments
Mine Site Proper - Occupational and On-site Exposures				
CO-01	*Permanent Camp, incl. dormitory and kitchen / dining / recreation complex*	582466.5	6972701.7	294 beds; within ~400 m of crusher and fine ore stockpile. Less than 300 m from power plant. Waste management b (incinerator) immediately south
CO-02	*Mine Dry and Office Complex*	582487.9	6972637.4	Capable of accommodating 85 workers during shift change
CO-03	*Assay Lab*	582190.0	6972642.1	Part of plant site. ~70 m from power plant
CO-04	*Truck Shop / warehouse*	582283.5	6972671.5	Part of plant site. ~200 m from power plant
CO-05	*Mine Access Road at Airstrip*	589903.2	6973349.8	
Other areas adjacent to the Mine Site and / or road – sensitive wildlife or ecological areas and / or of traditional resource acquisition / cultural / spiritual significance, etc.				
SA-01	*Mouth of Coffee Creek*	599265.0	6976770.0	Reflects Traditional knowledge. Camp site is designated as Category B land
SA-01a	*Mouth of Coffee Creek (same as VM4)*	599493.2	6977076.7	Yukon Wildlife Adventure Camp Location
SA-02	*Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth*	600375.5	6976672.7	SA-02 through 07 are intended to capture the importance of the Yukon River as a transit route, especially during the summer period
SA-03	*Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth*	597987.0	697767.07	Same as VM3 as used in the visual impact assessment
SA-04	*Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth*	596481.5	6977686.6	
SA-05	*Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth*	594477.5	6977597.4	
SA-06	*Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth*	592664.5	6977965.0	
SA-07	*Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth*	590020.0	6979143.4	
SA-08	*Confluence of Latte and Coffee Creeks*	595071.1	6971367.7	
SA-09	*Height of land across Yukon R. from Coffee Creek*	596943.8	6978818.5	
SA-10	*Height of land across Yukon R. from proposed Mine Site*	592412.0	6979525.0	
SA-11	*Height of land across Yukon R. to ea.*	601500.9	6976847.1	High use wildlife area
SA-12	*Ballarat Creek Area, N. of Yukon River*	603514.3	6977718.8	potential country food sources close to mine
SA-13	*Yukon River foreshore east of existing Coffee Creek camp*	596828.2	6977439.7	YWA Suggested New Camp Location
SA-14	*Wilderness Retreat, on Yukon River*	581850.0	6985410.0	
SA-15	*Representative harvesting area - height of land*	584429.1	6986561.8	

Note: These locations are also shown in **Figure 3-1**.

Five locations capture near worst-case noise conditions within the mine operational area, including at the permanent camp complex (CO-01).

The land and water near the mouth of Coffee Creek has had great historical importance to First Nations people and is currently highly valued. FAI SA-01 and SA-01a are intended to be broadly representative of areas around the mouth of Coffee Creek.

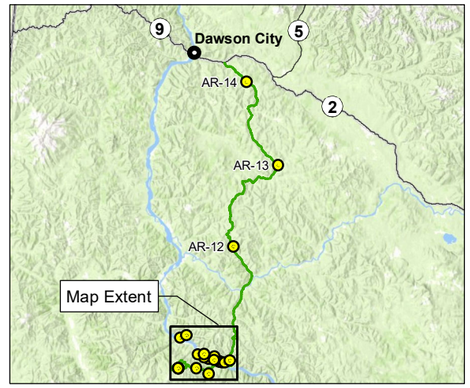
Seven locations are intended to capture noise that might be experienced by those travelling on the Yukon River or walking its shores (SA-02 to -08), extending from approximately 1.5 km upriver to 10 km downriver from the mouth of Coffee Creek. The Coffee Creek and Latte Creek valleys are important travel and resource access routes for White River First Nation and Tr'ondëk Hwëch'in, as well as other aboriginal people. Site SA-08 is located at the confluence of Coffee and Latte Creeks.

Locations SA-09, -10, -11 and -15 are located near the height of land across the Yukon River from the Mine Site. These are higher elevation areas that are across the river valley from the proposed Mine Site and partially face the Mine Site area. Thus they are minimally shielded from project-related sound based on topography. The areas are also representative of country food gathering, and wildlife use areas.

Location SA-12 is at the Ballarat Creek mouth on the Yukon River, which is an important staging and temporary residence area. Similarly locations SA-13 and -14 are areas of interest to outfitters and others as important camping and recreational areas.

COFFEE GOLD MINE

Focal Areas of Interest Included in the HHRA



SA-15
SA-14
Yukon River
SA-07
SA-10
SA-09
SA-06
SA-05
SA-04
SA-13
SA-03
SA-12
SA-01a
SA-01
SA-11
SA-02
CO-03
CO-04
CO-01
CO-02
CO-05
SA-08

Legend

- Highway
- Project Footprint
- Focal Areas of Interest
- Dawson City

Notes

1. This map is not intended to be a "stand-alone" document, but a visual aid of the information contained within the referenced Report. It is intended to be used in conjunction with the scope of services and limitations described therein.

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The focal areas of interest listed in **Table 3-2** and shown in **Figure 3-1** reflect only a very small subset of areas of interest to Yukon people within the RAA near the proposed mine site and southern portion of the mine access road. Nonetheless, they were selected as being broadly representative of potential worst-case conditions for project-related noise or changes in air quality and dust fall. Land and water areas farther removed from the proposed mine are expected to experience lower exposure potential than assessed at the FAI discussed herein.

3.3 RELEVANT NOISE HEALTH INDICATORS AND EFFECTS THRESHOLDS

The HHRA for noise involves the identification of appropriate human health endpoints, and comparing published guidelines for these endpoints to Project-related exposure levels. There has been a significant increase in knowledge about potential human health effects from noise exposures over the last three to four decades, reflected in literally many hundreds of published epidemiological and research studies. Such studies have been driven especially by health concerns associated with transportation-related noise (aircraft overflights, road, rail) and noise sources loud enough to damage hearing.

Effects of project-related noise on hearing impairment are not considered in this HHRA because noise levels known to be associated with auditory damage are much higher than those associated with more subtle health effects related to stress and annoyance or sleep disturbance; Noise levels that can cause hearing impairment are much higher than that those predicted to occur as a result of project construction or operation, except, perhaps, noise produced within several metres of various types of equipment. As indicated in **Tables 5.1-1** and **5.2-1 of the Noise IC section**, sound power levels for the various types of equipment during construction or operation are expected to fall within the range of 70 dBA (conveyor) to 127 dBA (D50KS Drill).

Protection of worker safety in the Yukon is regulated under the Yukon *Occupational Health and Safety Act* and the associated *Yukon Occupational Health Regulations.* Under Section 4, “Noise Control”, of this regulation, maximum permissible noise exposure thresholds are defined for exposures without hearing protection. Regulated permissible noise values are 85 dBA and higher, depending on the exposure duration. Because the project proponent has a regulatory requirement to prevent unprotected exposures above the regulated permissible values, no risks associated with auditory impairment are expected and this issue is beyond the scope of this HHRA and Project Proposal.

The available studies on noise health effects other than hearing loss have been critically reviewed and synthesized by the World Health Organization (WHO, 1999, 2009, 2010), Health Canada (Michaud et al 2008) and other agencies (ISO 2003, ANSI 2005, FTA 2006). Health indicators for noise exposures that have been shown to have a reasonable causal relationship between adverse human health effects and noise exposure are preferred for the prediction of health effects. These include, in particular, high annoyance, sleep disturbance, and interference with speech comprehension.

3.3.1 THRESHOLDS OF PERCEIVED CHANGE IN CONTINUOUS ENVIRONMENTAL NOISE

It is generally accepted that human responses to noise will not change appreciably if the magnitude of change relative to existing conditions is sufficiently low (i.e. ≤ 3 dBA) that it cannot be perceived by the majority of people and the tonal characteristics of the additional noise are appreciably the same as the existing noise. A change in continuous noise metrics of ≤ 3 dBA, therefore, is often used in noise health effects assessments as a threshold below which incremental health risks are unlikely.

Because the project construction and operational phase noises will be imposed on a noise field comprising of generally natural sounds (rustling leaves, running water, bird vocalizations, insect noises, thunder, etc.), it is expected that the specific tonal characteristics of the new noises will be different than existing noises, and thus more readily perceived if not sufficiently low to be masked by the natural noise field.

3.3.2 PERCENT HIGHLY ANNOYED

High annoyance with noise is a reliable and widely accepted indicator of human health effects due to environmental noise (ISO 2003, ANSI 2005, U.S. FTA 2006, Michaud et al 2008). The change in percent of an exposed group of people that feel highly annoyed (percent highly annoyed: %HA) is commonly used as a measure of community response to noise. Evaluation of annoyance as a major health effect tends to capture other health effects as well, including sleep disturbance and speech interference, since these also increase an individual's perception of being highly annoyed, especially when exposed to elevated noise over extended durations.

The change in %HA is quantified by the difference in %HA calculated for the existing condition versus the %HA calculated after consideration of a project's noise contribution. The %HA is calculated using the following equation (ISO 2003, ANSI 2005, Michaud et al 2008):

$$\%\boldsymbol{HA} = \frac{100}{\mathbf{1} + \boldsymbol{e}^{exp[\mathbf{10.4 - 0.132} * RL]}}$$

In this equation, the rating level (RL) is typically an adjusted L_{DN}, with adjustments made depending on the type of noise source and source characteristics (for example, impulsive or transient characteristics, or frequency range).

The Federal Transportation Authority (FTA) published the *Transit Noise and Vibration Impact Assessment* (U.S. FTA 2006) guidance for use in characterizing impacts for all mass transit projects, including rapid, light or commuter rail, diesel / electric buses and their storage and maintenance yards. The FTA adopted a 6.5% increase in %HA as the guideline for a severe noise impact. Similarly, Health Canada has used the change of 6.5% highly annoyed criterion in reviews of environmental assessments to indicate the potential severity of project noise impacts (Michaud et al 2008).

3.3.3 SLEEP DISTURBANCE

Uninterrupted sleep is a prerequisite for good physiological and mental functioning of healthy persons, while sleep disturbance is considered to be a major environmental noise effect. Poor sleep is associated with changes on metabolic and endocrine functions leading to changes in inflammatory markers, glucose regulation and weight control. Collectively, these changes, if significant, can lead to increased risk of cardiovascular disease. Sleep disturbance as an effect of noise typically includes difficulty falling asleep, awakenings, curtailed sleep duration, alterations of sleep stages or depth, and increased body movements during sleep (WHO 1999, 2009).

WHO (2009) provided a review of the observed health effects associated with human exposures to noise at night over various continuous noise ranges, and their major conclusions are summarized in **Table 3-3**. At around 50 dBA there is a slight increase in the percentage exposed people who are highly sleep disturbed, but generally outdoor noise levels less than 50 dBA are not associated with significant increases in sleep disturbance related endpoints.

Table 3-3 Effects of Different Levels of Night Noise on the Population's Health

Average Night Noise Level over a Year $L_{night, outside}$	Health Effects Observed in the Population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. L_{night}, outside of 30 dB is equivalent to the no observed effect level (NOEL) for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. Lnight,outside of 40 dB is equivalent to the lowest observed adverse effect level (LOAEL) for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

WHO (2009) has established 40 dB L_N as a target night noise guideline (NNG) and 55 dB as an interim guideline. These guidelines are meant to be applied to outdoor noise predictions and measures, even though it is understood that the vast majority of epidemiological studies have focussed on people sleeping indoors. As a general approximation, WHO (1999) applies an assumed 15 dBA decrease in noise from the outside to inside of most dwelling. When applying the WHO (2009) nighttime sleep disturbance thresholds to the Project noise effects assessment, it is important to appreciate that some people may be potentially exposed to project-related noise while sleeping outdoors in tents or lean-tos for which minimal attenuation of outdoor noise levels is expected.

It is also important to consider that at exposure levels between 40 and 55 dBA, the guidelines do not necessarily differentiate between effect (increase in body movement) and significant adverse effect (e.g., increase in cardiovascular disease due to sleep disturbance). Thus while an outdoor nighttime noise level of 40 dBA or less could result in measurable changes in body movement or arousals during the sleep period, significant adverse effects on health outcomes such as increased incidence of cardiovascular disease generally are not expected below an outdoor nighttime noise level of 55 dBA.

3.3.4 Interference with Speech Comprehension

Noise interference with speech comprehension can result in a number of personal disabilities, handicaps, and behavioural changes (WHO 1999). For effective outdoor speech comprehension, the (U.S. EPA 1974) advises that background outdoor noise levels be kept below 55 dBA for continuous noise. This level considers that 95% sentence intelligibility is acceptable in outdoor environments where there is a distance of up to two metres between speakers. This level is also considered appropriate based on people's tendency to speak in a louder voice when outdoors, where the separation between speakers is typically greater than indoors, and where outdoor interferences such as wind, water, and animal sounds may raise background noise levels to 50 dBA. Allowing for a 15 dBA reduction in sound level between outdoors and indoors, background indoor sound levels for continuous noise should be maintained below 40 dBA to sustain adequate speech comprehension.

3.3.5 Summary of Indicators and Effects Thresholds

The above discussed noise health effect indictors and effect thresholds, used in this HHRA, are summarized in **Table 3-4.**

Table 3-4 Summary of Human Health Risk Assessment Noise Health Indicators and Effects Thresholds

Indicator	Threshold
Threshold for Perceptible Change from Existing Conditions (L_{EQ})	**3 dBA** change, assuming similar sound characteristics between existing and new noise field
Percent Highly Annoyed (%HA) (L_{DN})	**6.5%** increase in %HA from existing conditions
Sleep Disturbance (L_N, outdoors)	**40 dBA:** minor to moderate effects; not significant **55 dBA:** significant adverse effects
Speech and learning interference (L_D, outdoors)	**55 dBA**

3.4 NOISE EXPOSURE ASSESSMENT

3.4.1 MINE SITE VICINITY

The estimated noise levels at the focal areas of interest under the existing conditions and during peak construction or mine operation, as discussed in **Section 10.0**, are summarized in **Table 3-5**. As discussed above, the predicted noise levels during construction or operation were developed assuming the generation of peak sound pressure levels continuously over time for the noise-generating equipment and infrastructure. The environmental noise associated with peak noise generation is in fact a prediction of maximum noise level (L_{MAX}). Because it is assumed that the noise sources generate sound at peak levels continuously (which is unrealistic but reflects a worst-case scenario for this HHRA), the L_{MAX} values shown in **Table 3-5** do not exhibit an temporal variability over a 24-h period or longer, and thus have the same value as the logarithmically averaged continuous noise levels predicted by the modelling; i.e. the tabulated L_{MAX} values are equal to L_{EQ} values.

In reality, operation of a haul truck, excavator, drill, or screening and conveyor system will never result in continuous maximum sound pressure level outputs over longer duration periods.

3.4.2 MINE ACCESS ROAD

Noise predictions along the mine access road were based on an assumed maximum traffic volume of eight (8) trucks per day, with only daytime operations since no nighttime road operation is proposed for the Project. It was assumed that the vehicles travel at a speed of 40 km/h (**Section 10.5.3.1**).

The predicted noise levels in the vicinity of the access road during a pass-by are illustrated in **Figure 3-2.** See also access road noise contour predictions for operating year 6 illustrated in Figures 4-3 to 4-7 of **Appendix 10-A**.

As discussed in **Section 10.4.3** of the Noise IC Analysis Section, baseline noise monitoring in the Project area provided estimates of daytime background (natural) noise levels in the range of 25 to 31 dBA during winter and 22 to 37 dBA during summer. During the predicted peak operational period (Year 6), a background noise level of 35 dBA is not predicted to be exceeded more than 515 m distance lateral to the access road centre line, while a noise level of 30 dBA is not predicted to be exceeded more than 1,100 m distance lateral to the centreline.

Table 3-5 Predicted Noise Levels (LMAX ≈ LEQ) at Focal Areas of Interest

FAI No.	Description	Existing Conditions	Peak Construction Period	Peak Operational Period
Mine Site Proper - Occupational and On-site Exposures				
CO-01	*Permanent Camp, incl. dormitory and kitchen / dining / recreation complex*	Winter: - Day: 24 to 31 dBA - Night: 22 to 26 dBA Summer: - Day: 32 to 45 dBA - Night: 27 to 57 dBA*	>65 dBA	60 to 65 dBA
CO-02	*Mine Dry and Office Complex*		>65 dBa	60 to 65 dBA
CO-03	*Assay Lab*		>65 dBA	60 to 65 dBA
CO-04	*Truck Shop/warehouse*		>65 dBA	60 to 65 dBA
CO-05	*Mine Access Road at Airstrip*		35 to 40 dBA	35 to 40 dBA
Other areas adjacent to the Mine Site and / or road – sensitive wildlife or ecological areas and / or of traditional resource acquisition / cultural / spiritual significance, etc.				
SA-01	*Mouth of Coffee Creek*	Winter: - Day: 24 to 31 dBA - Night: 22 to 26 dBA Summer: - Day: 32 to 45 dBA - Night: 27 to 57 dBA*	<20 dBA	<20 dBA
SA-01a	*Mouth of Coffee Creek (same as VM4)*		<20 dBA	<20 dBA
SA-02	*Yukon River - centre channel, ~1.5 km upriver from Coffee Creek mouth*		<20 dBA	<20 dBA
SA-03	*Yukon River - centre channel, ~1.5 km upriver from Coffee Creek mouth*		<20 dBA	<20 dBA
SA-04	*Yukon River - centre channel, ~3 km downriver from Coffee Creek mouth*		<20 dBA	<20 dBA
SA-05	*Yukon River - centre channel, ~5 km downriver from Coffee Creek mouth*		<20 dBA	21 dBA
SA-06	*Yukon River - centre channel, ~7 km downriver from Coffee Creek mouth*		<20 dBA	24 dBa
SA-07	*Yukon River - centre channel, ~10 km downriver from Coffee Creek mouth*		22 dBA	28 dBA
SA-08	*Confluence of Latte and Coffee Creeks*		20 dBA	21 dBA
SA-09	*Height of land across Yukon R. from Coffee Creek*		<20 dBA	<20 dBA
SA-10	*Height of land across Yukon R. from proposed Mine Site*		20 dBA	24 dBA
SA-11	*Height of land across Yukon R. to east*		<20 dBA	<20 dBA
SA-12	*Ballarat Creek Area, N. of Yukon River*		<20 dBA	<20 dBA
SA-13	*Yukon River foreshore east of existing Coffee Creek camp*		<20 dBA	<20 dBA
SA-14	*Wilderness Retreat, on Yukon River*		<20 dBA	<20 dBA

* includes periods with noise resulting from precipitation events

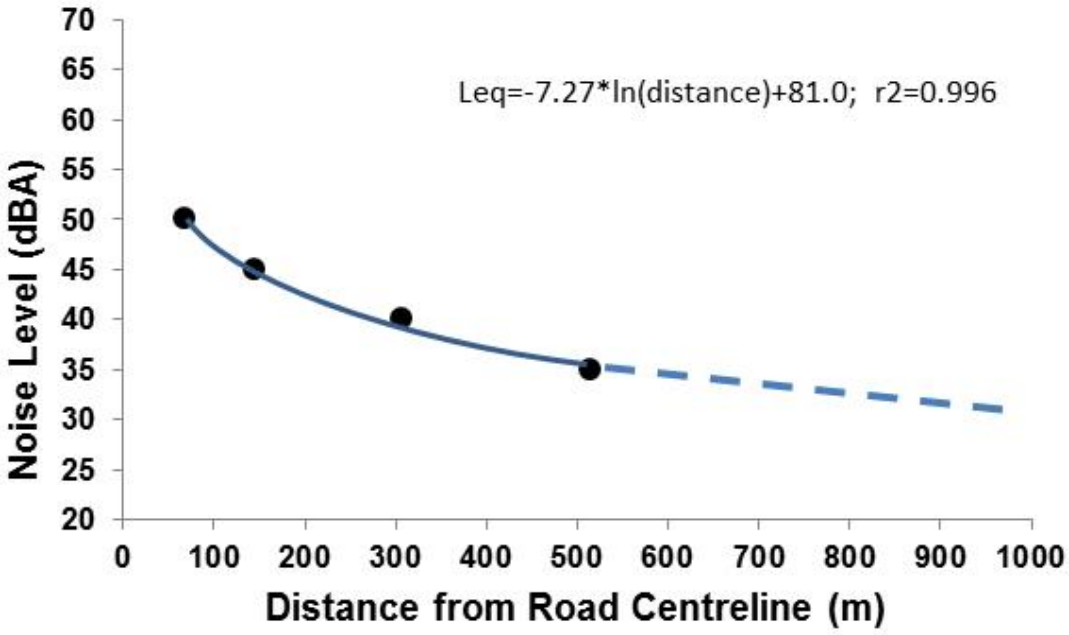

Figure 3-2 Predicted Attenuation of Noise Away from Road Centreline During a Truck Pass - Operating Year 6

3.5 RISK CHARACTERIZATION AND UNCERTAINTY ANALYSIS

The Project noise health risk potential is based on the predicted noise exposure levels for the noise health indicators in relation to threshold of health effects levels as summarized in **Table 3-4**.

3.5.1 MINE SITE VICINITY

The predicted noise during the peak construction or operation period is summarized in **Table 3-5**. Noise health risks associated human noise exposures while on the mine-site (i.e. for focal areas of interest (CO-01 through -05 and Mine Site areas with noise conditions) are not quantified herein since members of the public will generally not be present in these areas for extended periods. Health risks from occupational noise exposures will be managed in compliance Yukon regulations, as discussed previously.

For all areas adjacent to the proposed Mine Site (e.g. as represented by focal areas of interest SA-01 through -15), noise levels associated with construction or operational activities are expected to be lower than the existing measured background noise levels and thus will not be easily distinguishable from the background wilderness-type noise environment. In addition, noise at all modelled locations will be far lower than authoritative health effects thresholds relating to sleep disturbance or speech interference.

The highest predicted continuous sound level offsite was 28 dBA during peak operations at a location near the centre of the Yukon River, approximately 10 km downriver from the mouth of Coffee Creek (SA-07). It is conceivable that people will be able to faintly detect noise from mine operations when the ambient noise levels are very low (infrequent periods where sound levels are less than 25 dBA, for example, during low wind conditions and in the absence of wildlife or personal sounds). Mining related sound at SA-07 or closer to the mine noise sources, in the absence of natural topographic or vegetative sound barriers and attenuation, could conceivably be detected given the different frequency and tonal characteristics than natural sounds.

Nonetheless, the predicted Project-related noise levels are also far lower than levels associated with high annoyance in various European and United States epidemiological studies. This is illustrated in a meta-analysis of European annoyance and noise epidemiological studies, published by Van Gerven *et al*. (2009) (**Figure 3-3**). The significance of any relationship between high annoyance and day-night noise is uncertain for noise levels less than 45 dBA (Van Gerven *et al* 2009), based in part on high inter-individual variability, and in part based on a possible higher relative importance of background noise in comparison with transportation-related noise (for which noise and %HA associations have been developed). As illustrated in **Figure 3-3**, L_{DN} levels lower than around 50 dBA have not been associated with high annoyance in more than approximately 10% of the exposed population.

Overall, it is concluded that the peak construction or operations phase of the proposed Project are not likely to result in increases in noise relative to existing conditions that could approach or exceed risk-based thresholds for human health effects. The noise health risks associated with the Project, therefore, are considered to be acceptably low.

The noise predictions on which conclusions about noise health risks are based employ a number of simplifying assumptions that are likely to result in unrealistic predictions in comparison with the true case. Nonetheless, various aspects of the noise predictive modelling are likely to over-predict noise exposure potential, while the health effects thresholds discussed herein will generally tend to conservatively over-predict health effects for a given level of noise exposure. The major conclusions about noise health risks, therefore, are considered to have a high level of confidence.

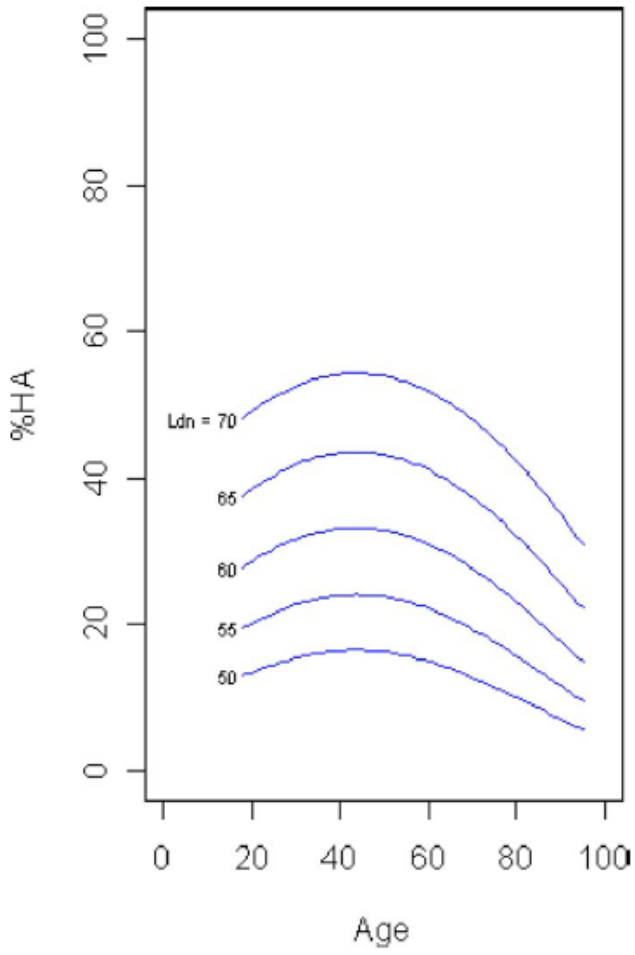

Figure 3-3 Predicted Percentage of Highly Annoyed Persons (%HA) as a Function of Age and Noise Level (Ldn) for Pooled European Data Sets (from Van Gerven et al., 2009)

3.5.2 MINE ACCESS ROAD

The predicted noise levels adjacent to the access road are illustrated in **Figure 3-2**. Peak noise on the roadway during a haul track pass-by could be in the range of 97 dBA at the point of origin, and is expected to decrease with lateral distance from the road centreline to approximately 47 dBA within 100 m of the centreline. Mine vehicles will only travel along the access road during daytime hours, and there will be no effects of traffic on sleep disturbance potential therefore. A speech interference threshold of 55 dBA (**Table 3-4**) might be exceeded during a haul truck pass-by at a distance of 35 m or less laterally from the centreline, and within this distance such influences are expected to be transient (≤ eight mine-related vehicle pass-bys per day).

The potential for increased incidence of high annoyance associated with the incremental Project-related noise is concluded to be negligible, given that traffic noise beyond approximately 0.5 km from the road will not be readily distinguishable from natural, ambient sound levels, there will be a peak noise level of 45 dBA or lower within 45 m of the centreline, and the noise source (vehicle pass-bys) will be infrequent and of short duration. Thus, there are unlikely to be chronic influences on hypertension based on human noise exposures.

Overall, no noise-related effects on human health are anticipated along areas adjacent to the mine access road during project operations.

4.0 AIR QUALITY HEALTH RISK ASSESSMENT

This section provides the details of the HHRA for air quality. The primary objective of HHRA is to evaluate whether contaminants associated with air emissions from Project activities could have an effect on human health.

Air quality predictions for the Project peak operational year are considered. Air dispersion modelling for the construction period was not completed (**Section 9.0 Air Quality and Greenhouse Gas Emissions Analysis**): A detailed evaluation of emissions sources based on the Project Description for each of construction and operations phases indicates that the potential for both dust generation / dust fall and emissions from internal combustion engines (or the incinerator) is far greater for the peak operational year than construction year.

The air quality predictions presented in **Section 9.0**, assume continuous operation of some of the emission sources over a 24-h period for some emissions sources, but not all, as summarized below.

Table 4-1 Modelled Air Emission Source Types for Peak Operational Year and Assumed Temporal Variations

Emission Source Grouping	CALPUFF Source Type	Temporal Variability (h)
Blasting	Volume	Twice weekly, occurring at 1400 h
Crusher	Volume	Operational April – December
Drilling	Volume	Assumed continuous and constant rate
Haul Roads	Road	Usage based on extraction and dumping tonnages
Material Handling	Volume	Varied based on extraction and dumping schedule
Overburden Removal	Volume	Varied based on grub removal schedule
Pit Exhaust	Volume	Varied based on extraction schedule
Power Plant	Point	Continuous
Incinerator	Point	Assumed operational between 1000and 1400 h
Haul Road Light Duty / Grader Exhaust	Volume	Continuous
Wind Erosion of Stockpiles	Volume	Varied with Wind Speed
Waste Rock Storage Facility Maintenance	Volume	Varied based on dumping schedule

Limited ambient air quality monitoring was completed in 2014-15 to determine existing air quality conditions, as discussed **in Section 9.0**. Measurements were made for $PM_{2.5}$, PM_{10}, TSP, and dust fall. The concentrations of other combustion-derived CACs including CO, NO_2 and SO_2 were assumed to be negligible given the remote and undeveloped nature of the LAA and RAA.

The observed winter-time levels of particulate matter (either as TSP, PM10 or PM2.5) were ≤4 µg/m^3. Summer-time concentrations for all fractions ranged from <1 µg/m^3 to as high as 200 µg/m^3, with the higher concentrations recorded having resulted from a forest fire in the region. The range of dust fall rates observed were from 0.12 to 0.27 mg/dm^2/day in the winter monitoring period and 0.12 to 0.71 mg/dm^2/day during summer monitoring periods.

Table 4-2 Airborne Particulates Measured at the Site in 2014-15

Parameter	Season	Minimum Ground-Level Concentration	Maximum Ground-Level Concentration	Average Ground-Level Concentration
Total Suspended Particulate Matter (TSP)	Winter	1 µg/m^3	65 µg/m^3	2 µg/m^3
	Summer	<1 µg/m^3	200 µg/m^3	41 µg/m^3
Particulate Matter <2.5 µm ($PM_{2.5}$)	Winter	1 µg/m^3	34 µg/m^3	2 µg/m^3
	Summer	<1 µg/m^3	177 g/m^3	40 µg/m^3

Given the low and expected ambient levels of CACs in the Project area, the predicted air quality and dust deposition rates associated with mining related emissions for the peak operational year were treated as negligible for the purposes of air quality predictions (**Section 9.0**). The mining associated levels of CACs were not added to levels arising from other sources regionally.

4.1 CONTAMINANTS OF POTENTIAL CONCERN

This HHRA considers the predicted acute (short-term, or ≤24 hours) and chronic (annual average) airborne concentrations of chemicals identified in emission sources related to the Project, both within the vicinity of the Mine Site and in areas adjacent to the mine access road.

All prioritized airborne contaminants associated with Project emissions and identified in the Air Quality Study (**Section 9.0**) were selected as COPC for the evaluation of human health risks. The exceptions were parameters related to climate change; i.e., greenhouse gases expressed as carbon dioxide equivalents, or climate forcing particulate matter.

The COPC selected for the evaluation of human health risks included CACs that are routinely assessed as priority contaminants in fuel combustion by-products; i.e.

- CO
- SO_2
- NO_2
- $PM_{2.5}$
- PM_{10}
- Total Suspended Particulates.

Yukon AAQO exist for these CACs, as discussed in **Section 4.4.**

Given the potential for dust generation in association with mining operations such as blasting, crushing, waste rock hauling and associated earth works, the prediction and management of dust is an important aspect of mine planning and management. Dust fall to areas within and surrounding the mine site was an important aspect of the air quality assessment for both the Mine Site and mine access road. Health risks associated with mining-related dust fall are assessed in **Section 5.0** herein **(Country Food Safety Risk Assessment)**.

The above-listed set of CACs does not include all priority contaminants from fuel combustion emissions: The US EPA (2007) has developed a prioritized list of mobile source air toxins (MSAT) emitted from gasoline and diesel combustion engines, based on their potential contributions to health risks as a result of breathing air toxins in outdoor air. The list of MSAT includes the following volatile organic compounds:

- Benzene
- 1,3-butadiene
- Formaldehyde
- Acetaldehyde
- Acrolein.

In addition, priority MSAT, as defined by the US EPA (2007) and California Department of Transportation (2014) include polycyclic organic matter, naphthalene, and diesel particulate matter.

While the above-listed individual Volatile Organic Compounds (VOCs) were not evaluated in the air quality modelling exercise directly, TVOC were modelled as a proxy for health risks associated with human inhalation exposures to these individual priority VOCs. Similarly, naphthalene and particulate organic matter were not evaluated directly in the air dispersion modelling; however, the dispersion of total polycyclic organic hydrocarbons (TPAHs) was completed based on estimates of TPAH in the emission sources and assuming that the broader range of TPAH exhibit physical-chemical properties the same as naphthalene.

Diesel particulate matter (DPM) was not included in air dispersion modelling as a contaminant of interest. Given that diesel fueled equipment and diesel power generation dominate the emission sources for fine particulate matter (as opposed to dust generation arising from the disturbance of soils and rocks), the predicted concentrations of DPM are expected to be similar to those of $PM_{2.5}$.

4.2 Focal Areas of Interest

Culturally important and sensitive receptor locations, collected termed focal areas of interest FAI where developed to inform the air quality HHRA, as discussed in **Section 3.2** and illustrated in Figure A for the noise HHRA. A common set of FAI was used for the noise HHRA and air quality HHRA.

4.3 Relevant Health Effects Thresholds

For the CACs, the Yukon AAQO were used as the basis for human health effect thresholds. According to Yukon Environment (2014):

> "The following standards are the maximum concentrations of pollutants acceptable in ambient air throughout the Yukon Territory. These Yukon ambient air quality standards will be used to determine the acceptability of emissions from proposed and existing developments."

Table 4-3 Yukon Ambient Air Quality Objectives

Parameter	Standard ($\mu g/m^3$)	Standard (ppm)	Standard (ppbv)
Sulphur Dioxide (SO_2)			
• 1-hour average	450[A]		172
• 24-hour average	160		57
• Annual arithmetic mean	25		11
Nitrogen Dioxide (NO_2)			
• 1-hour average	400[A]		213
• 24-hour average	200		106
• Annual arithmetic mean	60		32
Ground Level Ozone (O_3)			
• 8-hour running average			63
Carbon Monoxide (CO)			
• 1-hour average	14,300[A]	13	
• 8-hour average	5,500	5	
Fine Particulate Matter <2.5µm ($PM_{2.5}$)			
• 24-h average (calendar day)	28		
• Annual mean (calendar year)	10		
Coarse Particulate Matter <10µm (PM_{10})			
• 24-h average	50		
Total Suspended Particulate Matter (TSP)			
• 24-hour average	120		
• Annual geometric mean	60		

Note: [A] Values converted from ppbv to $\mu g/m^3$ assuming 25°C and 101.3 kiloPascals pressure

This air quality HHRA does not include predictions of exposures to ground level ozone (nor does the air quality study: **Section 10.0**). Ozone is produced through secondary atmospheric reactions particularly with (nitrogen oxides) NOx and VOCs in airsheds influenced, in particular, by fuel combustion emissions. For the purpose of this HHRA, it is assumed that health risks will be acceptably low from project-related ground level ozone production if the major precursor compounds are predicted to be below Yukon AAQO (for NO_2) and lower than thresholds of health effects via inhalation pathways (for various VOCs as discussed below).

Yukon AAQO do not exist for the individual MSAT listed above (benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, naphthalene, other PAHs including benzo[a]pyrene). For these airborne contaminants of potential concern, exposure limits recommended by toxicologists and epidemiologists from a range of provincial, federal and international regulatory agencies were reviewed for the identification of the most appropriate exposure limit for each COPC. These agencies include the following:

- Canadian Council of Ministers of the Environment (CCME)
- Health Canada (HC)
- World Health Organization (WHO)
- British Columbia Ministry of Environment (BC MOE)

- United States Environmental Protection Agency (US EPA)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- California Office of Environmental Health Hazard Assessment (OEHHA)
- Texas Commission on Environmental Quality (TCEQ)
- Netherlands National Institute of Public Health and the Environment.

Exposure limits specific to acute and chronic inhalation exposure scenarios were reviewed for each COPC. The following attributes were reviewed and summarised for each chemical and available exposure limit:

- Key toxicology (animal) or epidemiology (human) study
- Primary effect or chemical mode of action
- Point of departure or starting point for subsequent extrapolations and analyses (lowest effect dose that is adequately supported by dose-response data)
- Dosimetric adjustments for animal to human exposures, and
- Uncertainty factors.

In general, the most stringent exposure limit was used to determine the potential hazards associated with exposure to COPC in Project emissions to air, if the limit is adequately documented and scientifically defensible. The adopted exposure limits all include uncertainty factors that further reduce the presumed acceptable exposure limit for the protection of individuals who may be more sensitive to chemical exposure.

Threshold of health effect values used in this HHRA are summarized in **Table 4-4** for acute exposure periods and in **Table 4-5** for chronic exposures. The basis for selection of these values is discussed in more detail in **Appendix A: Toxicological Profiles for Contaminants of Potential Concern.**

Table 4-4 Summary of Acute Inhalation Exposure Limits for Mobile Source Air Toxins

Chemical	Averaging Time	Acute Inhalation Exposure Limit (µg/m^3)	Health Endpoint	Agency
Acetaldehyde	1 hour	470	Respiratory irritation	OEHHA
Acrolein	1 hour	2.5	Eye irritation	OEHHA
Benzene	1 hour	580	Immunological	TCEQ
1,3-Butadiene	1 hour	660	Developmental	OEHHA
	24 hour	15		US EPA
Formaldehyde	1 hour	50	Eye and nasal irritation	ATSDR
Naphthalene	1 hour	2,000	Eye and respiratory irritation	ACGIH

Table 4-5 Summary of Chronic Inhalation Exposure Limits for Mobile Source Air Toxins

Chemical	Averaging Time	Chronic Inhalation Exposure Limit (µg/m^3)	Health Endpoint	Agency
DPM	annual	5 0.03	Pulmonary inflammation Lung Cancer	US EPA OEHHA
Acetaldehyde	annual	390	Nasal irritation	Health Canada
		3.7	Nasal tumours	OEHHA
Acrolein	annual	2.7	Nasal irritation	TCEQ
Benzene	annual	9.8	Immunological / hematological	ATSDR
		1.3	Leukemia	US EPA
Benzo(a)pyrene (PAH group)	annual	0.00012	Lung Cancer	WHO
1,3-Butadiene	annual	2 0.3	Ovarian atrophy Leukemia	US EPA
Formaldehyde	annual	9	Eye, nasal, respiratory irritation	OEHHA
		2	Nasal tumors	Health Canada
Naphthalene	annual	3	Nasal irritation	US EPA
		0.3	Nasal tumours	OEHHA

4.3.1 WORKER EXPOSURES

The air quality predictions include areas adjacent to the Mine Site and access road, for which this HHRA considers exposures of various members of the public, as well as on-site areas. As discussed above, protection of worker safety is a statutory requirement under the Yukon Occupational Safety and Health Act. As such, the protection of worker health on site is a mandatory and legal requirement for the Coffee Project, and therefore not a focus of this Project Proposal health effects assessment, which addresses public health. Nonetheless, the occupational exposure limits for COPC are discussed here for informational purposes especially in light of on-site information on air quality as presented in **Section 9.0.**

Occupational exposure limits ("stated permissible concentrations" for various combustion by-products are formally defined within the *Yukon Occupational Health Regulations*, Section 27. AIR CONTAMINANTS, and in Tables 7 through 14. The Maximum Permissible Concentrations (MPCs) listed in the regulation assume an 8 h exposure period while working. If a worker were to stay on-site and specifically in camp throughout an entire 24-h day, the MPCs for systemic toxicants (those that increase in concentration in the internal body in proportion to the uninterrupted exposure duration) would be 3-fold lower than listed in the Regulations. Note that such an adjustment applies only to substances listed in Tables 7 and 14 of the Regulations (metals/metalloids, fluoride and CO from Table 7; carcinogens from Table 14).

	15-minute	8-hour
CACs		
• Dust	3,000 µg/m^3	1,000 µg/m^3
• CO	440,000 µg/m^3	55,000 µg/m^3
• NO_2	no value	9,000 µg/m^3
• SO_2	13,000 µg/m^3	13,000 µg/m^3
VOCs and PAHs		
• Acetaldehyde	270,000 µg/m^3	180,000 µg/m^3
• Acrolein	800 µg/m^3	250 µg/m^3
• Benzene	no value	32,000 µg/m^3
• 1,3-butadiene	2,750,000 µg/m^3	2,200,000 µg/m^3
• Formaldehyde	no value	3,000 µg/m^3
• Naphthalene	75,000 µg/m^3	50,000 µg/m^3

4.4 AIR QUALITY EXPOSURE ASSESSMENT

The predicted concentrations of CACs during the peak operational year, at breathing height, are provided in **Table 4-6**, **Table 4-7** and **Table 4-8**. In all cases, the predictions of airborne concentrations assume the implementation of dust suppression for mine area during the six months of spring and summer (April through October) but not between November 1st and March 31st, since the land is likely to be either substantially covered by snow and ice during this period, or generally wet.

Reichle *et al.* (2015) provided estimates of the composition by percent mass in total organic gas emissions of individual compounds from non-road diesel gas (compression ignition) vehicles. The speciation profiles reported therein assist with the apportionment of the total VOC data for air quality predictions into the individual MSAT contaminants of interest (**Table 4-9**). Only those compounds present at greater than 1% by mass in the total emitted organic compounds are listed, with the exception of naphthalene. VOCs and PAHs defined as contaminants of potential concern for this air quality HHRA are shown in the table as bolded entries.

The percent compositional data as presented in **Table 4-9** were used to speciate the VOC air concentration data at each focal area of interest for the purpose of estimating potential human exposures during the peak operational year. Key assumptions are that: (i) the compositional estimates by Reichle et al (2015) adequately describe the composition of individual compounds for the various emissions sources operating at the mine and along the NAR; and (ii) the composition does not change substantially (for example, through secondary reactions and photodegradation in the air) during the time it takes for the affected air mass to travel from the point of emissions to the point of human exposure.

There are a large number of scientific studies of the emission rates and composition of PAHs in gasoline and diesel engine exhausts. A summary of the PAH compositional profile from two recent studies (Mi *et al.,* 2000; Huang *et al.* 2013) is provided in **Table 4-10**. Based on these published studies, it was conservatively assumed that naphthalene comprises 80% of predicted TPAH concentrations, and benzo[a]pyrene comprises up to 2% of the TPAH concentration.

Table 4-6 Predicted Concentrations (µg/m^3) within the Breathing Zone at Focal Areas of Interest of Carbon Monoxide, Nitrogen Dioxide, and Sulphur Dioxide – Peak Operating Year (values that exceed their respective Yukon AAQO are highlighted in orange)

	CO			NO_2			SO_2		
	1-h Avg (Max)	8-h Avg (Max	Annual Average	1-h Avg (Max)	24-h Avg (Max	Annual Average	1-h Avg (Max)	24-h Avg (max	Annual Average
Yukon AAQO All values in µg/m^3	**14,300**	**5,500**		**400**	**200**	**60**	**450**	**160**	**25**
CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and **CO-02:** Mine Dry and Office Complex	955	1146	12.6	2653	365	33	24	20	0.17
CO-03: Assay Lab	686	724	8.6	1248	265	26	17	20	0.12
CO-04: Truck shop / warehouse	718	733	9.5	1360	300	24	20	20.1	0.20
CO-05: near airstrip	344	996	1.2	253	32	2.3	29	1.7	0.025
SA-01: Coffee Cr.	194	407	0.31	120	11	0.40	7.3	0.46	0.0072
SA-01a: Coffee Cr. Mouth (Same as VM4)	230	387	0.31	115	10	0.39	6.9	0.44	0.0071
SA-02: Yukon River - centre channel, ~1. km upriver from Coffee Creek mouth	125	300	0.26	91	9	0.34	5.3	0.37	0.0057
SA-03: Yukon River - centre channel, ~1.5 km downriver from Coffee Creek mouth	1333	700	0.7	394	24	0.79	19	0.92	0.017
SA-04: Yukon River - centre channel, ~3 km downriver from Coffee Creek mouth	766	505	0.5	236	15	0.58	14	0.65	0.012
SA-05: Yukon River - centre channel, ~5 km downriver from Coffee Creek mouth	477	469	0.4	152	12	0.47	9.0	0.53	0.0092
SA-06: Yukon River - centre channel, ~7 km downriver from Coffee Creek mouth	1766	1649	1.1	**493**	34	1.0	22	1.5	0.021
SA-07: Yukon River - centre channel, ~10 km downriver from Coffee Creek mouth	247	1306	0.77	359	27	1.2	16	1.3	0.02
SA-08: Confluence of Latte and Coffee Creeks	225	533	0.38	144	14	0.64	10	0.59	0.0096

	CO			NO_2			SO_2		
	1-h Avg (Max)	8-h Avg (Max	Annual Average	1-h Avg (Max)	24-h Avg (Max	Annual Average	1-h Avg (Max)	24-h Avg (max	Annual Average
SA-09: Height of land across Yukon R. from Coffee Creek	114	219	0.23	69	8	0.30	3.8	0.31	0.0049
SA-10: Height of land across Yukon R. from proposed Mine Site	571	380	0.40	175	11	0.49	11	0.52	0.0096
SA-11: Height of land across Yukon R. to ea.	206	744	0.50	190	16	0.76	10	0.85	0.013
SA-12: Ballarat Creek Area, N. of Yukon River	112	183	0.18	56	6	0.2	2.7	0.21	0.0036
SA-13: Yukon River foreshore east of existing Coffee Creek camp	632	524	0.46	198	14	0.55	12	0.64	0.011
SA-14: Wilderness Retreat, on Yukon River	151	312	0.69	102	18	1.0	5.2	1.5	0.011
SA-15: Representative harvesting area - height of land	50	309	0.39	90	13	0.70	3.3	0.45	0.0069

Table 4-7 Predicted Concentrations (µg/m^3) of Suspended Particulates within the Breathing Zone at Focal Areas of Interest – Peak Operating Year (values that exceed their respective Yukon AAQO are highlighted in orange)

	$PM_{2.5}$		PM_{10}	TSP	
	1-h Avg (max)	8-h Avg (max	Annual Average	1-h Avg (max)	24-h Avg (max
Yukon AAQO All values in µg/m^3	**28**	**10**	**50**	**120**	**60**
CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and **CO-02:** Mine Dry and Office Complex	121	12.2	821	2141	218
CO-03: Assay Lab	71	6.7	454	1005	94
CO-04: Truck shop / warehouse	93	8.2	602	1468	123
CO-05: Near airstrip	11	0.76	45	80	6
SA-01: Coffee Cr.	3.1	0.16	7.0	11	0.67
SA-01a: Coffee Cr. Mouth (Same as VM4)	3.1	0.16	7.0	10	0.65
SA-02: Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth	2.7	0.14	6.4	8.8	0.58
SA-03: Yukon River – centre channel, ~1.5 km downriver from Coffee Creek mouth	6.6	0.29	14	20	1.3
SA-04: Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth	4.8	0.22	11	14	0.93

	$PM_{2.5}$		PM_{10}	TSP	
	1-h Avg (max)	8-h Avg (max	Annual Average	1-h Avg (max)	24-h Avg (max
SA-05: Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth	3.8	0.18	8.5	11	0.76
SA-06: Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth	7.2	0.37	16	23	1.8
SA-07: Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth	6.5	0.45	21	34	2.5
SA-08: Confluence of Latte and Coffee Creeks	5.9	0.25	18	28	1.2
SA-09: Height of land across Yukon R. from Coffee Creek	2.3	0.12	5.5	6.7	0.50
SA-10: Height of land across Yukon R. from proposed Mine Site	3.7	0.20	10	18	0.83
SA-11: Height of land across Yukon R. to ea.	3.1	0.29	11	14	1.4
SA-12: Ballarat Creek Area, N. of Yukon River	1.8	0.10	4.2	4.6	0.38
SA-13: Yukon River foreshore east of existing Coffee Creek camp	4.5	0.21	10	13	0.90
SA-14: Wilderness Retreat, on Yukon River	4.5	0.41	14	21	2.4
SA-15: Representative harvesting area – height of land	1.9	0.28	6.3	11	1.6

Table 4-8 Predicted Concentrations (µg/m^3) of Total Volatile Organic Contaminants (TVOC) and Total PAHs (TPAH) within the Breathing Zone at Focal Areas of Interest – Peak Operating Year

	TVOC		TPAH	
	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average
CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and **CO-02:** Mine Dry and Office Complex	125	1.9	4.6E-02	7.2E-04
CO-03: Assay Lab	63	1.2	2.9E-02	8.2E-04
CO-04: Truck shop / warehouse	72	1.4	2.5E-02	5.6E-04
CO-05: near airstrip	11	0.150	2.2E-03	3.2E-05
SA-01: Coffee Cr.	2.0	0.017	4.6E-04	5.1E-06
SA-01a: Coffee Cr. Mouth (Same as VM4)	2.0	0.017	4.5E-04	5.0E-06
SA-02: Yukon River - centre channel, ~1.5 km upriver from Coffee Creek mouth	1.6	0.015	4.0E-04	4.4E-06
SA-03: Yukon River - centre channel, ~1.5 km downriver from Coffee Creek mouth	4.9	0.034	1.2E-03	9.5E-06
SA-04: Yukon River - centre channel, ~3 km downriver from Coffee Creek mouth	3.4	0.025	8.3E-04	7.2E-06

	TVOC		TPAH	
	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average
SA-05: Yukon River - centre channel, ~5 km downriver from Coffee Creek mouth	2.4	0.020	6.2E-04	5.9E-06
SA-06: Yukon River - centre channel, ~7 km downriver from Coffee Creek mouth	5.4	0.045	1.2E-03	1.2E-05
SA-07: Yukon River - centre channel, ~10 km downriver from Coffee Creek mouth	4.5	0.059	1.0E-03	1.6E-05
SA-08: Confluence of Latte and Coffee Creeks	1.8	0.029	6.7E-04	8.2E-06
SA-09: Height of land across Yukon R. from Coffee Creek	1.2	0.013	3.3E-04	3.9E-06
SA-10: Height of land across Yukon R. from proposed Mine Site	2.6	0.021	5.6E-04	6.5E-06
SA-11: Height of land across Yukon R. to ea.	2.2	0.035	6.1E-04	9.8E-06
SA-12: Ballarat Creek Area, N. of Yukon River	0.73	0.010	2.3E-04	3.0E-06
SA-13: Yukon River foreshore east of existing Coffee Creek camp	3.0	0.024	7.4E-04	6.9E-06
SA-14: Wilderness Retreat, on Yukon River	2.2	0.054	7.6E-04	1.5E-05
SA-15: Representative harvesting area - height of land	1.1	0.037	3.2E-04	1.1E-05

Table 4-9 Representative Compositional Profiles of Individual Compounds in Nonroad Diesel Equipment Emissions – Percent by Mass of Total Organic Gas Emissions (priority mobile source air toxics are listed in bold font)

CAS Number	Compound	Diesel Equipment Class		
		Pre-Tier 1	Tier 1	Tier 2
50-00-0	formaldehyde	19.9	20.4	26.6
74-85-1	ethylene	16.7	18.9	18.4
75-07-0	acetaldehyde	7.18	7.14	9.51
74-82-8	methane	1.74	7.09	8.28
	unidentified C9-C12+	5.00	0.70	6.08
71-43-2	benzene	1.88	1.97	5.07
108-88-3	toluene	1.17	1.97	3.43
4170-30-3	crotonaldehyde	1.94	3.85	3.16
123-38-6	propionaldehyde	1.39	3.55	1.99
107-02-8	acrolein	2.92	1.47	1.70
109-66-0	pentane	0.12	0.68	1.56
74-86-2	acetylene	2.91	3.41	1.12
1330-20-7	m- and p-xylene	1.48	1.09	1.07
106-99-0	1,3-butadiene	0.19	0.19	0.19
91-20-3	naphthalene	0.05	0.05	0.00

Table 4-10 PAH Composition in Diesel Exhaust

Aromatic Hydrocarbon	Mi *et al.*, 2000 N=5 operating modes		Huang et al., 2013		
	Average	St. Dev	Idle	Low Load	High Load
Naphthalene	80%	5.1%	78%	71.3%	64.1%
Acenaphthylene	4.6%	1.9%	5.6%	8.4%	6.3%
Acenaphthene	3.3%	0.75%	6.9%	8.4%	12.3%
Fluorene	4.7%	2.7%	Not analyzed	Not analyzed	Not analyzed
Anthracene	3.1%	1.7%	Not detected	Not detected	0.3%
Phenanthrene	0.62%	0.64%	Not detected	Not detected	0.1%
Fluoranthene	0.64%	0.44%	Not detected	Not detected	Not detected
Pyrene	0.62%	0.41%	9.9%	Not detected	13.9%
Benzo[a]anthracene	0.10%	0.04%	Not detected	Not detected	0.7%
Chrysene	0.21%	0.07%	Not detected	Not detected	0.6%
Benzo[b]fluoranthene	0.23%	0.16%	Not detected	Not detected	0.1%
Benzo[k]fluoranthene	0.15%	0.05%	Not detected	Not detected	Not detected
Benzo[a]pyrene	0.20%	0.13%	Not detected	Not detected	1.6%
Indeno[1,2,3-c,d]pyrene	0.52%	0.19%	Not detected	Not detected	Not detected
Dibenzo[a,h]anthracene	0.32%	0.34%	Not detected	Not detected	Not detected

4.5 RISK CHARACTERIZATION AND UNCERTAINTY

Risk characterization involved the comparison of predicted exposure concentrations to the exposure limit for each chemical identified in Project emissions. In the case of acute and chronic inhalation exposure to chemicals in air, potential exposures were described as air concentrations (i.e., µg chemical/m^3 air). The predicted exposures documented in **Section 4.4** were compared to exposure limits (**Appendix A**) defined in terms of acceptable air concentrations or dose rates.

A Risk Quotient (RQ) provides a direct comparison of predicted chemical exposure to a chemical exposure limit. Although a quantitative number, the RQ is inherently uncertain as it is based on predicted and uncertain estimates of exposure and toxicity estimates. Conservative assumptions are made throughout the risk assessment process to address these uncertainties and not underestimate potential human health risks. The exposure assessment, in particular, is based on point estimates using the highest predicted (modelled) air concentrations, which assumed “worst-case” air emission and dispersion conditions.

These conservative assumptions preclude the treatment of the RQ value as a numerical measurement of health risk. Rather, the RQ value is useful as a screening tool to determine whether, having evaluated a worst-case scenario, refinement of the assumptions of chemical exposure or the toxicity, is required.

4.5.1 RISK QUOTIENTS FOR THRESHOLD CHEMICALS

For non-carcinogenic chemicals, inhalation exposure was defined by the sum of ambient (measured) air concentrations plus predicted air concentrations for the Existing, Future with Project, or Future without Project emissions scenarios. Predicted exposures were compared to a threshold exposure limit expressed as reference concentration. The following is an example of an RQ equation for a threshold chemical:

$$\text{Risk Quotient (RQ)} = \frac{\text{Ambient air concentration during peak operational year } (\mu g/m^3)}{\text{Reference Concentration } (\mu g/m^3)}$$

An RQ value less than or equal to 1 (RQ $\leq$ 1.0) indicates that the total predicted exposure (considering various emission sources plus ambient levels) is at or below the recommended safe exposure limit for a non-carcinogen (AHW 2011).

An RQ value greater than unity, or one (i.e., >1.0) does not indicate that adverse health effects are expected to occur, considering the inherent conservatism in the risk assessment process. Rather, an RQ > 1 triggers the need for additional discussion of the significance of the estimated risk within the context of the exposure and toxicity assumptions made in the HHRA (AHW 2011).

4.5.2 RISK QUOTIENTS FOR NON-THRESHOLD CHEMICALS

For carcinogenic chemicals such as benzene or benzo[a]pyrene, the incremental lifetime cancer risks specific to Project emissions are estimated from predicted exposure concentrations, as follows:

$$\text{Risk Quotient (RQ)} = \frac{\text{Project-only Air Concentration } (\mu g/m^3)}{\text{Risk Specific Concentration } (\mu g/m^3)}$$

An RQ value less than or equal to 1 (RQ $\leq$ 1.0) indicates that the incremental increase in lifetime cancer risk as a result of Project emissions is within an acceptable range (i.e., at or below 1 in 100,000). Again, an RQ > 1 requires further evaluation of the exposure and toxicity assumptions to determine the significance of the estimated risk (AHW 2011).

4.5.3 EXPOSURE TO CRITERIA AIR CONTAMINANTS DURING THE PEAK OPERATING YEAR

Risk quotients for on-site and off-site exposures were estimated for exposures to CACs via inhalation by dividing the predicted short (acute) or annual average (chronic) airborne CAC concentration (**Table 4-6** and **Table 4-7**) by the relevant Yukon AAQO (**Table 4-3**). The resulting risk quotients are presented in **Table 4-11**.

The highest risk quotient for carbon monoxide, either on- or off-site was 0.21 and no health risks from this CAC are anticipated as a result of the Project.

For NO_2, the risk quotients for acute exposures (1-hour, 24-hour) exceeded 1.0 at focal areas of interest CO-01/02, C0-03 and CO-04. These areas are located within the working Mine Site, and if the exposure concentrations of NO_2 are compared with the *Yukon Occupational Health Regulations* maximum permissible levels (8-h = 9,000 µg/m^3) (**Section 4.3.1)**, then the risk quotients are as shown below:

	RQ for 1-h avg (max) NO_2	RQ for 24-h avg (max) NO_2
• CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and CO-02: Mine Dry and Office Complex	0.29	0.041
• CO-03: Assay Lab	0.14	0.029
• CO-04: Truck shop / warehouse	0.15	0.033
• CO-05: near airstrip	0.028	0.0036

An RQ >1.0 was calculated for the maximum observed 1-hour average NO_2 concentration at location SA-06, a site on the Yukon River, near its centre, approximately 6 km downriver from the mouth of Coffee.

Table 4-11 Risk Quotients for Predicted Exposures to CACs During Mine Operations – CO, NO_2, SO_2 (values that exceed a value of one are highlighted in orange)

	CO		NO_2			SO_2		
	1-h Avg (Max)	8-h Avg (Max)	1-h Avg (Max)	24-h Avg (Max)	Annual Average	1-h Avg (Max)	24-h Avg (Max)	Annual Average
CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and **CO-02:** Mine Dry and Office Complex	0.067	0.21	6.6	1.8	0.55	0.053	0.13	0.0068
CO-03: Assay Lab	0.048	0.13	3.1	1.3	0.43	0.038	0.13	0.0048
CO-04: Truck shop / warehouse	0.050	0.13	3.4	1.5	0.40	0.044	0.13	0.0080
CO-05: Near airstrip	0.024	0.18	0.63	0.16	0.038	0.064	0.011	0.0010
SA-01: Coffee Cr.	0.014	0.074	0.30	0.055	0.007	0.016	0.0029	0.00029
SA-01a: Coffee Cr. Mouth (Same as VM4)	0.016	0.070	0.29	0.050	0.007	0.015	0.0028	0.00028
SA-02: Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth	0.008	0.055	0.23	0.045	0.0057	0.012	0.0023	0.00023
SA-03: Yukon River – centre channel, ~1.5 km downriver from Coffee Creek mouth	0.093	0.13	**1.0**	0.12	0.013	0.042	0.0058	0.00068
SA-04: Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth	0.054	0.092	0.59	0.075	0.010	0.031	0.0041	0.00048
SA-05: Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth	0.033	0.085	0.38	0.060	0.0078	0.020	0.0033	0.00037
SA-06: Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth	0.123	0.30	**1.2**	0.17	0.017	0.049	0.0094	0.00084
SA-07: Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth	0.017	0.237	0.90	0.14	0.020	0.036	0.0081	0.00080
SA-08: Confluence of Latte and Coffee Creeks	0.016	0.097	0.36	0.070	0.011	0.022	0.0037	0.00038
SA-09: Height of land across Yukon R. from Coffee Creek	0.008	0.040	0.17	0.040	0.0050	0.008	0.0019	0.00020
SA-10: Height of land across Yukon R. from proposed Mine Site	0.040	0.069	0.44	0.055	0.0082	0.024	0.0033	0.00038
SA-11: Height of land across Yukon R. to ea.	0.014	0.135	0.48	0.080	0.013	0.022	0.0053	0.00052
SA-12: Ballarat Creek Area, N. of Yukon River	0.008	0.033	0.14	0.030	0.003	0.006	0.0013	0.00014
SA-13: Yukon River foreshore east of existing Coffee Creek camp	0.044	0.095	0.50	0.070	0.0092	0.027	0.0040	0.00044
SA-14: Wilderness Retreat, on Yukon River	0.011	0.057	0.26	0.090	0.017	0.012	0.0094	0.00044
SA-15: Representative harvesting area - height of land	0.004	0.056	0.23	0.065	0.012	0.007	0.0028	0.00028

Table 4-12 Risk Quotients for Predicted Exposures to CACs During Mine Operations – $PM_{2.5}$, PM_{10}, TSP (values that exceed a value of one are highlighted in orange)

		$PM_{2.5}$		PM_{10}	TSP	
		24-h Avg (Max)	Annual Average	24-h Avg (Max)	24-h Avg (Max)	Annual Average
CO-01: **CO-02:**	Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and Mine Dry and Office Complex	4.3	1.2	16	18	3.6
CO-03:	Assay Lab	2.5	0.67	9.1	8.4	1.6
CO-04:	Truck shop / warehouse	3.3	0.82	12	12	2.1
CO-05:	Near airstrip	0.39	0.076	0.90	0.67	0.10
SA-01:	Coffee Cr.	0.11	0.016	0.14	0.092	0.011
SA-01a:	Coffee Cr. Mouth (Same as VM4)	0.11	0.016	0.14	0.083	0.011
SA-02:	Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth	0.10	0.014	0.13	0.073	0.010
SA-03:	Yukon River – centre channel, ~1.5 km downriver from Coffee Creek mouth	0.24	0.029	0.28	0.17	0.022
SA-04:	Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth	0.17	0.022	0.22	0.12	0.016
SA-05:	Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth	0.14	0.018	0.17	0.09	0.013
SA-06:	Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth	0.26	0.037	0.32	0.19	0.030
SA-07:	Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth	0.23	0.045	0.42	0.28	0.042
SA-08:	Confluence of Latte and Coffee Creeks	0.21	0.025	0.36	0.23	0.020
SA-09:	Height of land across Yukon R. from Coffee Creek	0.08	0.012	0.11	0.06	0.008
SA-10:	Height of land across Yukon R. from proposed Mine Site	0.13	0.020	0.20	0.15	0.014
SA-11:	Height of land across Yukon R. to ea.	0.11	0.029	0.22	0.12	0.023
SA-12:	Ballarat Creek Area, N. of Yukon River	0.06	0.010	0.08	0.04	0.006
SA-13:	Yukon River foreshore east of existing Coffee Creek camp	0.16	0.021	0.20	0.11	0.015
SA-14:	Wilderness Retreat, on Yukon River	0.16	0.041	0.28	0.18	0.040
SA-15:	Representative harvesting area – height of land	0.07	0.028	0.13	0.09	0.027

A maximum 1-hour average concentration of NO_2 greater than the Yukon AAQO of 400 µg/m^3 was approached for SA-03 and SA-07 as well (both also on the river near the proposed Mine Site). The 99th percentile concentration at SA-06, however, was only 15 µg/m^3: a value that is more than an order of magnitude lower than the 1-hour Yukon AAQO for NO_2. This, along with the fact that NO_2 concentrations have been over-estimated in the air quality dispersion modelling based on an assumption that all NO_x is NO_2, suggests that health risks are acceptably low based on acute exposure potential.

Yukon AAQO for PM2.5 and TSP are predicted to be exceeded based on the maximum 24-h concentrations and annual average concentrations for the peak operational year for locations CO-01/02, CO-03 and CO-04, thus resulting in risk quotients greater than 1.0 (**Table 4-12**). As for NO_2, however, the maximum observed concentrations of airborne particulates, measured as either of the three size fractions, did not exceed the *Yukon Occupational Health Regulations* maximum permissible levels for dust of 3,000 µg/m^3 (15 minutes) or 9,000 µg/m^3 (8-hours) (**Section 4.4.1**).This comparison should be made with caution, however, since the maximum permissible levels may not adequately account for health effects especially associated with the finest fractions of airborne particulates, as captured by $PM_{2.5}$ estimates, and the averaging times are different (i.e. 15-minutes and 8-hours for the maximum permissible levels; 24-hours and annual averages for the Yukon AAQO particulate fractions). While occupational health risks are managed from a regulatory perspective as total dust, the predicted outdoor concentrations of fine particulate matter (2.5 µm in diameter) at Mine Site areas frequented by workers either on- or off-shift will require more careful consideration during mine operations.

As summarized in **Table 4-13**, exposures to any of the individual volatile organic contaminants at the focal areas of interest are not predicted to exceed health-based exposure thresholds, either for acute or chronic exposure scenarios.

Finally, the predicted annual average concentrations of benzo[a]pyrene, as representative potentially carcinogenic PAH that often occurs in combustion emissions, can be estimated from the range of concentrations of airborne TPAH at the focal areas of interest **(3.0×10^{-6} µg/m^3 to 8.2×10^{-4} µg/m^3 : Table 4-8**) and the expected percent contribution of benz[a]pyrene to TPAH in mining related emissions sources (i.e. up to 2% as discussed in **Section 4.4**). The estimated range of observed concentrations from these data is **6.0×10^{-8} µg/m^3 to 1.6×10^{-5} µg/m^3**.

As presented in **Table 4-13,** the lowest credible chronic exposure threshold for benzo[a]pyrene, based on incidence of lung cancer, is **1.2×10^{-4} µg/m^3**. Applying this toxicity reference value to the estimated range of concentrations of benzo[a]pyrene as discussed above results in calculated \RQ)\ for the focal areas of interest in the range of 5×10^{-4} to 1.4×10^{-1} (assay lab, outdoors).

These RQ values are all much lower than 1.0, therefore cancer risks from benzo[a]pyrene inhalation in association with mine operational emissions sources are predicted to be acceptably low.

Table 4-13 Risk Quotients for Predicted Exposures to Volatile Organic Contaminants During Peak Operations

	Formalehyde		Acetaldehyde		Benzene		Acrolein		1,3-butadiene		Naphthalene	
	1 h Avg. (Max.)	Annual Average	1 h Avg. (Aax. Value)	Annual Average	1 h avg. (max. value)	Annual Average	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average
CO-01: Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and **CO-02:** Mine Dry and Office Complex	6.7E-01	2.5E-01	2.5E-02	4.9E-02	1.1E-02	7.4E-02	1.5E+00	2.1E-02	1.6E-02	1.2E-02	3.1E-05	3.2E-03
CO-03: Assay Lab	3.4E-01	1.6E-01	1.3E-02	3.1E-02	5.5E-03	4.7E-02	7.4E-01	1.3E-02	8.0E-03	7.6E-03	1.6E-05	2.0E-03
CO-04: Truck shop / warehouse	3.8E-01	1.9E-01	1.5E-02	3.6E-02	6.3E-03	5.5E-02	8.4E-01	1.5E-02	9.1E-03	8.9E-03	1.8E-05	2.3E-03
CO-05: Near airstrip	5.9E-02	2.0E-02	2.2E-03	3.9E-03	9.6E-04	5.9E-03	1.3E-01	1.6E-03	1.4E-03	9.5E-04	2.8E-06	2.5E-04
SA-01: Coffee Cr.	1.1E-02	2.3E-03	4.0E-04	4.4E-04	1.7E-04	6.6E-04	2.3E-02	1.8E-04	2.5E-04	1.1E-04	5.0E-07	2.8E-05
SA-01a: Coffee Cr. Mouth (Same as VM4)	1.1E-02	2.3E-03	4.0E-04	4.4E-04	1.7E-04	6.6E-04	2.3E-02	1.8E-04	2.5E-04	1.1E-04	5.0E-07	2.8E-05
SA-02: Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth	8.5E-03	2.0E-03	3.2E-04	3.9E-04	1.4E-04	5.9E-04	1.9E-02	1.6E-04	2.0E-04	9.5E-05	4.0E-07	2.5E-05
SA-03: Yukon River – centre channel, ~1.5 km downriver from Coffee Creek mouth	2.6E-02	4.5E-03	9.9E-04	8.7E-04	4.3E-04	1.3E-03	5.7E-02	3.7E-04	6.2E-04	2.2E-04	1.2E-06	5.7E-05
SA-04: Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth	1.8E-02	3.3E-03	6.9E-04	6.4E-04	3.0E-04	9.8E-04	4.0E-02	2.7E-04	4.3E-04	1.6E-04	8.5E-07	4.2E-05
SA-05: Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth	1.3E-02	2.7E-03	4.9E-04	5.1E-04	2.1E-04	7.8E-04	2.8E-02	2.2E-04	3.0E-04	1.3E-04	6.0E-07	3.3E-05
SA-06: Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth	2.9E-02	6.0E-03	1.1E-03	1.2E-03	4.7E-04	1.8E-03	6.3E-02	4.9E-04	6.8E-04	2.9E-04	1.4E-06	7.5E-05
SA-07: Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth	2.4E-02	7.8E-03	9.1E-04	1.5E-03	3.9E-04	2.3E-03	5.3E-02	6.4E-04	5.7E-04	3.7E-04	1.1E-06	9.8E-05

	Formalehyde		Acetaldehyde		Benzene		Acrolein		1,3-butadiene		Naphthalene	
	1 h Avg. (Max.)	Annual Average	1 h Avg. (Aax. Value)	Annual Average	1 h avg. (max. value)	Annual Average	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average	1 h Avg. (Max. Value)	Annual Average
SA-08: Confluence of Latte and Coffee Creeks	9.6E-03	3.9E-03	3.6E-04	7.5E-04	1.6E-04	1.1E-03	2.1E-02	3.1E-04	2.3E-04	1.8E-04	4.5E-07	4.8E-05
SA-09: Height of land across Yukon R. from Coffee Creek	6.4E-03	1.7E-03	2.4E-04	3.3E-04	1.0E-04	5.1E-04	1.4E-02	1.4E-04	1.5E-04	8.2E-05	3.0E-07	2.2E-05
SA-10: Height of land across Yukon R. from proposed Mine Site	1.4E-02	2.8E-03	5.3E-04	5.4E-04	2.3E-04	8.2E-04	3.0E-02	2.3E-04	3.3E-04	1.3E-04	6.5E-07	3.5E-05
SA-11: Height of land across Yukon R. to ea.	1.2E-02	4.7E-03	4.5E-04	9.0E-04	1.9E-04	1.4E-03	2.6E-02	3.8E-04	2.8E-04	2.2E-04	5.5E-07	5.8E-05
SA-12: Ballarat Creek Area, N. of Yukon River	3.9E-03	1.3E-03	1.5E-04	2.6E-04	6.4E-05	3.9E-04	8.5E-03	1.1E-04	9.2E-05	6.3E-05	1.8E-07	1.7E-05
SA-13: Yukon River foreshore east of existing Coffee Creek camp	1.6E-02	3.2E-03	6.1E-04	6.2E-04	2.6E-04	9.4E-04	3.5E-02	2.6E-04	3.8E-04	1.5E-04	7.5E-07	4.0E-05
SA-14: Wilderness Retreat, on Yukon River	1.2E-02	7.2E-03	4.5E-04	1.4E-03	1.9E-04	2.1E-03	2.6E-02	5.8E-04	2.8E-04	3.4E-04	5.5E-07	9.0E-05
SA-15: Representative harvesting area – height of land	5.9E-03	4.9E-03	2.2E-04	9.5E-04	9.6E-05	1.4E-03	1.3E-02	4.0E-04	1.4E-04	2.3E-04	2.8E-07	6.2E-05

Overall, the examination of predicted airborne concentrations of CACs, VOCs and TPAH in off-site areas in comparison with relevant acute and chronic inhalation toxicity reference values indicates that human health risks associated with air quality during the peak operational period are acceptably low.

The maximum shorter duration concentrations of NO_2 and both shorter term (24-hour) and annual average concentrations of airborne particulates ($PM_{2.5}$, TSP) are predicted to exceed their respective Yukon AAQO outdoors in the area of the mine camp / dining hall / recreational complex, mine dry / office complex, assay lab and truck shop warehouse. The predicted concentrations, however, will not exceed maximum permissible levels for NO_2 or dust as defined in the *Yukon Occupational Health Regulations.*

5.0 TRADITIONAL AND NON-TRADITIONAL FOOD GATHERING RISK ASSESSMENT

The overall purpose of this country food safety risk assessment is to provide a good understanding of the potential health risks associated with the influence of the Coffee Gold mine on desired human ingestion of plants, birds and mammals that ingest the affected plants, and of fish. This is particularly important in light of the traditional and ongoing importance of local and regional country food and medicinal plant resources to First Nations people and others.

Changes in soil quality from mining-related dust deposition - and the associated direct deposition of dust to plant surfaces - could result in increased human exposures to those trace elements that occur at much higher concentrations in mining-related dust than in the existing forested area surface soils in general. In addition, areas with altered soil quality or water quality could result in altered trace metal uptake into edible forest and stream resources. Finally, mining-related influences on surface waters are of interest in terms of uptake of various trace elements into edible fish tissue. While the focus herein is on the evaluation of potential for contaminant uptake via dietary intake, we also assess the exposures and risks for people while hunting and gathering in mining affected areas through direct exposures from incidental soil ingestion.

It is important that people who value the use of traditional and non-traditional food and medicinal resources have adequate confidence in the integrity of those resources within potential harvesting areas near a mine site or other industrial activity. Perceptions of contamination in relation to various human activities can be a deterrent to harvesting, whether use of the resource could result in increased contaminant exposures or not, and the reduced ability to rely on current-use traditional resources has important implications for food security, cultural identity, and community health and wellbeing overall.

As summarized in **Table 1-1**, this country food safety risk assessment includes the following components:

- Identification of chemicals for which adverse health effects could occur through chronic exposures in areas affected by deposition to the landscape of Project emissions.
- Estimation of expected chemical composition of dust fall, near the mine site and along the NAR.
- Quantification of chemical concentrations in soil and plants and wildlife as a result of direct deposition of airborne chemicals or dust fall.
- Comparison of estimated exposures via use of country foods with health-based exposure limits.

5.1 LAND AND RESOURCE USE OF RELEVANCE TO CONTAMINANT EXPOSURE POTENTIAL

Information about traditional and non-traditional land and resource use, as discussed in **Sections 4.3** and **24** of the Project Proposal, shapes our understanding about how project-related changes in resource quality could affect human health and wellbeing, especially with regard to contaminant concentrations.

Among the important and commonly used edible plants in the Coffee Creek area and along the NAR are raspberries, blueberries, blackberries, salmonberries, highbush cranberries, lowbush cranberries, blueberries, kinnikinnik (bearberry), crowberry, Bear root, and Labrador tea. Morel mushrooms are routinely harvested in the spring in coniferous forest areas that experienced a summer wildfire in the preceding year.

Historical or current hunting efforts have focussed in the fortymile woodland caribou, moose, wolves, bears, thinhorn sheep, porcupine, beaver, muskrat, rabbits, ptarmigan, grouse, ducks, and geese. In addition, trapping provides not just furs but also meat resources. The animals that are often trapped include lynx, fox, wolverine, mink, marten, and snowshoe hare. The ridges surrounding Coffee Creek and the northern bank of the Yukon River across from Coffee Creek have been important trapping areas, and some of the earliest registered traplines are documented in that area.

Coffee Creek is well known for being an important fishing location which was used in the past. TH, SFN and WRFN would travel to Coffee Creek and fish. Several fish are fished in the Coffee Creek area including Chinook and Chum salmon, whitefish and grayling (**Section 24.0**).

The plants and animals listed above are by no means an exhaustive list of country resources that were used traditionally and continue to be used.

Hunting patterns have shifted in recent years as some resources such as woodland caribou and moose have become less abundant. A number of voluntary restrictions on traditional hunting are in place for woodland caribou in particular. Nonetheless, the traditional and non-traditional resources are generally considered to be abundant and many people place a high importance on their value for subsistence and other needs.

5.2 CONTAMINANTS OF POTENTIAL CONCERN

The possible sources of mining-related contaminants to the environment, and potentially to traditional and non-traditional food and medicinal resources, include the following:

- Wet and dry deposition of combustion by-products emitted by trucks or vehicles in transit, or by non-road equipment and activities at the mine, such as diesel power generation, or haul trucks
- Dust generation during mine operations and along the NAR, followed by dust fall to the surrounding landscape
- Deposition of waste rock on the surface of the land, and other mining-related physical disturbances, to the extent that the geological materials that are accessible at the land surface have atypically high concentrations of some trace element, and
- Entry of mine contact water into fish-bearing streams and the Yukon River, to the extent that the contact water has higher concentrations of one or more trace elements than found naturally, and if areas of impaired water quality are habitat and foraging areas for edible fish.

Each of these is evaluated further in the subsections below towards the development of a list of plausible COPC.

5.2.1 DEPOSITED COMBUSTION BY-PRODUCTS

There are a large variety of organic compounds in emissions from diesel and gasoline powered equipment. A subset of these is discussed in **Section 4.0** herein as those MSAT considered to be priority contaminants by the United States Environmental Protection Agency (US EPA), based on a combination of their percent contribution to the overall mass of emitted organic compounds and their potential to cause toxic responses in humans at relatively low exposure levels.

Of the priority COPC discussed in **Section 4.1** [i.e., benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, naphthalene, and other polycyclic aromatic matter (as represented herein by benzo[a]pyrene)], very few have the potential to deposit to and persist in soils or on other solid substrates such as plant surfaces. Only those chemicals with sufficiently limited volatility can become deposited and persist.

The specific chemicals with sufficiently low volatility to transfer from air to soil and plants were identified based on the following physical-chemical properties (US EPA 2005):

- Molecular weight > 200 grams / mole
- Henry's Law Constant < 0.00001 atmosphere-m^3 / mole
- Vapour pressure $\leq$ 0.001 mm Hg.

Chemicals that may be subsequently taken up and accumulated in animal tissues were identified based on the following physical-chemical property (US EPA 2005):

- Log Kow > 3.5.

The relevant physical-chemical properties for each non-gaseous COPC are summarized below in **Table 5-1**. Only Benzo[a]pyrene meets all of the above conditions for non-volatility and bioaccumulation.

Table 5-1 Physical-Chemical Properties of Priority Substances Assessed as Mobile Source Air Toxics

COPC	Volatility			Bioaccumulation	
	Molecular Weight (grams/mole)	Henry's Law Constant (atmosphere-m3/mole)	Vapour Pressure (mm Hg)1	Log Kow	Reference
Acetaldehyde	44.05	0.0000789	904.4	0.61	US EPA 2005
Formaldehyde	30.03	0.000000336	5236.4	0.35	US EPA 2005
Acrolein	56.06	0.00012	269.8	-0.01	US EPA 2005
Benzene	78.06	0.0056	95	2.1	US EPA 2005
1,3-Butadiene	54.09	0.074	2110	1.99	ATSDR 2012
Benzo[a]pyrene	252.32	0.0000011	5.50E-9	6	US EPA 2005
Naphthalene	128.18	0.00048	0.08512	3.3	US EPA 2005

Note: Assuming 1 atmosphere = 760 mm Hg
Bold – meets criteria for volatility or bioaccumulation

Formaldehyde was also identified as being sufficiently non-volatile based on one physical-chemical property (Henry's Law Constant); however, the log Kow for formaldehyde is well below the threshold for accumulation in animal tissue; therefore, formaldehyde could potentially occur in soil and plants as a result of deposition from air, but is not a COPC based on uptake into various animals.

As discussed in **Sections 4.4** and 4.5, the maximum predicted annual average concentration of benzo[a]pyrene in air, prior to deposition, at any of the focal areas of interest was 1.6×10^{-5} µg/m^3 (or 16 pg/m^3) at the proposed assay lab area (CO-03). The maximum predicted annual average air concentration of benzo[a]pyrene at focal areas off-site was 3.2×10^{-7} µg/m^3 (or 0.32 pg/m^3) at SA-07, a site near the middle of the Yukon River approximately 10 km downriver from the mouth of Coffee Creek.

According to the International Agency on Cancer Research (2012), "average concentrations of individual PAHs in the ambient air in urban areas typically range from 1 to 30 ng/m^3" (1,000 to 30,000 pg/m^3). The location of the proposed project in the central Yukon is remote from anthropogenic sources of PAHs. According to Wang *et al* (2010), the average annual concentrations of the individual PAHs fluorene and pyrene (which generally occur in air at higher concentrations than benzo[a]pyrene) at the very remote Alert high arctic monitoring site in 2004 were in the range of approximately 35 to 50 pg/m^3. Data presented by Hung et al (2005) from high arctic stations including Alert indicate that TPAHs from monitoring data collected in 1993 through 2000 were in the range of 113 to 516 pg/m^3 in the vapour phase and 38 to 392 pg/m^3 in the particulate phase. The PAH composition in air at high latitudes is dominated by lower molecular weight, more volatile PAHs, given the greater propensity for long range atmospheric transport in comparison with higher molecular weight PAHs such as benzo[a]pyrene. Estimated concentrations of benzo[a]pyrene at high arctic monitoring stations for this same period were in the range of 3 to 17 pg/m^3. Ma *et al* (2013) measured PAHs in the boundary air layer from the North Pacific toward the Arctic Ocean during the summer of 2010. TPAH concentrations ranged from 910 to 7,400 pg/m^3. Sofowote *et al* (2010) provide ambient air quality concentration data from a remote Yukon location (Little Fox Lake), between August 2007 and December 2008: The arithmetic mean concentrations of individual PAHs in the vapour phase were in the range of 0.1 to 93 pg/m^3 and in the particulate phase were in the range of 2 to 20 pg/m^3. Unfortunately, analytical data for benzo[a]pyrene and various other individual PAHs was not reported "*either because their concentrations were consistently below their method detection limits in one or both phase or they had poor sampling efficiencies*".

PAHs were not measured as part of the baseline air quality studies for the Coffee Project air quality assessment. The available studies, however, suggest that the annual average ambient air concentrations of individual PAHs including benzo[a]pyrene in the central Yukon, in areas not affected by local sources, is greater than 2 pg/m^3.

Because the predicted air concentrations of benzo[a]pyrene associated with Project emissions during the peak operational year are within the expected range of ambient concentrations, we conclude that there is no potential for increases in soil or plant tissue concentrations of benzo[a]pyrene as a result of deposition.

Therefore, air to soil / plant partitioning, and trophic transfers were not assessed quantitatively as part of this HHRA.

Similarly, the predicted ranges of formaldehyde in air (annual average concentrations) before deposition (**Sections 4.4** and 4.5) were within or lower than the expected range of remote background conditions for off-site areas adjacent to the Mine Site (SA-01 to SA-15). In remote areas of Canada, formaldehyde occurs in air a concentration in the range of 0.4 to 2.5 µg/m^3 (Finlayson-Pitts and Pitts, 2000).

Predicted airborne formaldehyde concentrations at off-site focal areas of interest were in the range from 0.003 to 0.2 µg/m^3 during peak mine operations. Worst-case air concentrations near ground level of formaldehyde within the core Mine Site area (e.g. near around the assay lab site) are expected to be as high as approximately 0.5 µg/m^3 on average during peak mine operations, which is still within the range of remote background concentrations.

Formaldehyde is produced naturally; for example, from the oxidation of methane by hydroxyl radicals and biogenically by freshwater algae and other biota. Formaldehyde is also photochemically produced in the snowpack and is transferred to the atmosphere through the air-snow interface (Sumner and Shepson, 1999). Formaldehyde is also common in various anthropogenic emissions.

Given that the airborne concentrations of formaldehyde, before deposition, are lower than published estimates of remote ambient air concentrations, this exposure pathway was not assessed further. Formaldehyde was ruled out as a COPC for human health risks.

5.2.2 DUST FALL

The chemical composition of mining related dusts, and the associated dust fall, is related to the chemistry of potential source materials, which primarily include the overburden and waste rock (arising from host rock), ore, and borrow sources that could be used to construct portions of or top-dress the NAR. In addition, some portion of dust fall will be made up of naturally occurring dust including the deposition of tree pollen seasonally, other organic debris from forests, forest fire ash, *et cetera.* Representative trace element compositional profiles of dust fall under existing conditions at the mine site location are presented in the **Baseline Air Quality and Noise** appendices (**Appendix 9-A**, **Appendix 9-B**, **Appendix 10-A**).

Mining related dust fall could alter soil quality or result in increased metal / metalloid uptake to the extent that one or more individual trace elements occurs in the dust source materials at concentrations that are both (i) higher than the average concentrations in local and regional soils or existing road bed, and (ii) higher than soil quality criteria, guidelines or standards that represent risk-based thresholds of soil concentrations designed to be protective of human health.

In order to define the COPC for evaluating risks of dust fall via incidental soil ingestion or uptake into country foods, the available baseline chemistry data for various types of waste rock or ore, and candidate borrow source areas, were screened against relevant health risk based soil screening levels.

The soil concentration thresholds used to screen this large data set included the following:

- British Columbia Contaminated Sites Regulation Schedule 5 soil matrix standards, Parkland land use, human health protection thresholds based on incidental soil ingestion. These values are substantially the same as soil standards adopted in the Yukon *Contaminated Sites Regulation.* If no value was provided for a specific trace element, the value used was the following:
- British Columbia Contaminated Sites Regulation Schedule 10 Generic Numerical Soil Standards, Parkland land use. These standards were developed by the BC Ministry of Environment primarily through consideration of US EPA Integrated Risk Information System (IRIS) chronic oral reference doses, along with standardized assumptions per BC MOE policy regarding soil ingestion rates and a risk quotient of 0.2 to account for the fact that the oral pathway only was accounted for in the exposure estimate. If no value was provided for a trace element, the value used was the following:
- US EPA Region 9 residential areas screening levels based on incidental ingestion, dust inhalation and dermal uptake exposure routes from soil.

The available information on the chemistry of waste rock and ore is extensive, much of it obtained during exploratory drilling. Various representative rock samples from the Project site have been analyzed in the laboratory using inductively coupled plasma – optical emission spectrometry (ICP-OES) ($n \leq 23{,}986$ individual host rock samples excluding additional gold analyses; $n \leq 2{,}275$ individual ore samples excluding additional gold analyses). Many samples have also been collected and analyzed in the laboratory using inductively coupled plasma – mass spectrometry (ICP-MS) ($n = 324$ individual host rock samples; $n \leq 35$ individual ore samples).

Table 5-2 (host rock or waste rock) and **Table 5-3 (**ore) list the maximum observed concentration for each element based on ICP-OES or ICP-MS analyses in comparison with the human health risk-based soil screening value, as described above. Those maximum concentrations that exceeded their respective risk-based screening value are high-lighted in orange.

Those trace elements with maximum observed concentrations greater than their risk-based screening values in both ore and host rock included arsenic, cadmium, cobalt, chromium, iron, manganese, nickel, lead, antimony, tellurium, uranium, and tungsten. Mercury was observed at a concentration greater than its screening value in host rock samples only. These elements where further evaluated as COPC based on the overall statistical distribution for the sample data.

Human exposures to trace elements associated with host rock (waste rock) or ore, either via dust fall or direct exposures in mining affected surface areas, can be best estimated based on the average concentrations of these materials: The process of dust generation and settlement, or human exposures as they move around the environment, will tend to result in the averaging over time of exposure levels even if there is smaller scale spatial variability trace element concentrations in the source materials. This is particularly true for any chronic exposure scenarios, as assessed herein.

As summarized in **Table 5-4**, the 50th percentile concentration in one or more rock types exceeded the risk-based soil screening value only for arsenic, antimony, and tellurium. 50th percentile concentrations of arsenic were up to 25 times higher than the BC Contaminated Sites Regulation (CSR) soil quality standard for human health protection based on soil intake and arsenic is considered to be a contaminant of potential concern.

Telluriium has an estimated average abundance in the upper continental crust of 0.75 parts per million (ppm) (Wedepohl, 1995). Hu and Gao (2008) provide an estimate of Tl average abundance in the upper continental crust of 0.53 ppm. Both of these estimates are useful as predictions of ambient soil and sediment concentrations of Tl, which occurs especially in sulphur and selenium complexes. Since the US EPA Region 9 soil screening level used to screen the Coffee host rock and ore data is lower than the expected average concentration in the upper continental crust, and since the average measured concentrations of Tl did not exceed 0.8 ppm, the observed concentrations are assumed to be within the range of background soil concentrations. Therefore Tl is not considered to be a COPC for health risks associated with exposures to waste rock or mine-derived dusts.

Table 5-2 Screening of Maximum Observed Element Concentration in Waste Rock Against Human Health Risk-based Soil Standards (values exceeding their risk-based soil screening level are highlighted in orange)

Element	*Units*	Risk-based Soil Screening Level	Basis	Granite		Gneiss		Schist	
				ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS
Ag	ppm	390	US EPA R9	0.7	0.14	100	0.58	9.2	0.5
Al	%	7.7	US EPA R9	2.36	1.06	5.38	4.23	6.02	2.98
As	ppm	100	BCCSR Sch.5	10000	3020	4240	2110	6950	1405
B	ppm	16000	US EPA R9	10	10	10	10	10	10
Ba	ppm	6500	BCCSR Sch.5	600	150	4080	1320	3490	2410
Be	ppm	160	US EPA R9	2.9	1.59	4.1	2.17	4.3	2.33
Cd	ppm	3	BCCSR Sch.5	0.5	0.46	3.5	0.32	4.6	0.35
Co	ppm	23	US EPA R9	72	10.5	78	30.6	108	28.6
Cr	ppm	100	BCCSR Sch.5	21	12	636	366	1420	231
Cu	ppm	15000	BCCSR Sch.5	42	14.3	3960	29.2	2770	105.5
Fe	%	5.5	US EPA R9	10.1	2.76	10.8	7.26	8.78	4.69
Hg	ppm	15	BCCSR Sch.5	4	1.73	17	5.22	16	0.75
Li	ppm	160	US EPA R9		20.6		30.5		29.7
Mn	ppm	1800	BCCSR Sch.10	16600	2360	5860	1160	6280	928
Mo	ppm	390	US EPA R9	5	5	131	6.93	29	10.3
Ni	ppm	670	US EPA R9	12	4.6	446	133.5	1930	102.5
Pb	ppm	400	BCCSR Sch.5	38	38.9	1420	87.8	396	48.8
Sb	ppm	31	US EPA R9	330	85	531	83.1	634	66.1
Se	ppm	390	US EPA R9		0.8		1.3		1.4
Sn	ppm	47000	US EPA R9		3.3		15.2		3.9
Sr	ppm	47000	US EPA R9	138	112	1420	763	10000	1630
Tl	ppm	0.47	US EPA R9	10	2.14	10	3.15	10	1.4
U	ppm	16	BCCSR Sch.10	50	45.5	110	29.4	50	9.64
V	ppm	390	US EPA R9	70	11	272	177	298	165
W	ppm	63	US EPA R9	10	18.55	450	6.74	1140	2.17
Zn	ppm	1000	BCCSR Sch.5	128	88	540	72	781	141

Table 5-3 Screening of Maximum Observed Element Concentration in Ore Against Human Health Risk-based Soil Standards (values exceeding their risk-based soil screening level are highlighted in orange)

Element	Units	Risk-based Soil Screening Level	Basis	Granite		Gneiss		Schist	
				ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS
Ag	*ppm*	390	US EPA R9	9.36	0.12	9.6	0.3	206	0.64
Al	*%*	7.7	US EPA R9	1.22	0.94	2.9	1.14	3.69	0.9
As	*ppm*	100	BCCSR Sch.5	10000	10000	10000	1700	10000	5910
B	*ppm*	16000	US EPA R9	10	10	10	10	10	10
Ba	*ppm*	6500	BCCSR Sch.5	430	90	3210	590	3730	1270
Be	*ppm*	160	US EPA R9	2.1	1	3.9	1.33	2.8	1.23
Cd	*ppm*	3	BCCSR Sch.5	<0.5	0.31	2.2	0.1	4.5	0.11
Co	*ppm*	23	US EPA R9	11	10.6	111	6.6	103	16.2
Cr	*ppm*	100	BCCSR Sch.5	15	77	240	107	324	93
Cu	*ppm*	15000	BCCSR Sch.5	21	17	143	58.4	1180	26.4
Fe	*%*	5.5	US EPA R9	10.8	5.94	9.6	2.34	12.4	4.09
Hg	*ppm*	15	BCCSR Sch.5	10	8.5	12	1.77	15	1.61
Li	*ppm*	160	US EPA R9		3.9		5.1		4.1
Mn	*ppm*	1800	BCCSR Sch.10	23700	1740	5630	1490	2810	844
Mo	*ppm*	390	US EPA R9	8	4.24	30	3.09	20	3.27
Ni	*ppm*	670	US EPA R9	6	3.7	158	23.2	501	66.1
Pb	*ppm*	400	BCCSR Sch.5	59	41.5	1190	52.3	253	28.9
Sb	*ppm*	31	US EPA R9	468	145.5	3190	160.5	10000	49.4
Se	*ppm*	390	US EPA R9		<1		<1		<1
Sn	*ppm*	47000	US EPA R9		3.2		1.7		0.9
Sr	*ppm*	47000	US EPA R9	124	61	286	92.2	2680	402
Tl	*ppm*	0.47	US EPA R9	10	2.71	10	1.17	10	1.52
U	*ppm*	16	BCCSR Sch.10	160	53.1	130	19.6	40	5.98
V	*ppm*	390	US EPA R9	24	12	179	17	120	34
W	*ppm*	63	US EPA R9	<10	9.35	30	1.98	1350	1
Zn	*ppm*	1000	BCCSR Sch.5	165	42	181	41	379	57

Table 5-4 Statistical Distribution (as Percentiles) of Trace Elements in Samples of Host Rock and Ore, Coffee Deposit, in Comparison with Risk-Based Soil Screening Levels. (Concentrations that exceeded risk-based screening-values are high-lighted in orange)

Concentration Percentiles	Granite				Gniess				Schist			
	Waste Rock		Ore		Waste Rock		Ore		Waste Rock		Ore	
	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS
Arsenic (ppm): Risk Based Soil Screening Level = 100 ppm												
n	*975*	*57*	*168*	*13*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	2540	2832	7190	8661	578	557	3840	1642	737	334	8070	5520
0.75	582	457	3290	5100	118	100.625	1840	1163	89	102	3800	3035
0.50	118	82	2040	2195	26	25.2	966	1015	24	24.9	2450	1430
0.25	23	10.5	1350	870	7.0	8.8	507	812	8.0	13.75	1320	1220
cadmium (ppm): Risk Based Soil Screening Level = 3 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	*<0.5*	0.09	*<0.5*	0.21	*<0.5*	0.06	*<0.5*	0.09	*<0.5*	0.16	*<0.5*	0.11
0.75	*<0.5*	0.03	*<0.5*	0.06	*<0.5*	0.03	*<0.5*	0.06	*<0.5*	0.08	*<0.5*	0.09
0.50	*<0.5*	0.02	*<0.5*	0.04	*<0.5*	0.02	*<0.5*	0.05	*<0.5*	0.04	*<0.5*	0.08
0.25	*<0.5*	0.01	*<0.5*	0.02	*<0.5*	0.01	*<0.5*	0.03	*<0.5*	0.02	*<0.5*	0.07
cobalt (ppm): Risk Based Soil Screening Level = 23 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	5.0	4.0	11	8.1	20	18	22	6.6	22	23	23	16
0.75	2.0	1.9	3.0	2.0	10	8.1	9.0	5.8	16	18	15	14
0.50	1.0	1.4	2.0	1.2	4.0	2.3	3.0	2.9	13	13	12	12
0.25	1.0	1.1	1.0	0.9	2.0	1.5	2.0	2.1	9.0	10	8.0	11

Concentration Percentiles	Granite				Gniess				Schist			
	Waste Rock		Ore		Waste Rock		Ore		Waste Rock		Ore	
	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS
chromium (ppm): Risk Based Soil Screening Level = 100 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	12	8.2	8.0	76	104	108	48	98	157	166	66	92
0.75	8.0	6.0	6.0	5.8	34	22	16	62	70	78	24	82
0.50	6.0	5.0	4.5	4.5	11	7.5	8.0	6.5	43	42	15	30
0.25	4.0	4.0	3.0	3.0	6.0	5.0	5.0	6.0	24	28	9.0	17
iron (%): Risk Based Soil Screening Level = 5.5 %												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	2.5	2.0	4.3	4.6	3.7	3.2	5.2	2.1	4.3	4.3	4.7	3.7
0.75	1.5	1.4	2.1	1.6	2.2	2.0	2.6	1.8	3.5	3.4	3.6	3.1
0.50	1.2	1.2	1.5	1.3	1.3	1.2	1.5	1.6	3.0	3.0	3.1	2.7
0.25	0.9	1.0	1.0	1.0	0.9	0.9	1.0	1.2	2.4	2.5	2.5	2.6
mercury (ppm): Risk Based Soil Screening Level = 15 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*10*	*856*	*11*
0.95	1.0	0.8	3.0	5.5	1.0	0.8	3.0	1.6	1.0	1.6	3.0	1.1
0.75	*<1*	0.2	1.0	1.3	*<1*	0.09	1.0	1.3	*<1*	1.3	1.0	0.5
0.50	*<1*	0.03	1.0	0.6	*<1*	0.02	1.0	0.7	*<1*	0.7	1.0	0.5
0.25	*<1*	0.01	*<1*	0.3	*<1*	0.01	*<1*	0.5	*<1*	0.5	*<1*	0.4
manganese (ppm): Risk Based Soil Screening Level = 1800 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	1690	1142	2620	1649	786	638	1090	1015	880	753	1060	781
0.75	670	723	485	213	446	398	462	394	624	607	665	664
0.50	444	506	117	73	284	265	250	300	496	526	514	567
0.25	140	154	37	54	193	169	130	193	385	429	372	502

Concentration Percentiles	Granite				Gniess				Schist			
	Waste Rock		Ore		Waste Rock		Ore		Waste Rock		Ore	
	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS	ICP-OES	ICP-MS
antimony (ppm): Risk Based Soil Screening Level = 31 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	36.3	29	169	134	26	21	141	137	25	17	391	45
0.75	6	3.0	36	36	5.0	3.9	47	50	6.0	4.8	54.2	23
0.50	2	1.0	15	25	2.0	1.5	21	40	2.0	2.5	22	19
0.25	*<2*	0.3	10	7.3	*<2*	0.5	10	19	*<2*	0.8	10	12
tellurium (ppm): Risk Based Soil Screening Level = 0.47 ppm												
n		*57*		*14*		*188*		*10*		*79*		*11*
0.95		1.5		2.4		0.7		1.2		0.6		1.1
0.75		0.5		1.2		0.4		1.1		0.4		0.6
0.50		0.3		0.8		0.2		0.6		0.3		0.4
0.25		0.2		0.6		0.1		0.5		0.2		0.3
uranium (ppm): Risk Based Soil Screening Level = 16 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	20	23	60	52	*10*	16	40	19.4875	*<10*	4.4	10	5.6
0.75	10	8.6	20	20	*<10*	6.6	15	18.0875	*<10*	2.4	*<10*	3.4
0.50	*<10*	6.5	10	12	*<10*	3.7	10	13.35	*<10*	1.7	*<10*	2.8
0.25	*<10*	5.6	*<10*	8.9	*<10*	2.4	*<10*	11.0875	*<10*	1.1	*<10*	2.6
tungsten (ppm): Risk Based Soil Screening Level = 63 ppm												
n	*975*	*57*	*168*	*14*	*14296*	*188*	*1251*	*10*	*8715*	*79*	*856*	*11*
0.95	*<10*	7.7	*<10*	3.5	*<10*	1.3	*<10*	1.6	*<10*	1.0	*<10*	0.95
0.75	*<10*	0.26	*<10*	0.35	*<10*	0.45	*<10*	0.89	*<10*	0.45	*<10*	0.65
0.50	*<10*	0.10	*<10*	0.17	*<10*	0.23	*<10*	0.51	*<10*	0.28	*<10*	0.46
0.25	*<10*	0.07	*<10*	0.09	*<10*	0.16	*<10*	0.31	*<10*	0.16	*<10*	0.32

In contrast, estimates by various researchers of the average abundance in the upper continental crust of antimony (Sb) are within the range of 0.2 to 0.75 ppm (Hu and Gao, 2008). The 50th percentile concentrations of Sb in **Table 5-4** are in the range of 1 to 40 ppm.

It is important to note, however, that the 50th percentile concentration of Sb exceeded the health-based soil screening value of 31 ppm only for gneiss ore samples analyzed by ICP-MS, which included only ten samples. Since the ICP-OES data set included 1,251 individual samples for gneiss ore, the 50th percentile concentration estimate of 21 ppm is considered to be a far more robust and accurate estimate of the central tendency of antimony concentration than for the ICP-MS dataset. Since the 50th percentile value of 21 ppm is much lower than the risk-based soil screening value of 31, ppm, antimony is not considered to be a COPC for human health risks.

Overall, only arsenic was selected as a COPC for human health risks in association with dust at or near the mine-site, based on the detailed information available on the chemical composition of potential waste rock or ore.

The areas adjacent to the NAR are too far removed from mine operations such as the open pits, waste rock deposits, or temporary ore stockpiling and crushing to be influenced by dust fall originated from the ore, waste rock, or localized disturbance of mineralized materials (see dust fall predictions documented in **Section 9.0**). There will be no transport of waste rock, ore, or concentrates along the road in a manner that could result in dust. Dust generation and dust fall along the road, therefore, will be a result of vehicular traffic over the road bed.

The road bed will potentially be top-dressed from time-to-time and new sections will have a road base and road bed constructed from locally sourced borrow materials (**Section 2.4.12, 2.5.8**). The chemical composition of possible borrow source materials has been evaluated, as described in **Section 11.0**. The trace element composition of the various samples of candidate borrow source materials is screened in **Table 5-5** against relevant health-based soil screening levels as discussed above. Only four samples out of 38 total exhibited a trace element concentration greater the health risk-based soil screening values, for two trace elements:

- Arsenic: KamRd02, Section 5; KamRd03, Section 7; and KamRd04, Section 8 (all schist), and
- Chromium: KamRd34, Section 4b (schist).

Materials that these samples are representative of will not be used to construct or top dress the road in a manner that leaves them surface accessible and thus could result in dust generation and dust fall.

Table 5-5 COPC Screening for Potential Borrow Source Materials, Northern Access Route (values exceeding their risk-based soil screening level are highlighted in orange)

Sample	Road Section	Rock Type	Ag	Al	As	B	Ba	Be	Cd
			ppm	%	ppm	ppm	ppm	ppm	ppm
Health-based soil screening level			**390**	**7.7**	**100**	**16000**	**6500**	**160**	**3**
Source[1]			***EPA***	***EPA***	***CSR 5***	***EPA***	***CSR 5***	***EPA***	***CSR 5***
KamRd61	Section 1	Schist	0.02	0.39	0.6	*<10*	240	0.39	0.01
KamRd62	Section 1	Schist	0.03	0.42	0.7	*<10*	300	0.23	0.03
KamRd59	Section 2a	Alluvium	0.14	0.50	6.7	*<10*	360	0.23	0.11
KamRd57	Section 2b	Phyllite	0.11	0.34	9.2	*<10*	80	0.12	0.01
KamRd58	Section 2b	Placer Material	0.11	0.40	14.7	*<10*	170	0.44	0.07
KamRd54	Section 3a	Volcanic	0.05	0.57	2.2	*<10*	140	0.2	0.04
KamRd56	Section 3a	Phyllite	0.02	1.46	19.4	*<10*	100	0.33	0.02
KamRd38	Section 3b	Gneiss+Schist	0.01	1.11	0.3	*<10*	120	0.19	0.02
KamRd39	Section 3b	Volcanic	0.01	1.34	4.2	*<10*	240	0.5	0.07
KamRd40	Section 3b	Volcanic	0.04	1.30	2.6	*<10*	700	0.85	0.07
KamRd36-1	Section 4a	Granite	0.03	0.37	0.2	*<10*	120	0.09	0.02
KamRd36-2	Section 4a	Gneiss	0.09	1.33	0.4	*<10*	560	0.21	0.06
KamRd37	Section 4a	Volcanic	0.06	1.26	7.0	*<10*	430	1.05	0.12
KamRd33	Section 4b	Gneiss	0.02	2.08	0.3	*<10*	240	0.33	0.05
KamRd34	Section 4b	Schist	0.27	1.88	0.4	*<10*	590	0.35	0.35
KamRd35	Section 4b	Schist	0.16	1.53	0.2	*<10*	2140	0.21	0.25
KamRd29	Section 4c	Marble	0.01	0.30	0.6	*<10*	60	0.06	0.25
KamRd31	Section 4c	Schist	0.12	1.70	1.3	*<10*	220	0.33	0.03
KamRd32	Section 4c	Alluvium	0.09	1.34	1.8	*<10*	380	0.21	0.12
KamRd02	Section 5	Schist	0.19	1.62	**273[2]**	*<10*	290	0.39	0.12
KamRd30	Section 5	Schist	0.07	2.06	0.5	*<10*	610	0.36	0.11
KamRd03	Section 7	Schist	0.02	1.07	**114**	*<10*	200	0.19	0.03
KamRd04	Section 8	Schist	0.12	1.68	**104**	*<10*	190	0.28	0.57
KamRd24	Section 9	Placer Material	0.01	1.26	4.0	*<10*	110	0.22	0.05
KamRd25	Section 9	Gneiss	0.10	2.63	2.4	*<10*	530	0.26	0.04
KamRd21	Section 10	Gneiss	0.01	1.45	1.8	*<10*	60	0.12	0.04
KamRd23	Section 10	Placer Material	0.02	1.37	3.8	*<10*	180	0.22	0.05
KamRd27	Section 10	Schist	0.01	1.84	1.3	*<10*	80	0.15	0.04
KamRd05	Section 11	Schist	0.01	0.63	33.6	*<10*	120	0.17	0.02
KamRd06	Section 11	Gneiss	0.02	1.13	46.3	*<10*	140	0.12	0.02
KamRd08	Section 12	Schist	*<0.01*	2.17	8.7	*<10*	2140	0.16	0.01
KamRd12	Section 12	Gneiss	0.02	1.85	3.9	*<10*	130	0.23	0.01
KamRd13	Section 12	Schist	0.05	2.37	9.5	*<10*	30	0.2	0.06
KamRd14	Section 12	Gneiss	0.01	1.37	2.9	*<10*	40	0.24	0.03
KamRd15-1	Section 12	Schist	0.04	1.09	8.1	*<10*	200	0.21	0.09
KamRd15-2	Section 12	Granite	0.04	0.56	5.9	*<10*	70	0.17	0.06
KamRd10	Section 13	Alluvium	0.07	1.26	22.4	*<10*	230	0.27	0.14
KamRd28	Section 14	Alluvium	0.04	1.07	8.0	*<10*	110	0.38	0.07

Notes: [1] EPA = US EPA Region 9; CSR 5 = BC Contaminated Sites Regulation Schedule 5; CSR 105 = BC Contaminated Sites Regulation Schedule 10
[2] bolded and highlighted values exceed their respective soil screening levels

Table 5-5 COPC Screening for Potential Borrow Source Materials, Northern Access Route values exceeding their risk-based soil screening level are highlighted in orange) cont'd

Sample	Road Section	Rock Type	Co	Cr	Cu	Fe	Hg	Li	Mn
			ppm	ppm	ppm	%	ppm	ppm	ppm
Health-based soil screening level			**23**	**100**	**15000**	**5.5**	**15**	**160**	**1800**
Source			***EPA***	***CSR 5***	***CSR 5***	***EPA***	***CSR 5***	***EPA***	***CSR 10***
KamRd61	Section 1	Schist	1.5	4	2.9	1.07	<0.01	1.2	127
KamRd62	Section 1	Schist	1.5	8	2.1	0.58	<0.01	1.5	165
KamRd59	Section 2a	Alluvium	3.2	13	12.2	1.02	0.02	4.6	141
KamRd57	Section 2b	Phyllite	0.5	14	20.7	0.64	0.14	1.1	33
KamRd58	Section 2b	Placer Material	1.9	14	18.2	1.35	0.03	2.4	72
KamRd54	Section 3a	Volcanic	3.8	16	5.7	1.63	<0.01	9.1	317
KamRd56	Section 3a	Phyllite	9.1	25	8.7	2.39	<0.01	14.8	224
KamRd38	Section 3b	Gneiss+Schist	5.4	20	8.8	1.67	<0.01	18.5	292
KamRd39	Section 3b	Volcanic	8.5	44	12.3	2.27	<0.01	22.5	513
KamRd40	Section 3b	Volcanic	9.4	80	13	2.22	<0.01	19.5	402
KamRd36-1	Section 4a	Granite	0.3	5	2.4	0.28	<0.01	2	76
KamRd36-2	Section 4a	Gneiss	4.3	40	31	2.18	0.01	36	497
KamRd37	Section 4a	Volcanic	6.6	56	9.4	2.3	<0.01	19.7	576
KamRd33	Section 4b	Gneiss	11.4	16	8.9	2.87	0.01	12.4	429
KamRd34	Section 4b	Schist	13.4	**102**	55.8	3.02	0.01	16.3	497
KamRd35	Section 4b	Schist	7.8	94	50.7	2.09	0.01	10.7	339
KamRd29	Section 4c	Marble	0.8	3	1.8	0.37	<0.01	3.6	190
KamRd31	Section 4c	Schist	10.6	36	12.2	2.86	<0.01	20.5	220
KamRd32	Section 4c	Alluvium	8.2	31	16.5	2.15	0.01	8.4	380
KamRd02	Section 5	Schist	19.5	103	96.3	2.7	<0.01	4.4	379
KamRd30	Section 5	Schist	12.9	63	59.8	2.95	<0.01	9.3	337
KamRd03	Section 7	Schist	5.8	44	20.7	1.44	<0.01	3.7	247
KamRd04	Section 8	Schist	10.6	28	21.8	2.57	0.01	9.5	433
KamRd24	Section 9	Placer Material	9.6	22	9.9	2.26	<0.01	5.1	462
KamRd25	Section 9	Gneiss	11.8	57	31.1	3.74	<0.01	24.2	448
KamRd21	Section 10	Gneiss	12.1	28	14.8	2.13	<0.01	2.9	453
KamRd23	Section 10	Placer Material	8.8	45	14.6	2.26	<0.01	6	366
KamRd27	Section 10	Schist	11.8	20	13.4	2.61	<0.01	3.7	442
KamRd05	Section 11	Schist	1.3	10	8.6	1.62	<0.01	4.2	368
KamRd06	Section 11	Gneiss	6.6	11	5.1	1.67	<0.01	6.2	340
KamRd08	Section 12	Schist	9.9	5	3.8	4.01	<0.01	10.6	656
KamRd12	Section 12	Gneiss	14.5	27	19.1	2.88	<0.01	8.3	397
KamRd13	Section 12	Schist	19.7	62	70.7	3.19	<0.01	10.9	644
KamRd14	Section 12	Gneiss	11.6	37	9.5	2.27	<0.01	3.2	456
KamRd15-1	Section 12	Schist	3.1	8	22.8	1.86	<0.01	3.8	575
KamRd15-2	Section 12	Granite	1.6	7	17.1	1.21	<0.01	1.9	205
KamRd10	Section 13	Alluvium	6.7	25	27.2	2.02	0.01	6.7	358
KamRd28	Section 14	Alluvium	4.3	14	9.8	1.57	0.02	6.3	354

Notes: [1] EPA = US EPA Region 9; CSR 5 = BC Contaminated Sites Regulation Schedule 5; CSR 105 = BC Contaminated Sites Regulation Schedule 10
[2] bolded and highlighted values exceed their respective soil screening levels

Table 5-5 COPC Screening for Potential Borrow Source Materials, Northern Access Route values exceeding their risk-based soil screening level are highlighted in orange) - cont'd

Sample	Road Section	Rock Type	Mo	Ni	Pb	Sb	Se	Sn	Sr
			ppm	ppm	ppm	ppm	ppm	ppm	ppm
Health-based soil screening level			**390**	**670**	**400**	**31**	**390**	**47000**	**47000**
Source			***EPA***	***EPA***	***CSR 5***	***EPA***	***EPA***	***EPA***	***EPA***
KamRd61	Section 1	Schist	0.33	1.2	5.1	0.11	0.8	0.6	15.3
KamRd62	Section 1	Schist	0.14	2.6	2.8	0.06	0.6	0.4	12.2
KamRd59	Section 2a	Alluvium	0.52	8.8	6.5	0.67	0.6	0.2	18.7
KamRd57	Section 2b	Phyllite	0.73	1.4	2.4	0.93	0.9	0.3	6.1
KamRd58	Section 2b	Placer Material	1.1	8.6	17.2	1	0.7	0.2	9.8
KamRd54	Section 3a	Volcanic	0.96	15.6	7.3	0.25	0.6	0.4	52.1
KamRd56	Section 3a	Phyllite	0.14	17.7	4.9	0.05	0.7	0.5	3.5
KamRd38	Section 3b	Gneiss+Schist	0.22	9.7	4.5	*<0.05*	0.2	0.6	12.2
KamRd39	Section 3b	Volcanic	0.28	29.2	11.3	0.16	0.4	0.9	117
KamRd40	Section 3b	Volcanic	0.5	62	21.2	0.14	0.6	1.2	41.6
KamRd36-1	Section 4a	Granite	0.31	1.5	4.6	*<0.05*	0.3	*<0.2*	6
KamRd36-2	Section 4a	Gneiss	0.75	12.3	2.6	*<0.05*	0.7	0.8	9.6
KamRd37	Section 4a	Volcanic	1.43	42.6	13.2	0.21	0.7	1.1	85
KamRd33	Section 4b	Gneiss	0.15	4.1	2.5	*<0.05*	0.2	0.3	46.7
KamRd34	Section 4b	Schist	2.32	66.6	2.8	0.06	2	0.7	20.9
KamRd35	Section 4b	Schist	1.79	39	1.5	*<0.05*	0.9	0.4	11.2
KamRd29	Section 4c	Marble	*<0.05*	1.5	2.3	0.06	0.4	*<0.2*	240
KamRd31	Section 4c	Schist	0.33	29.8	3.2	0.06	1.1	0.3	11.8
KamRd32	Section 4c	Alluvium	0.71	15.7	3.5	0.09	0.5	0.4	43.5
KamRd02	Section 5	Schist	1.4	82.6	1.5	0.44	1.2	0.4	38.3
KamRd30	Section 5	Schist	0.56	26.5	1	*<0.05*	0.6	0.2	30.4
KamRd03	Section 7	Schist	0.33	11.1	3.5	0.14	0.3	0.5	22.7
KamRd04	Section 8	Schist	0.97	10.9	8.3	0.24	0.9	0.3	36
KamRd24	Section 9	Placer Material	0.53	9.4	2.5	0.17	0.2	0.2	44.9
KamRd25	Section 9	Gneiss	0.83	16.4	3.2	*<0.05*	1	0.7	19.8
KamRd21	Section 10	Gneiss	0.1	12.5	0.6	*<0.05*	0.3	0.3	28.3
KamRd23	Section 10	Placer Material	0.36	12.2	1.9	0.07	0.3	0.4	156.5
KamRd27	Section 10	Schist	0.12	7.2	0.3	*<0.05*	0.2	0.2	34
KamRd05	Section 11	Schist	0.15	1.1	1.4	0.07	0.7	0.3	13.2
KamRd06	Section 11	Gneiss	0.13	2.6	4.7	0.09	*<0.2*	0.6	28.4
KamRd08	Section 12	Schist	0.07	2.2	0.7	*<0.05*	0.4	0.7	24.2
KamRd12	Section 12	Gneiss	0.1	7.7	1.6	0.06	0.3	0.2	60
KamRd13	Section 12	Schist	0.34	26.6	2	0.25	0.3	0.2	96.2
KamRd14	Section 12	Gneiss	0.21	14.2	0.5	*<0.05*	0.3	0.3	17.6
KamRd15-1	Section 12	Schist	0.52	2	3.2	*<0.05*	0.6	0.9	30
KamRd15-2	Section 12	Granite	0.18	0.8	3.2	*<0.05*	1	0.6	11.7
KamRd10	Section 13	Alluvium	0.59	10	4.2	0.31	0.4	0.4	47.1
KamRd28	Section 14	Alluvium	0.86	6.3	9.4	0.26	0.4	0.8	22.7

Notes: [1] EPA = US EPA Region 9; CSR 5 = BC Contaminated Sites Regulation Schedule 5; CSR 105 = BC Contaminated Sites Regulation Schedule 10
[2] bolded and highlighted values exceed their respective soil screening levels

Table 5-5 COPC Screening for Potential Borrow Source Materials, Northern Access Route values exceeding their risk-based soil screening level are highlighted in orange) cont'd

Sample	Road Section	Rock Type	Tl	U	V	W	Zn
			ppm	ppm	ppm	ppm	ppm
Health-based soil screening level			**0.47**	**16**	**390**	**63**	**1000**
Source			***EPA***	***CSR 10***	***EPA***	***EPA***	***CSR 5***
KamRd61	Section 1	Schist	0.33	1.2	5.1	0.11	0.8
KamRd62	Section 1	Schist	0.14	2.6	2.8	0.06	0.6
KamRd59	Section 2a	Alluvium	0.52	8.8	6.5	0.67	0.6
KamRd57	Section 2b	Phyllite	0.73	1.4	2.4	0.93	0.9
KamRd58	Section 2b	Placer Material	1.1	8.6	17.2	1	0.7
KamRd54	Section 3a	Volcanic	0.96	15.6	7.3	0.25	0.6
KamRd56	Section 3a	Phyllite	0.14	17.7	4.9	0.05	0.7
KamRd38	Section 3b	Gneiss+Schist	0.22	9.7	4.5	*<0.05*	0.2
KamRd39	Section 3b	Volcanic	0.28	29.2	11.3	0.16	0.4
KamRd40	Section 3b	Volcanic	0.5	62	21.2	0.14	0.6
KamRd36-1	Section 4a	Granite	0.31	1.5	4.6	*<0.05*	0.3
KamRd36-2	Section 4a	Gneiss	0.75	12.3	2.6	*<0.05*	0.7
KamRd37	Section 4a	Volcanic	1.43	42.6	13.2	0.21	0.7
KamRd33	Section 4b	Gneiss	0.15	4.1	2.5	*<0.05*	0.2
KamRd34	Section 4b	Schist	2.32	66.6	2.8	0.06	2
KamRd35	Section 4b	Schist	1.79	39	1.5	*<0.05*	0.9
KamRd29	Section 4c	Marble	*<0.05*	1.5	2.3	0.06	0.4
KamRd31	Section 4c	Schist	0.33	29.8	3.2	0.06	1.1
KamRd32	Section 4c	Alluvium	0.71	15.7	3.5	0.09	0.5
KamRd02	Section 5	Schist	1.4	82.6	1.5	0.44	1.2
KamRd30	Section 5	Schist	0.56	26.5	1	*<0.05*	0.6
KamRd03	Section 7	Schist	0.33	11.1	3.5	0.14	0.3
KamRd04	Section 8	Schist	0.97	10.9	8.3	0.24	0.9
KamRd24	Section 9	Placer Material	0.53	9.4	2.5	0.17	0.2
KamRd25	Section 9	Gneiss	0.83	16.4	3.2	*<0.05*	1
KamRd21	Section 10	Gneiss	0.1	12.5	0.6	*<0.05*	0.3
KamRd23	Section 10	Placer Material	0.36	12.2	1.9	0.07	0.3
KamRd27	Section 10	Schist	0.12	7.2	0.3	*<0.05*	0.2
KamRd05	Section 11	Schist	0.15	1.1	1.4	0.07	0.7
KamRd06	Section 11	Gneiss	0.13	2.6	4.7	0.09	*<0.2*
KamRd08	Section 12	Schist	0.07	2.2	0.7	*<0.05*	0.4
KamRd12	Section 12	Gneiss	0.1	7.7	1.6	0.06	0.3
KamRd13	Section 12	Schist	0.34	26.6	2	0.25	0.3
KamRd14	Section 12	Gneiss	0.21	14.2	0.5	*<0.05*	0.3
KamRd15-1	Section 12	Schist	0.52	2	3.2	*<0.05*	0.6
KamRd15-2	Section 12	Granite	0.18	0.8	3.2	*<0.05*	1
KamRd10	Section 13	Alluvium	0.59	10	4.2	0.31	0.4
KamRd28	Section 14	Alluvium	0.86	6.3	9.4	0.26	0.4

Notes: [1] EPA = US EPA Region 9; CSR 5 = BC Contaminated Sites Regulation Schedule 5; CSR 105 = BC Contaminated Sites Regulation Schedule 10
[2] bolded and highlighted values exceed their respective soil screening levels

All other materials exhibited trace element concentrations that were far below the health risk-based soil screening values. As a result, it is not possible for dust generated from the tested borrow materials to result in trace concentrations in adjacent soils as a result of dust fall that are higher than the risk-based screening levels. Therefore trace elements in dust fall adjacent to the NAR are not considered to be contaminants of potential concern for human health.

5.2.3 Surface Accessible Highly Mineralized Mine Wastes and Disturbances

The chemistry of host rock and ore is reviewed above in the context of dust generation and dust fall. Following the same reasoning for surface accessible geological materials at mine closure, arsenic was determined to be a contaminant of potential concern with regard to human exposures in the vicinity of waste rock deposits and mining-related disturbances in more highly mineralized areas of the decommissioned mine site, assuming no measures to reduce such exposures by capping / encapsulation, consolidation, or other approaches. No other trace elements were observed to occur in potential waste rock samples or ore at concentrations in excess of the health risk-based soil screening levels.

5.2.4 Changes in Water Quality and Fish Tissue

Inclusion of this exposure scenario in the HHRA is only relevant if there are predicted changes in water quality (e.g., uranium water and sediment concentrations) during operations or post closure after the application of mitigations.

No residual effects on water quality (and hence fish tissue quality) are expected due to erosion and sedimentation, or atmospheric deposition, with the implementation of proposed mitigation measures, BMPs, and management plans.

A water balance/water quality model (**Appendix 12-C**) was used to predict Project and other influences on surface water quality in association with leaching from disturbed mine materials and wastes or HLF residues, including leaching of nitrogen residues generated from blasting. Predicted maximum monthly concentrations of various substances as presented in **Appendix 12-B** are summarized below for each of the Coffee Creek/Latte Creek watershed areas and the Yukon River, and compared with Canadian Drinking Water Quality Guidelines, as a screening approach for assessing health effects.

The predicted maximum monthly concentrations in surface waters arising from the project do not reflect a potential for shorter term episodic bouts of higher instantaneous concentrations, depending on the particulars of localized surface water – groundwater interactions, evaporation, sediment re-suspension or settlement, and temporary disequilibria in sorbed-phase dissolved-phase partitioning. The comparisons provided in **Table 5-6** to Canadian Drinking Water Guidelines are nonetheless instructive for identifying substances in surface waters that could result in health effects based on extended periods of local surface water use as a potable water supply.

Table 5-6 Highest Predicted Maximum Monthly Concentrations (mg/L) of Nutrients and Trace Elements

Substance	Canadian Drinking Water Guidelines (2014), Maximum Acceptable Concentration (MAC)	Coffee Creek/ Latte Creek/ Hidden Creek/ YT-24 Tributary		Yukon River
		Baseline Conditions[1,2]	With Project	With Project
Ammonia -N	No value (not required)		0.36	0.031
Nitrate-N	10	0.85	11	0.18
Nitrite-N	1	0.085	0.037	0.014
Sulphate	≤500 (aesthetic objective)	469	370	28
P		0.27	0.058	0.23
WAD CN	0.2	0.0034	0.0022	0.00090
D-Al		0.741	0.35	0.19
Ag	No value (not required)	0.0001	0.000015	0.000041
As	0.010	0.013	0.0067	0.0025
Ca	No value (not required)	143	140	32
Cd	0.005	0.000222	0.000042	0.00052
Cr	0.05	0.0019	0.0014	0.0028
Cu	≤1.0 (aesthetic objective)	0.013	0.0037	0.0081
Fe	≤0.3 (aesthetic objective)	9.7	0.93	3.2
Hg	0.001	0.000021	0.000012	0.0000089
Mg	No value (not required)	55	45	8.8
Mn	≤0.05 (aesthetic objective)	0.31	0.12	0.18
Mo		0.0030	0.030	0.0014
Ni		0.020	0.0020	0.0099
Pb	0.010	0.0045	0.00040	0.0024
Sb		0.0015	0.0062	0.0019
Se	0.05	0.00097	0.00074	0.00050
Tl		0.00012	0.00021	0.000032
U	0.02	0.10	0.10	0.0030
Zn	≤5.0 (aesthetic objective)	0.046	0.019	0.037

Notes: (shaded cells indicate values that exceed Canadian drinking water guidelines)

[1] data from **Section 12.0, Appendix 12-A**; [2] measured total, not dissolved, concentration for trace elements.

The highest predicted maximum monthly concentrations were greater than their respective drinking water guideline for Fe and Mn. The guideline values are aesthetic objectives, however, and do not indicate any potential for direct health risks. In addition, baseline data collections show that the aesthetic objectives for Fe and Mn are exceeded based on average concentrations observed under baseline (pre-Project) conditions for portions of especially Latte Creek, Halfway Creek and the Yukon River.

For nitrate-N, the Canadian drinking water quality value was exceeded by the predicted maximum monthly concentration only for YT-24, and only by a value of 10%. Thus, it is unlikely that nitrate will exceed human health drinking water guidelines as a result of the Project, given the conservatism incorporated in predictions developed using the water quality model.

Predicted maximum monthly concentrations of uranium in Coffee and Latte Creeks (locations CC1.5, CC3.5, CC4.5), Halfway Creek (HC2.5, HC5.0) and YT-24 are higher than the current Canadian water quality guidelines, but the predicted concentrations in the Yukon River do not exceed the drinking water guideline. A more detailed evaluation of potential health risks associated with use of local surface water supplies for potable water is provided below.

5.3 FOCAL AREAS OF INTEREST

Dust fall predictions (as total dry mass in mg/dm^2/year) at and near the Mine Site during the peak operational year are presented in **Section 9.0**. As discussed in **Section 3.2** herein for the noise HHRA, predicted changes at focal areas of interest were used to develop a good understanding about potential project-related changes at locations where people may permanently or temporarily reside, or frequently use for recreational, food-gathering or spiritual purposes, *et cetera*. The focal areas of interest for the mine operational phase, in areas adjacent to the Mine Site, are the same as used in the noise HHRA and air quality HHRA, as presented in **Table 3-1** and **Figure 5-1** because they also are based on the locations where people reside, regularly visit, or use resources.

5.4 HUMAN SOIL AND FOOD ARSENIC EXPOSURES FROM DUST FALL

Since the concentration of arsenic is expected to exceed 100 mg/kg for the major portion of ore and waste rock handled during mine operations, it is conceivable that dust originating from various mining activities will have elevated concentrations of arsenic. As discussed in **Section 5.2.2**, no other trace element occurs in ore or waste rock at a concentration that will result in health risks in humans. Similarly, the concentrations of organic compounds deposited on soils and plant surfaces after emission from diesel equipment or other fossil fuel powered equipped are predicted to be sufficiently low that they will not result in a measurable increase in environmental concentrations relative to existing conditions.

Dust fall in areas adjacent to the NAR is likely to have a chemical composition that is similar to naturally occurring dusts, based on the chemistry of candidate borrow source materials that could be used to construct new sections of road or top-dress existing road surfaces. Dust fall within and near the Mine Site, however, could deliver concentrations of arsenic to soils and plant surfaces that exceed natural background levels.

Predictive estimates for dust fall concentrations at and near the Mine Site at focal areas of interest are discussed in **Section 9.0.** The estimated concentrations during the peak operational year (Year 6) of total

suspended particulates (TSP, as mg/m^3) and dust fall (as mg dust/dm^2/day) are provided in **Table 5-7.**

Table 5-7 also provides predictions for each site of arsenic flux to soil, as dust fall, and the realized concentrations over the 10 years of mine operation based on cumulative loadings. These predictions are based on the following assumptions:

(i) The 50th percentile concentrations of arsenic in mined rock and ore (**Table 5-4**) adequately reflects the dust fall concentration over the operational life of the mine.

   a. The average arsenic concentration in waste rock was estimated to be 56 mg/kg, which is the average of the 50th percentile concentrations for granite, gneiss and schist waste rock.

   b. The average arsenic concentration in ore was estimated to be 1,820 mg/kg, which is the average of the 50th percentile concentrations for granite, gneiss and schist ore.

   Soil arsenic concentrations were predicted separately for dust fall originating from waste rock and from ore. In reality, the true dust fall composition is likely to reflect an integrated contribution from waste rock, ore, and natural existing dust sources, with the relative composition varying across sites.

(ii) Dust fall rates over the ten-year period are generally equal to the geometric mean value of predicted 24-h average dust fall rates for the peak operational year.

(iii) All of the arsenic that is introduced to soil as dust fall over the ten-year period is retained in the top 2 cm of soil. None is removed through dissolution and downward movement or other processes.

(iv) The soil bulk density is 1.6 g/cm^3.

Based on these assumptions, the highest estimated arsenic concentration in soils as a result of mining-related dust fall was 0.056 mg/kg (ppm) or 56 µg/kg (ppb) at site CO-01/CO-02 (permanent camp complex and mine/dry office area).

The predicted soil arsenic concentrations in **Table 5-7** would be incremental to the mass of arsenic contained in soil naturally or from other sources. Arsenic soil concentrations in the Coffee Creek area under existing (pre-mining) conditions are discussed in the **Appendix 15-A Vegetation Baseline Report**. Soil samples within the Coffee area exhibited an average arsenic concentration of 26 mg/kg. Soil samples collected adjacent to the Coffee area exhibited an average arsenic concentration of 15 mg/kg, and soil samples from nearby the Coffee area (farther away than the adjacent soil sites) had an average arsenic concentration of approximately 7.5 mg/kg.

Table 5-7 Predicted Concentrations of Total Suspended Particulate, Dust Fall, and Arsenic Loading to Soil

Focal Area of Interest		Total Suspended Solids (mg/m^3)		Predicted Dust fall ($mg/dm^2 \cdot d$)		Predicted Annual Arsenic Flux to Soil ($mg/dm^2 \cdot yr$)		Predicted Arsenic Concentration in Soil (mg/kg)	
		Max. of 24-h Averages	Annual Geomean of 24-h Averages	Max. of 24-h Averages	Annual Geomean of 24-h Averages	Based on 50th %tiles of Ore Chemistry	Based on 50th %tiles of Waste Rock Chemistry	Based on 50th %tiles of Ore Chemistry	Based on 50th %tiles of Waste Rock Chemistry
CO-01: **CO-02:**	Permanent Camp, incl. dormitory and kitchen / dining / recreation complex and Mine Dry and Office Complex	2141	218	22	2.7	1.8E+00	5.5E-02	5.6E-02	1.7E-03
CO-03:	Assay Lab	1005	94.2	15	1.4	9.2E-01	2.8E-02	2.9E-02	8.9E-04
CO-04:	Truck shop/warehouse	1468	123	15	1.8	1.2E+00	3.7E-02	3.8E-02	1.2E-03
CO-05:	Near airstrip	79.5	6.2	0.94	0.054	3.6E-02	1.1E-03	1.1E-03	3.5E-05
SA-01:	Coffee Cr.	11	0.67	0.12	0.005	3.3E-03	1.0E-04	1.0E-04	3.1E-06
SA-01a:	Coffee Cr. Mouth (Same as VM4)	10	0.65	0.11	0.005	3.1E-03	9.5E-05	9.7E-05	3.0E-06
SA-02:	Yukon River – centre channel, ~1.5 km upriver from Coffee Creek mouth	8.8	0.58	0.11	0.004	2.8E-03	8.8E-05	8.9E-05	2.7E-06
SA-03:	Yukon River – centre channel, ~1.5 km downriver from Coffee Creek mouth	20	1.3	0.22	0.009	5.9E-03	1.8E-04	1.9E-04	5.7E-06
SA-04:	Yukon River – centre channel, ~3 km downriver from Coffee Creek mouth	14	0.93	0.14	0.006	4.2E-03	1.3E-04	1.3E-04	4.1E-06
SA-05:	Yukon River – centre channel, ~5 km downriver from Coffee Creek mouth	11	0.76	0.11	0.005	3.4E-03	1.1E-04	1.1E-04	3.3E-06
SA-06:	Yukon River – centre channel, ~7 km downriver from Coffee Creek mouth	23	1.8	0.24	0.013	8.7E-03	2.7E-04	2.7E-04	8.3E-06
SA-07:	Yukon River – centre channel, ~10 km downriver from Coffee Creek mouth	34	2.5	0.23	0.024	1.6E-02	5.0E-04	5.0E-04	1.6E-05
SA-08:	Confluence of Latte and Coffee Creeks	28	1.2	0.26	0.010	6.4E-03	2.0E-04	2.0E-04	6.2E-06
SA-09:	Height of land across Yukon R. from Coffee Creek	6.7	0.50	0.10	0.004	2.5E-03	7.7E-05	7.8E-05	2.4E-06
SA-10:	Height of land across Yukon R. from proposed Mine Site	18	0.83	0.17	0.006	3.7E-03	1.1E-04	1.2E-04	3.6E-06
SA-11:	Height of land across Yukon R. to ea.	14	1.4	0.28	0.014	9.4E-03	2.9E-04	2.9E-04	9.1E-06
SA-12:	Ballarat Creek Area, N. of Yukon River	4.6	0.38	0.09	0.003	2.0E-03	6.0E-05	6.1E-05	1.9E-06
SA-13:	Yukon River foreshore east of existing Coffee Creek camp	13	0.90	0.13	0.006	4.1E-03	1.3E-04	1.3E-04	4.0E-06
SA-14:	Wilderness Retreat, on Yukon River	21	2.4	0.25	0.030	2.0E-02	6.2E-04	6.3E-04	1.9E-05
SA-15:	Representative harvesting area - height of land	11	1.6	0.33	0.022	1.5E-02	4.5E-04	4.6E-04	1.4E-05

The maximum predicted arsenic concentration associated with mining operational dust fall (0.056 mg/kg) represents approximately 0.7% of the average observed arsenic concentration in nearby soil samples of 7.5 mg/kg. This estimated incremental addition to the existing soil concentrations of arsenic are about an order of magnitude lower than the expected level of analytical precision based on modern analytical methods, which is approximately 5% as a relative percent difference between two samples.

Based on these predictions, we conclude that dust fall associated with mine operations, at or near the location with the maximum predicted dust fall rates, will not result in a detectable change in soil concentrations of arsenic. By extension, there is no potential for increased uptake into biota or increased arsenic exposure potential for humans involved in hunting and gathering activities.

It should also be noted that areas beyond the Mine Site, as represented by focal areas of interest SA-01 through SA-15, are likely to have far lower mining-related dust fall rates than in the proposed area of the permanent camp and mine dry / office complex as discussed above. The highest predicted dust fall rate for areas beyond the mine was for location SA-07, on the Yukon River approximately 10 km down river from the mouth of Coffee Creek (geometric mean of average 24-h dust fall rates of 0.010 mg/dm^2•day). Based on the conservative assumptions documented above, the soil arsenic concentration in upland areas in the vicinity of SA-07 (for example on the vegetated portion of the island in the Yukon River immediately adjacent to this site) was predicted to be 0.0005 mg/kg, assuming that all dust fall for this site originates from ore. This value is less than 0.01% of the expected natural arsenic concentration in soil.

The dust fall rates predicted for the peak operational year at any of the sites beyond the Mine Site proper were generally similar to or lower than observed dust deposition rates under existing conditions, as documented in the **Air Quality and Noise Baseline Report** (i.e., in the range of 0.12 to 1.4 mg/dm^2 • d), so the predicted negligible concentration of mining-related dust fall to soil arsenic concentrations is primarily a reflection of the fact that the off-site focal areas of interest are sufficiently far removed from mining operations to be beyond the influence of any mining-related dust fall.

5.5 HUMAN SOIL AND FOOD ARSENIC EXPOSURES IN WASTE ROCK DISPOSAL AREAS

One of the potential influences on the environment of the Project is the placement of a large mass of waste rock on the slopes adjacent to the mine, and waste rock was shown above to contain arsenic as a COPC. During mine operation, institutional controls (e.g. signage, security staff) would likely prevent or minimize human exposure to on-site waste rock storage areas. However, following mine closure such institutional controls may no longer exist, and waste rock may or may not be covered with clean overburden.

Given the mine's remote wildland setting, post-closure human exposure to uncovered waste rock disposal areas would likely be limited to a recreational or hunting / gathering scenario. People who in the future might visit waste rock disposal areas might include hunters, fishers, users of all-terrain vehicles, or First Nation hunters / gatherers. The risks to such individuals, following mine closure, was evaluated through

evaluation of exposure using near worst-case assumptions. It was assumed that people might camp at the former mine site area atop or in proximity to waste rock. During such temporary occupancy, people out on the land might be exposed to waste rock arsenic contamination via the exposure pathways shown in **Figure 5-1**.

Human health exposure and risks were quantified in terms of daily and lifetime-average contaminant intake doses (mg/kg/day) for the routes of exposure deemed to be complete exposure pathways as shown in Figure A. Internalized doses of arsenic for each relevant exposure pathway were calculated for both adults and toddlers (toddlers can have considerably higher potential incidental soil ingestion rates) using standardized Health Canada equations (see **Appendix B**), based on exposure assumptions shown below in **Table 5-8.** Exposure assumptions were based on a combination of default Health Canada values and / or based on professional judgement (PJ) around realistic future site use.

Exposure and contaminant intakes were evaluated separately for each of the three waste rock types, in order to determine whether different risk management strategies may be necessary for the different rock types. Plants and berries were assumed to be able to grow in abundance in the waste rock despite limited nutrients and organic content.

Plant and small mammal tissue concentrations were estimated using Bechtel Jacobs (1998) and Sample et al. (1998) recommended “General Estimate” bioaccumulation models. These are exponential relationships between soil concentration and dry weight tissue concentrations that were developed through field and laboratory validation research. With respect to small mammals, three models are available for different feeding niches, insectivores (e.g. shrews), omnivores (e.g. mice), and herbivores (e.g. rabbit). The most conservative of these models for arsenic is that for herbivores. Consequently the herbivore model was used as the primary small mammal bioaccumulation model. Herbivores such as rabbit are also likely a potential country food item. Bechtel Jabobs has indicated their plant bioaccumulation model is primarily for above ground plant parts, and not roots or berries; however, in the absence of a root or berry model the general plant model was used to predict arsenic concentrations in all edible vegetation, including berries.

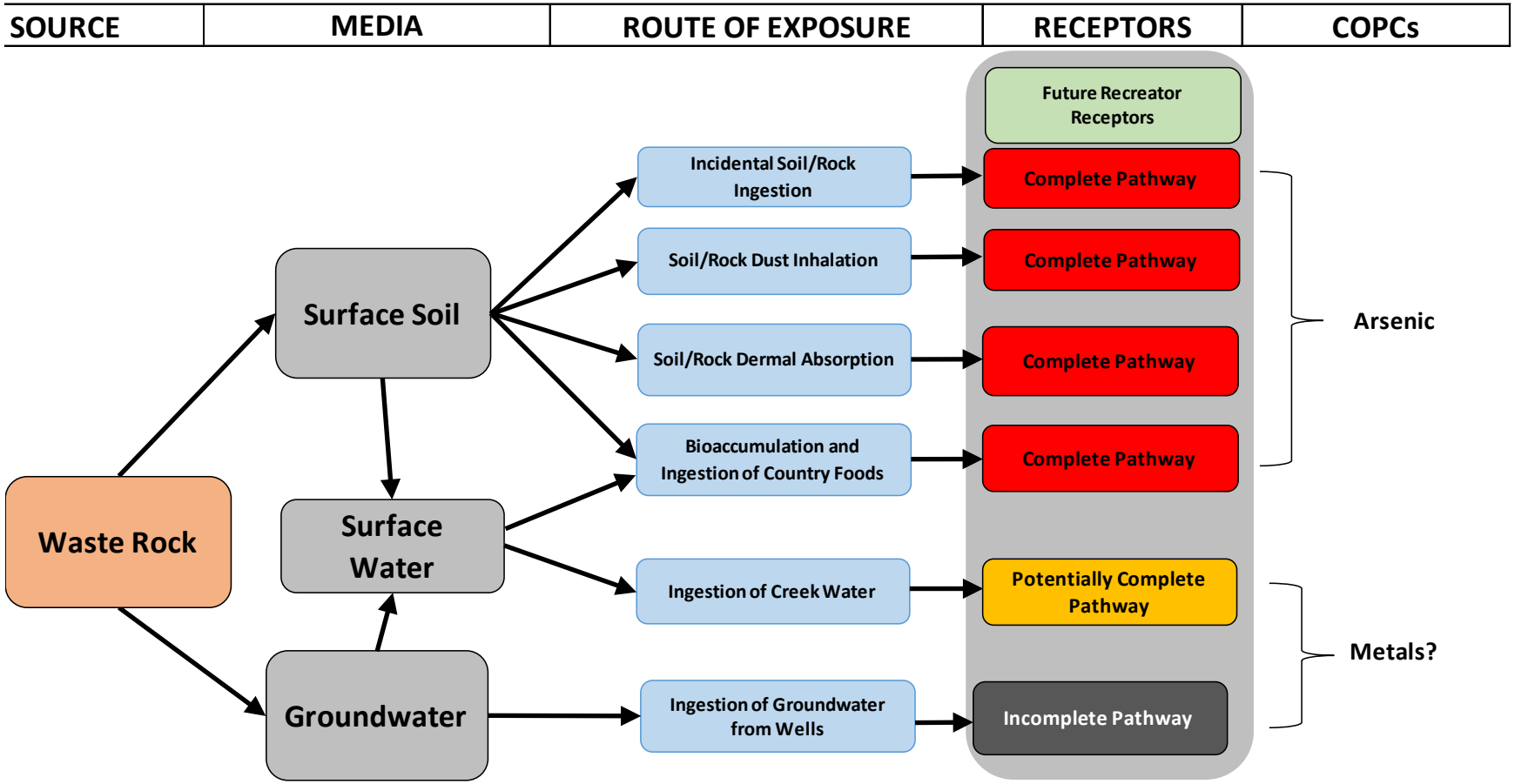

Figure 5-1 Focal Areas of Interest for the Noise and Air Quality Human Health Risk Assessment

Table 5-8 Post-Closure Human Exposure Assumptions

Exposure Assumptions	Toddler	Adult	Ref
Soil arsenic exposure concentration (Cs) (mg/dkg)	50th percentile: Granite WR Gneiss WR Schist WR	50th percentile: Granite WR Gneiss WR Schist WR	Table 5.2-4
Plant arsenic exposure concentration (Cp) (mg/wkg)	Modelled: Cp=(e^0.564*lnCs-1.992)(1-0.85)	Modelled: Cp=(e^0.564*lnCs-1.992)(1-0.85)	Bechtel Jacobs (1998)
Small mammal arsenic exposure concentration (Csm)(mg/wkg)	Modelled: Csm=(e^0.7354*lnCs-4.5796)(1-0.68)	Modelled: Csm=(e^0.7354*lnCs-4.5796)(1-0.68)	Sample et al. (1998)
Exposure duration (hours/day)	24	24	PJ
Exposure duration (days/week)	7	7	PJ
Exposure duration (weeks/year)	2	2	PJ
Years exposed (years)	4.5	20	PJ
Incidental soil ingestion rate (kg/day)	0.00008	0.00002	HC (2010a)
Surface area hands (cm2)	430	890	HC (2010a)
Surface area lower arms and legs (cm2) ½ full arms and legs surface areas	1290	4110	HC (2010a) / PJ
Soil loading hands (kg/cm2)	1E-07	1E-07	HC (2010a)
Soil loading arms and legs (kg/cm2)	1E-08	1E-08	HC (2010a)
Soil particulate concentration in air (kg/m3)	7.60E-10	7.60E-10	HC (2010a)
Inhalation rate (m3/hour)	0.3	0.7	HC (2010a)
Country food ingestion rate (kgw/day) (0.17 kg = 6 oz, and is roughly 1-1.5 cups berries).	0.17	0.1 (assumed 60% adult rate)	PJ
Body weight (kg)	16.5	70.7	HC (2010a)
Life expectancy (years)	80	80	HC (2010a)

Dry weight tissue concentrations were converted to wet weight plant concentrations by assuming an 85% and 68% moisture content for plants and small mammals, respectively, based on information in **Table 4-1** of US EPA (1993). Predicted plant and small mammal concentrations based on 50th percentile soil concentrations were similar and are shown in **Table 5-9**.

Table 5-9 Predicted Plant and Small Mammal Arsenic Concentrations

COPC	Granite Waste Rock	Gneiss Waste Rock	Schist Waste Rock
	50th percentile	50th percentile	50th percentile
Arsenic			
Soil (mg/kg dry weight)	118	26.0	24.0
Plants (mg/kg wet weight)	0.30	0.13	0.12
Small Mammals - herbivores (mg/kg wet weight)	0.26	0.05	0.04

Note: Concentrations in tissue shown as wet weight concentration (mg/ kg wet weight)

Given that plants and berries are likely to be the predominant country food items harvested and consumed from the site, and given that plants are predicted to contain higher arsenic concentrations than small mammals, the following quantification of risks associated with country foods ingestion focussed on consumption of country foods containing concentrations of arsenic as predicted in plants.

5.6 ARSENIC HUMAN HEALTH TOXICOLOGICAL REFERENCE VALUES

Excessive chronic exposure to arsenic can cause skin lesions (hyperpigmentation, keratosis) and blackfoot disease (blood vessel damage). Arsenic is also a Class A, Type 1 human carcinogen, potentially inducing multiple types of cancer (skin, lung, liver, kidney, bladder).

Toxicological reference values for arsenic from three leading health agencies, the US EPA Integrated Risk Information System (IRIS), Health Canada (2010b), and the Agency for Toxic Substances and Disease Registry (ATSDR, 2007), were short listed for possible use in this HHRA, and are summarized in **Table 5-10.**

With respect to non-cancer health effects the ATSDR's acute oral reference doses re likely most relevant to a wildlands setting with short-term (acute) human use, and has been used in risk calculations presented in **Section 5.7**. Oral reference doses are generally conservative benchmarks, with incorporated safety factors, representing values below which adverse health effects are not anticipated, but, above which risk of health effects increases. With respect to cancer, Health Canada's slope factors have been used in risk calculations presented in **Section 5.7** given they are based on slightly more recent literature review.

Table 5-10 Human Health Toxicological Reference Values for Arsenic

Exposure Duration	TRV	Health Effect	Ref
Non-Cancer Threshold Level Effects			
Chronic (months to years) oral reference dose	0.003 mg/kg-d (0.014 mg/kg-d LOAEL / UF of 3)	Skin lesions (keratosis, hyperpigmentation) and blackfoot disease	US EPA IRIS (1991) - Tseng (1968, 1977)
Acute (2-3 weeks) oral reference dose	0.005 mg/kg-d (0.05 mg/kg-d LOAEL / UF of 10)	Face edema, gastrointestinal symptoms (nausea, vomiting, diarrhea)	ATSDR (2007) – Mizuta et al. (1956)
Cancer Risk			
Oral slope factor	1.5 mg/kg-d-1	Skin cancer	US EPA IRIS (1995) – Tseng (1968, 1977), US EPA (1988)
Oral slope factor	1.8 mg/kg-d-1	Bladder, lung, liver cancer	Health Canada (2006) – Morales (2000)
Inhalation unit risk, Inhalation Slope Factor	4.3 (mg/m3)-1 = ~15.1 mg/kg-d-1	Lung cancer	US EPA IRIS (1995) – Multiple sources 1982-1983
Inhalation unit risk, Inhalation Slope Factor	6.4 (mg/m3)-1 = 27 mg/kg-d-1	Lung cancer	Health Canada (1993) – Higgins et al. (1986)

5.7 Food and Soil Arsenic Risk Characterization and Uncertainty

The potential for individuals involved in food collection to develop non-cancer adverse health effects through use of the site's waste rock disposal areas was estimated by comparing the average daily intake doses while on site with the acute oral reference dose presented in **Section 5.6**. Such a comparison represents risk as a simple hazard quotient (magnitude site exposure dose exceeds safe dose) based on the following calculation.

$$HQ = \frac{ADD}{RFD}$$

Where:

HQ = Hazard Quotient

ADD = Average Daily Dose (mg/kg/day) of contaminant for period of exposure

RFD = Reference Dose, dose below which no effect anticipated (mg/kg/day)

HQ values in excess of one represent a scenario whereby site dose exceeds the health-based benchmark value. HQ values were also summed for multiple exposure pathways to produce a combined hazard index (HI). In Yukon and BC, under their CSR, which are applicable to mine sites post-closure, HQ/HI values in excess of one are generally expected to undergo remediation and/or risk management.

The chance / probability for individuals involved in food collection to also develop cancer at some point in life through use of the site was estimated by multiplying the lifetime average daily intake doses from site use with oral and inhalation slope factors presented in **Section 5.6** based on the following calculation.

$$ILCR = LADD * SF$$

Where:

ILCR = Incremental Lifetime Cancer Risk, i.e. probability of developing cancer

LADD = Lifetime Average Daily Dose (mg/kg/day)

SF = Slope Factor, assumed linear relationship between LADD and cancer probability $(mg/kg/day)^{-1}$

In Yukon and BC, under the CSR ILCR values in excess of $1X10^{-5}$ (i.e. 1 in 100,000 chance of developing cancer through site exposure) are expected to undergo remediation and / or risk management.

The results of the risk calculations are presented in **Table 5-11** to **Table 5-14**. While hazard quotients / indices for non-cancer health effects (face edema and gastrointestinal symptoms) were all less than the regulatory benchmark value of one, incremental lifetime cancer risks for both the adult and toddler exceeded the regulatory benchmark value of $1X10^{-5}$ by a factor of roughly two. These cancer risks were predominantly associated with ingestion of food (plants), as opposed to direct soil / waste rock exposures. In addition, risks only exceeded the CSR benchmark value for food ingestion from country items grown on granite waste rock, given its higher arsenic concentrations. However, gneiss and shist waste rock food ingestion scenarios posed cancer risks only marginally below the acceptable $1X10^{-5}$ cancer risk benchmark, and there is moderate uncertainty around all of these risk calculations (i.e. risks could be higher or lower than modelled).

Table 5-11 Adult Arsenic Exposure – Hazard Indices

COPC and Exposure Medium					Hazard Index (HI) All Exposure Routes (∑ HQs)	
	Soil Ingestion HQ	Soil Dermal HQ	Dust Inhalation HQ	Food Ingestion HQ	HI – Oral TRV Based	HI – Inhalation TRV Based
Granite Waste Rock (50th percentile)						
Arsenic, Inorganic	6.7E-03	1.3E-03	4.2E-06	1.4E-01	**1.5E-01**	**4.2E-06**
Gneiss Waste Rock (50th percentile)						
Arsenic, Inorganic	1.5E-03	2.9E-04	9.3E-07	6.3E-02	**6.4E-02**	**9.3E-07**
Schist Waste Rock (50th percentile)						
Arsenic, Inorganic	1.4E-03	2.6E-04	8.6E-07	5.8E-02	**5.9E-02**	**8.6E-07**

Table 5-12 Adult Arsenic Exposure – Incremental Lifetime Cancer Risks (ILCR)

COPC and Exposure Medium					Total ILCR (∑ ILCRs)	
	Soil Ingestion ILCR	Soil Dermal ILCR	Dust Inhalation ILCR	Food Ingestion ILCR	ILCR – Oral TRV Based	ILCR – Inhalation TRV Based
Granite Waste Rock (50th percentile)						
Arsenic, Inorganic	1.7E-06	1.1E-07	5.5E-09	**2.1E-05**	**2.3E-05**	5.5E-09
Gneiss Waste Rock (50th percentile)						
Arsenic, Inorganic	3.8E-07	2.5E-08	1.2E-09	9.0E-06	9.4E-06	1.2E-09
Schist Waste Rock (50th percentile)						
Arsenic, Inorganic	3.5E-07	2.3E-08	1.1E-09	8.3E-06	8.7E-06	1.1E-09

Table 5-13 Toddler Arsenic Exposure – Hazard Indices

COPC and Exposure Medium					Hazard Index (HI) All Exposure Routes (∑ HQs)	
	Soil Ingestion HQ	Soil Dermal HQ	Dust Inhalation HQ	Food Ingestion HQ	HI – Oral TRV Based	HI – Inhalation TRV Based
Granite Waste Rock (50th percentile)						
Arsenic, Inorganic	1.1E-01	2.4E-03	9.0E-06	3.6E-01	4.8E-01	9.0E-06
Gneiss Waste Rock (50th percentile)						
Arsenic, Inorganic	2.5E-02	5.3E-04	2.0E-06	1.6E-01	1.8E-01	2.0E-06
Schist Waste Rock (50th percentile)						
Arsenic, Inorganic	2.3E-02	4.9E-04	1.8E-06	1.5E-01	1.7E-01	1.8E-06

Table 5-14 Toddler Arsenic Exposure – Incremental Lifetime Cancer Risks

COPC and Exposure Medium					Total ILCR (∑ ILCRs)	
	Soil Ingestion ILCR	Soil Dermal ILCR	Dust Inhalation ILCR	Food Ingestion ILCR	ILCR – Oral TRV Based	ILCR – Inhalation TRV Based
Granite Waste Rock (50th percentile)						
Arsenic, Inorganic	2.2E-06	4.7E-08	2.6E-09	**1.2E-05**	**1.4E-05**	2.6E-09
Gneiss Waste Rock (50th percentile)						
Arsenic, Inorganic	4.9E-07	1.0E-08	5.8E-10	5.1E-06	5.6E-06	5.8E-10
Schist Waste Rock (50th percentile)						
Arsenic, Inorganic	4.5E-07	9.5E-09	5.4E-10	4.7E-06	5.2E-06	5.4E-10

As indicated above, there is a moderate degree of uncertainty regarding the waste rock area arsenic exposure risk calculations. This uncertainty stems from uncertainty / variability around all of the various exposure assumptions used to quantify arsenic exposure (**Table 5-10**). However, the following three exposure assumptions likely introduce the greatest uncertainty into the above predictions of risk.

- Post-closure waste rock soil arsenic concentrations. 50th percentile concentrations of all exploratory drilling rock chemistry data were used in the risk calculations, under the premise that these would represent an average exposure concentration for a normally distributed range of concentrations and mobile human receptors. Post closure arsenic concentrations and exposure concentrations for the site as a whole, or in localized portions of the site could be higher or lower than this 50th percentile concentration.
- Food arsenic concentrations. Food concentrations were estimated using literature-based bioaccumulation models available for plants and small mammals. The regression equations for "General Estimates" were used. The authors of these models indicate they can also be adjusted to predict more conservative upper 95th percentile concentrations of arsenic in plants and small mammals. Such tissue concentrations would be slightly higher for small mammals but appreciably higher for plants, and would result in higher predicted arsenic cancer risks. There is also considerable uncertainty on whether these models accurately predict uptake from waste rock, can be used for berries or roots, and whether additional arsenic may accumulate on vegetation as a result of dust deposition onto foliage.
- Country foods harvest assumptions, in particular whether people will plausibly collect edible plants / berries or hunting small mammals from waste rock areas, and the amount of food items consumed on a daily, and lifetime basis. This HHRA used what we believed are reasonable, neither overly conservative nor under-conservative exposure assumptions to calculate risk. However, greater site use and harvesting than modelled would represent higher risks.

The above screening level calculations suggest that waste rock arsenic contamination may indirectly pose adverse risk to human health, largely attributed to ingestion of country foods (plants, berries, small mammals, etc.) growing on or foraging in uncovered waste rock disposal areas. Given such approach and conclusions risk management of waste rock arsenic contamination will likely be warranted at closure.

5.8 HUMAN INTAKE OF URANIUM IN SURFACE WATERS USED AS A POTABLE WATER SOURCE

As discussed in **Section 5.2.4**, the predicted maximum monthly concentrations of uranium in surface waters from some portions of Coffee Creek, Latte Creek, Halfway Creek and YT24 exceeded the Canadian drinking water guideline for uranium of 0.02 mg/L (or 20 µg/L). The Canadian "Maximum Acceptable Concentration" (MAC) for uranium was last revised in 1999, and is based on the occurrence of lesions in kidneys (which according to the then available experimental data may be rapidly reversible after exposure ceases).

A predicted maximum monthly uranium concentration of 0.033 mg/L for Latte Creek site CC1.5 and a concentration of 0.10 mg/L for YT24 are taken as highly conservative estimates of the uranium concentrations in local surface waters used for potable water. Additional exposure assumptions used in the assessment are provided below. As discussed in **Section 12.0 Surface Water Quality**, the creeks that interact with the Project exhibit naturally elevated concentrations of uranium, especially during low flow fall and winter months. The observed maximum uranium concentration for Latte Creek site CC1.5 was 0.032 mg/L, and for YT4 was 0.0028 mg/L.

Table 5-15 Exposure Assumptions for Human Ingestion of Uranium in Potable Water Sources from Project-area Creeks

Exposure Assumptions	Toddler	Adult	Ref
Surface water uranium concentration	33 µg/L and 100 µg/L	33 µg/L and 100 µg/L	
Exposure duration (days/week)	7/7	7/7	PJ
Exposure duration (weeks/year)	2/52	2/52	PJ
Drinking water ingestion rate (L/d)	0.6	1.5	HC (2012)
Body weight (kg)	16.5	70.7	HC (2012)

The estimates of human uranium exposures from use of site water for potable water based on these assumptions are tabulated below.

Table 5-16 Uranium Dose Estimates - Drinking Water Use Scenario

Assumed Uranium Concentration in Water Supply	Estimated Chronic Dose (µg • kg bw-1 d-1)	
	Toddlers	**Adults**
33 µg/L	0.046	0.027
100 µg/L	0.14	0.082

In this exposure calculation, the dose is amortized based on the expected number of days on site per year, but not based on the total number of years spent at the site relative to total life expectancy or some portion thereof.

Health Canada (2012) provides a human tolerable daily intake (TDI) for uranium of 0.6 µg kg bw^{-1} d^{-1} based on chemical (non-radiological) risks. As discussed in CCME (2007), the long half-life and slow rate of decay of uranium isotopes general results in chemical toxicity being a more important mode of toxic action than radiological toxicity, and thus chemical toxicity is likely to be a more sensitive toxicological mode of action for humans.

The ingestion of surface waters by humans to meet drinking water needs is not the only potential uranium exposure route: Additional exposure routes may include dust inhalation, incidental soil ingestion, dermal uptake and food ingestion. To account for this, only 20% of the TDI is allocated herein to the drinking water exposure pathway for the purpose of characterizing risk (i.e., 0.2 times 0.6 µg kg bw^{-1} d^{-1} = 0.12 µg kg bw^{-1} d^{-1} (adjusted TDI). This is consistent with Health Canada (2012) guidance.

The doses presented in **Table 5-17** are divided by the adjusted TDI to calculate risk quotients for uranium exposures via drinking water, as presented below.

Table 5-17 Drinking Water Estimated Risk Quotients

Assumed Uranium Concentration in Water Supply	Risk Quotient	
	Toddlers	**Adults**
33 µg/L	0.38	0.23
100 µg/L	1.2	0.68

A risk quotient that is substantially lower than 1.0 generally provides confidence that estimated contaminant exposure levels are lower than thresholds of health effects, whereas a risk quotient that approaches or exceeds a value of 1.0 suggests some potential for health risks in the absence of additional evidence to the contrary.

The risk quotients are based on highly conservative estimates of chronic exposures to uranium in surface waters affected by the project, since they are upper concentration estimates (not average water concentrations) for limited areas of the overall watershed of interest. In addition, only 20% of the estimated allowable dose is allocated to the drinking water pathway, while it is highly unlikely that the remaining exposures routes contribute an internalized uranium exposure that is 80% of the Health Canada (2012) TDI of 0.6 µg kg bw^{-1} d^{-1}. On the other hand, use of local creek water in the Project area for a period of greater than the assumed 2 weeks (14 days) per year would increase exposure estimates and risk quotients in proportion to the additional days spent obtaining water from the areas of interest.

Overall, this analysis suggests that health risks associated with drinking water ingestion of uranium in surface waters affected by the project are likely to be acceptably low. The management of water quality based on Project execution, however, should aim to prevent uranium concentrations over shorter duration summer-time periods from exceeding approximately 50 µg/L on average. Total uranium concentrations at some locations such as CC-1.0 (Latte Creek) routinely approach concentrations of 20 to 30 µg/L under summer-time, open flow conditions.

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APPENDIX A
TOXICITY PROFILES

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ACRONYMS AND ABBREVIATIONS

Acronym / Abbreviation	Definition
CAC	Criterion Air Contaminant
CO	Carbon monoxide
dB	Decibels: unit of sound measurement
dBA	Decibels: A-weighted sound characterizations
dm	Decimetre: one tenth of a metre, or ten centimetres
DPM	Diesel Particulate Matter
FAI	Focal Area(s) of Interest
HI	Hazard Index
HIA	Health Impact Assessment
HHRA	Human Health Risk Assessment
HQ	Hazard Quotient
ICP-OES	Inductively coupled plasma – optical emission spectrometry
ICP-MS	Inductively coupled plasma – mass spectrometry
ILCR	Incremental Lifetime Cancer Risk
km	kilometres
K_{OW}	Octanol-water partition co-efficient
LAA	Local Assessment Area
L_{EQ}	Equivalent noise level: a logarithmically averaged noise level over a set period such as 24-h
L_D	Daytime nose level: from 6:00 a.m. to 10:00 p.m.
L_{MAX}	Maximum noise level
L_N	Night-time noise level: from 10:00 p.m. to 6:00 a.m.
MSAT	Mobile Source Air Toxics
MPC	Maximum Permissible Concentration
NO	Nitrogen oxide
mg/dm2	Unit of dustfall or particulate deposition: milligrams per square decimeter.
NO_2	Nitrogen dioxide
NO_X	Nitrogen oxides including NO and NO_2
$PM_{2.5}$	Fine particulate matter with an aerodynamic particle diameter less than 2.5 µm
PM_{10}	Fine particulate matter with an aerodynamic particle diameter less than 10 µm
RAA	Regional Assessment Area
RfD	Reference Dose
RQ	Risk Quotient
SO_2	Sulfur dioxide
TPAH	Total Polycyclic Aromatic Hydrocarbons
TSP	Total Suspended Particulates

Acronym / Abbreviation	Definition
TVOCs	Total Volatile Organic Compounds
µg/m^3	Unit of concentration in air: micrograms per cubic meter or air
µm	micrometer
VOCs	Volatile Organic Contaminants
%HA	Percent Highly Annoyed: sound health effects indicator

A-1 INTRODUCTION

Hazard identification is the process in which the potential adverse health effects resulting from exposure to a chemical agent are determined. The outcome of this process is the identification of a safe exposure level at which adverse health effects are not predicted to occur: that is, an exposure limit. This appendix describes the exposure limits identified for the assessment of human health effects associated with exposure to chemicals identified in Project emissions. Exposure limits specific to acute and chronic inhalation were reviewed for all chemicals identified in Project emissions.

A-1.1 EXPOSURE LIMIT SELECTION

The Kaminak Coffee Gold Mine Project (Coffee Project or Project) is a proposed gold development project in west-central Yukon, approximately 130 kilometres (km) south of Dawson City. The Project is located on Crown Land within the traditional territory of Tr'ondëk Hwëch'in and the asserted area of White River First Nation. A portion of Kaminak's claim block is located in Selkirk First Nation's traditional territory. The Project is scoped as an open pit gold mine using a cyanide heap leach process to extract ore. It would consist of an 18-month construction period, followed by a 10-year mine life with an average operation rate of five million tonnes per annum of heap leach feed, producing 1.9 million ounces of gold over the life of the mine.

It is important to consider how the Project, through construction, operations, closure and post-closure could influence the health and well-being of people who use the land and frequent areas in the vicinity of the proposed minesite or mine access route. Both a formalized human health risk assessment (HHRA) and health impact assessment (HIA) were completed to provide a better understanding about how the Project might affect human (and community) health and well-being. The HIA Technical Report is provided under separate cover, while the purpose of this Technical Report is to document the scope, methods, results and conclusion of a quantitative HHRA for the proposed Project. Collectively, the HIA and HHRA form the basis for evaluating the effects of the Project on community health and well-being.

A major reason for undertaking a HHRA is to identify those circumstances where risks to the health of individuals and larger groups of people cannot be confidently discounted, and to develop appropriate risk management approaches for the purpose of preventing adverse health outcomes. Conclusions about risk that arise from this HHRA are an important step for identifying any potential need for risk management actions, or mitigations, against any adverse health outcomes.

A-1.2 HUMAN HEALTH RISK ASSESSMENT VERSUS HEALTH IMPACT ASSESSMENT

HHRA is a tool that is useful for examining how changes in environmental quality (for example, air quality, environmental noise, water quality, soil quality) could influence human health. The underlying premise is that changes in environmental quality can drive changes in the characteristics and magnitude of human exposures to stressors such as noise, or chemicals such as those trace elements present at atypically high concentrations in mine wastes or ore.

A-2 ACETALDEHYDE

A-2.1 INHALATION EXPOSURE LIMITS

A-2.1.1 ACUTE INHALATION

Table A-2.1-1 Acute Inhalation Exposure Limits for Acetaldehyde

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	1-hour REL 8-hour REL	470 300	Respiratory irritation Nasal irritation	Human Rat	Prieto et al. 2000 Appelman et al. 1982; 1986	OEHHA 2014; 2008
TCEQ	1-hour ESL	15	Odour	-	-	TCEQ 2014

- not available

The OEHHA (2014) recommend a 1-hour REL of 470 µg/m^3 for acetaldehyde. This REL was derived from responses observed in asthmatic individuals following controlled, short-term (2-5 minutes) exposures to acetaldehyde (Prieto et al. 2000). A LOAEL of 142 mg/m^3 for bronchoconstriction was identified from this study. The OEHHA (2008) applied a 300-fold uncertainty factor to this LOAEL account for use of a LOAEL (10), variability in human response (√10) and potential asthma exacerbation in children (10). This exposure limit was determined by the OEHHA (2008) to also be protective of potential eye irritation associated with acute exposure to acetaldehyde, following review of another controlled exposure study in humans (Silverman et al. 1946).

An 8-hour REL of 300 µg/m^3 is also recommended for acetaldehyde by the OEHHA (2014). This REL was derived from a NOAEL of 270 mg/m^3 for the degeneration of olfactory epithelium in rats intermittently exposed (6 hours/day, 5 days/week) to acetaldehyde over a 4-week period (Appelman et al. 1982; 1986). The 8-hour REL was not considered for the acute exposure assessment as it was based on a subchronic exposure study and is intended for repeated 8-hour exposures.

The TCEQ (2014) recommend an acute (1-hour) ESL of 15 µg/m^3 for acetaldehyde based on odour; no supporting documentation was provided for this-ESL.

The OEHHA 1-hour REL of 470 µg/m^3 was considered the most appropriate health-based guideline for the assessment of acute exposure to acetaldehyde as it was based on acute responses in humans and considered sensitive individuals. Although considered protective of eye and nasal irritation, the exposure

limit was specific to respiratory irritation and therefore acetaldehyde was only included in the chemical group for respiratory irritation following acute inhalation exposures.

A-2.1.2 CHRONIC INHALATION

Table A-2.1-2 Chronic Inhalation Exposure Limits for Acetaldehyde

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	TC RsC	390 17	Nasal lesions Nasal tumours	Rat	Appelman et al. 1982; 1986 Woutersen et al. 1986	Health Canada 2000
METRO VANCOUVER	-		-	-	-	MV 2011
OEHHA	REL RsC	140 3.7	Nasal lesions Nasal tumours	Rat	Appelman et al. 1982; 1986 Woutersen et al. 1986	OEHHA 2014; 2011; 2008
RIVM	-	-	-	-	-	RIVM 2001
TCEQ	ESL	45	-	-	-	TCEQ 2014
US EPA	RfC RsC	9 5	Nasal lesions Nasal tumours	Rat	Appelman et al. 1982; 1986 Woutersen and Appleman 1984	US EPA 1991
WHO	-	-	-	-	-	WHO 2000

- not available

IARC (2014) has classified acetaldehyde as possibly carcinogenic to humans (Group 2B). Health Canada (2000), the OEHHA (2011) and US EPA (1991) have established chronic inhalation guidelines based on evidence in rats of an association between chronic inhalation exposure to acetaldehyde and nasal tumours (Woutersen et al. 1986; Woutersen and Appleman, 1984). These agencies also established guidelines based on nasal lesions in rats (Appelman et al. 1982; 1986) following acetaldehyde inhalation.

Health Canada (2000) developed a TC of 390 µg/m^3 for the noncarcinogenic effects of acetaldehyde following chronic inhalation exposure. This guideline was based on the Appelman et al. (1982; 1986) inhalation studies reporting nasal lesions in rats intermittently exposed (6 hours/day, 5 days/week) to acetaldehyde over a 4-week period. The THRESH program (Howe 1995) was used to calculate a BMC_{05} of 218 mg/m^3 for non-neoplastic lesions in the nasal olfactory epithelium of male rats. The BMC_{05} was adjusted for continuous exposure (6h/24h, 5d/7d) and an uncertainty factor of 100 applied to account for extrapolation from an animal study (10) and variability in human response (10) (Health Canada 2000).

An additional uncertainty factor to account for use of a short term study was not considered appropriate based on evidence that there was *no indication that severity of the critical effects increases with duration of exposure* (Health Canada 2000).

A TC_{05} of 86 mg/m^3 was also recommended by Health Canada (2000) based on the incidence of tumours in the nasal cavity of rats exposed to acetaldehyde for 6 hours/day, 5 days/week over 28 months (Woutersen et al., 1986). The TC_{05} was derived using multistage modeling and adjusted for continuous exposure (Health Canada 2000). The TC_{05} is associated with a 5% increase in tumour incidence over background. Dividing the TC_{05} by a factor of 5,000 results in an RsC of 17 µg/m^3 for a 1 in 100,000 incremental cancer risk level.

The OEHHA (2014) recommends an REL of 140 µg/m^3 for the noncarcinogenic effects of acetaldehyde following chronic inhalation exposure. This REL was derived from the same rat inhalation studies (Appelman et al. 1982; 1986) identified for the 8-hour OEHHA REL. A study NOAEL of 270 mg/m^3 for degeneration of olfactory epithelium was identified. The OEHHA (2008) applied benchmark modelling (US EPA 2003) to determine a BMC_{05} of 178 mg/m^3 for the incidence of degeneration of olfactory epithelium. The BMC_{05} was converted to a human equivalent concentration of 242 mg/m^3 using a pharmacokinetic model specific to acetaldehyde (Teeguarden et al. 2008) and adjusted for continuous exposure to result in a BMC_{05HEC} of 43.2 mg/m^3. A cumulative uncertainty factor of 300 was applied to account for subchronic exposure (√10), extrapolation from an animal study (√10), variability in human response (√10) and potential asthma exacerbation in children (10) (OEHHA 2008).

A unit risk factor of 0.0000027 per µg/m^3 was recommended for acetaldehyde by the OEHHA (2011). Similar to Health Canada, this risk factor was calculated from the incidence of nasal tumours in rats (obligate nose breathers) following exposure to acetaldehyde for 6 hours/day, 5 days/week and up to 28 months (Woutersen et al. 1986). However, the OEHHA (2011) also applied an interspecies surface area correction factor (based on relative bodyweight) to account for potential exposure of the entire human respiratory tract, including the lung. The unit risk factor translates to an RsC of 3.7 µg/m^3, assuming an acceptable incremental cancer risk of 1 in 100,000.

The TCEQ (2014) recommends a chronic ESL of 45 µg/m^3 for acetaldehyde; however, no supporting documentation was provided for this exposure limit.

The US EPA (1991) recommends an RfC of 9 µg/m^3 for acetaldehyde based on noncarcinogenic effects following chronic exposure. A NOAEL of 273 mg/m^3 for degeneration of olfactory epithelium was identified from the Appelman et al. (1982; 1986) studies. The US EPA adjusted the NOAEL for continuous exposure

and calculated a $NOAEL_{HEC}$ of 8.7 mg/m^3 for a gas:respiratory effect in the extra thoracic region. A 1,000-fold uncertainty factor was applied to the $NOAEL_{HEC}$ to account for use of a subchronic study (10), extrapolation from an animal study/ incompleteness of the database (10) and variability in human response (10).

The US EPA (1991) recommends a unit risk factor of 0.0000022 per µg/m^3 for acetaldehyde. This risk factor was calculated from the incidence of nasal tumours in rats following exposure to acetaldehyde for 6 hours/day, 5 days/week for 27 months (Woutersen and Appleman 1984). The unit risk factor translates to an RsC of 5 µg/m^3 assuming a 1 in 100,000 incremental cancer risk.

The TC of 390 µg/m^3 recommended by Health Canada was selected for the evaluation of noncarcinogenic effects following chronic inhalation exposure to acetaldehyde. The Health Canada TC was considered more appropriate than the exposure limits recommended by the US EPA and OEHHA as the TC was developed using benchmark modelling to determine a POD (unlike the US EPA) and the study on which all of these guidelines were based did not indicate that an additional safety factor for use of a subchronic study (as assigned by the US EPA and OEHHA) was warranted. Acetaldehyde was included in the chemical group for nasal irritation following chronic inhalation exposures.

The RsC of 3.7 µg/m^3 (OEHHA) was selected for the assessment of carcinogenic effects following chronic inhalation exposure to acetaldehyde. Both the OEHHA and Health Canada selected results from the more recently published Woutersen et al. (1986) study for the determination of a unit risk factor. The OEHHA accounted for the fact that humans are not obligate nose breathers and adjusted the unit risk estimate to take into account the greater surface area of the human respiratory tract. Acetaldehyde was included in the chemical group for nasal tumours following chronic inhalation exposures.

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A-3 ACROLEIN

A-3.1 INHALATION EXPOSURE LIMITS

A-3.1.1 ACUTE INHALATION

Table A-3.1-1 Acute Inhalation Exposure Limits for Acrolein

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	1-hour MRL	7	Decreased respiratory rate; respiratory tract irritation	Human	Weber-Tschopp et al. 1977	ATSDR 2013; 2007
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	1-hour REL 8-hour REL	2.5 0.7	Eye irritation Respiratory irritation	Human Rat	Darley et al. 1960; Weber-Tschopp et al. 1977; Dorman et al., 2008	OEHHA 2014; 2008
TCEQ	1-hour ReV	11	Decreased respiratory rate; eye and respiratory tract irritation	Human	Weber-Tschopp et al. 1977	TCEQ 2014
US EPA	-	-	-	-	-	US EPA 2003a

- not available

The ATSDR (2013), OEHHA (2014) and TCEQ (2014) all recommend 1-hour exposure limits for acrolein based on eye, nasal and respiratory irritation reported in controlled human exposure studies (Weber-Tschopp et al. 1977; Darley et al. 1960). The US EPA does not recommend an acute exposure limit for acrolein but cites the clinical study by Weber-Tschopp et al. (1977) as the most comprehensive for describing the acute effects of acrolein inhalation in humans (US EPA 2003a).

The ATSDR (2013) 1-hour MRL for acrolein is 7 µg/m^3. The MRL was based on a LOAEL of 0.3 ppm (700 mg/m^3) for decreased respiratory rate as well as nose and throat irritation in human volunteers exposed to acrolein for 60 minutes (Weber-Tschopp et al. 1977). An uncertainty factor of 100 was applied to the LOAEL to account for use of a LOAEL (10) and variation in human response (10) (ATSDR 2007).

The OEHHA (2014) recommend a 1-hour REL of 2.5 µg/m^3 for acrolein. The OEHHA (2008) identified a LOAEL of 0.06 ppm (140 µg/m^3) for eye irritation in human volunteers following short-term (5-minutes) exposure to acrolein (Darley et al. 1960). Ocular irritation was first reported by Weber-Tschopp et al. (1977) during 40 minutes exposure to increasing concentrations of acrolein at a similar LOAEL (0.07 ppm or

160 µg/m^3). Acute REL values of 2.3 and 2.7 µg/m^3 were determined for each study LOAEL after an uncertainty factor of 60 was applied to account for use of a LOAEL for a mild effect (6) and variation in human response (10). No time adjustment was made to the RELs as the critical effect was a sensory irritancy effect. The geometric mean of the REL values from these studies (i.e., 2.5 µg/m^3) was selected as the 1-hour REL for acrolein (OEHHA 2008).

An 8-hour REL of 0.7 µg/m^3 was also recommended for acrolein by the OEHHA (2014). This REL was based on a subchronic study where a NOAEL of 0.2 ppm (465 µg/m^3) was identified for lesions in the respiratory epithelium of rats exposed to acrolein 6 hours/day, 5 days/week over 65 days (Dorman et al. 2008). The 8-hour REL for acrolein was not considered for the acute exposure assessment as it was based on subchronic exposure data in animals and is intended for repeated 8-hour exposures.

An acute ReV of 11 µg/m^3 is recommended for acrolein by the TCEQ (2014). Similar to the ATSDR (2007), the TCEQ (2010) identified a LOAEL of 0.3 ppm (700 mg/m^3) for eye, nose, throat irritation and decreased respiratory rate in human volunteers exposed for 60 minutes to acrolein (Weber-Tschopp et al. 1977). An uncertainty factor of 63 was applied to the LOAEL to account for use of a LOAEL (6.3) and variation in human response (10) (TCEQ 2010).

The 1-hour exposure limit of 2.5 µg/m^3 (OEHHA, 2008) was selected for the assessment of acute exposure to acrolein as it was based on the most sensitive human response (eye irritation) to acute acrolein exposure and supported by more than 1 study. This acute exposure limit for acrolein is considered very conservative. As described below, the limit identified for chronic exposure to acrolein, based on nasal lesions, is very similar to this 1-hour exposure limit. Although protective of nasal and respiratory irritation, the 1-hour exposure limit was specific to eye irritation and therefore acrolein was only included in the chemical group for eye irritation following acute inhalation exposures.

A-3.1.2 CHRONIC INHALATION

Table A-3.1-2 Chronic Inhalation Exposure Limits for Acrolein

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2012
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	TC	0.4	Nasal Lesions	Rat	Cassee et al. 1996	Health Canada 2000
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	REL	0.35	Nasal Lesions	Rat	Dorman et al. 2008	OEHHA 2013; 2008

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
RIVM	-	-	-	-	-	RIVM 2001
TCEQ	ReV	2.7	Nasal Lesions	Rat	Dorman et al. 2008	TCEQ 2014
US EPA	RfC	0.02	Nasal Lesions	Rat	Feron et al. 1978	US EPA 2003b
WHO	-	-	-	-	-	WHO 2000

- not available

Health Canada (2000) recommends a TC of 0.4 µg/m^3 for chronic exposure to acrolein. The THRESH program (Howe 1995) was used to calculate a BMC_{05} of 0.14 mg/m^3, the air concentration representing a 5% increase in the incidence of nasal lesions in rats following inhalation (nose-only) exposure to acrolein for 6 hours/day over a 3 day period (Cassee et al. 1996). The BMC_{05} was adjusted for continuous exposure and an uncertainty factor of 100 applied to account for use of an animal study (10) and variability in human response (10). No uncertainty factor was applied for less than chronic exposure as

Health Canada (2000) noted the degenerative changes observed by Cassee et al. (1996) following short-term exposures were consistent with observations in longer term bioassays in rats (Feron et al. 1978) and hamsters (Feron and Kruysse, 1977).

The OEHHA (2014) recommends a chronic REL of 0.35 µg/m^3 for acrolein. Similar to the 8-hour REL, the chronic REL was based on a NOAEL of 0.2 ppm (465 µg/m^3) for lesions in the respiratory epithelium of rats exposed to acrolein 6 hours/day, 5 days/week for 13 weeks (Dorman et al. 2008). The OEHHA (2008) calculated a $NOAEL_{HEC}$ of 0.03 ppm (70 µg/m^3) after adjusting the NOAEL for continuous exposure and applying a dosimetric adjustment factor (DAF) of 0.85 based on comparative modeling of gas flux in human and rat nasal passages with formaldehyde. An uncertainty factor of 200 was applied to account for extrapolation from an animal study (√10), use of a subchronic study (√10), variability in human response (10) and use of a DAF for formaldehyde, an analogue chemical, to determine the human exposure concentration for acrolein (2).

The TCEQ (2014) recommends an ReV of 2.7 µg/m^3 for chronic exposure to acrolein. Similar to the OEHHA (2008), this guideline was based on a NOAEL of 0.2 ppm (465 µg/m^3) for hyperplasia of the respiratory epithelium of rats exposed to acrolein 6 hours/day, 5 days/week for 13 weeks (Dorman et al. 2008). The study investigated duration and concentration effects for several exposure groups and evaluated the histopathology and recovery of the respiratory tract post-exposure. The TCEQ (2014) calculated a NOAEL(HEC) of 35.7 ppb (83 µg/m^3) for acrolein after adjusting the NOAEL for continuous exposure. An uncertainty factor of 30 was applied to the NOAEL(HEC) account for extrapolation from an animal study (3) and variability in human response (10). No adjustment was made for use of a subchronic

response as the TCEQ (2014) concluded that concentration played more of a role in the irritant effects of acrolein than duration of exposure. Unlike the OEHHA, the TCEQ (2014) did not consider the use of a DAF based on formaldehyde appropriate for acrolein due to differences in nasal dosimetry patterns for acrolein and formaldehyde and did not apply an additional 2-fold uncertainty factor for use of a DAF.

The US EPA (2003b) recommends an RfC of 0.02 µg/m^3 for acrolein based on a LOAEL of 0.9 mg/m^3 for nasal lesions in rats exposed to acrolein for 5 days/week over 13 days (Feron et al. 1978). The US EPA calculated a LOAEL(HEC) of 0.02 mg/m^3 after adjusting for continuous exposure and applied a 1000-fold uncertainty factor to account for use of a minimal LOAEL (3), use of a subchronic study (10), extrapolation from an animal study (3) and variability in human response (10). The US EPA selected the Feron et al. (1978) study over the Cassee et al. (1996) selected by Health Canada, based on the reporting of results for a higher number of test animals (including both sexes of rats, hamsters and rabbits), a longer exposure duration, and better characterization of multiple endpoints and the dose-response by Feron et al. (1978).

The US EPA (2003b) recommend the lowest chronic inhalation guideline for acrolein, however, this guideline was based on an older study which identified a LOAEL which required a higher uncertainty factor. The OEHHA and TCEQ identified guidelines for acrolein based on the most recent study for nasal irritation in rats which identified a NOAEL for nasal lesions (Dorman et al. 2008), however the OEHHA REL included use of a DAF that is not considered relevant to acrolein (TCEQ 2014). Therefore, the ReV of 2.7 µg/m^3 recommended by the TCEQ (2014) was considered the most appropriate for the assessment of chronic inhalation exposure to acrolein. Acrolein was included in the chemical group for nasal irritation following chronic inhalation exposures.

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A-4 BENZENE

A-4.1 INHALATION EXPOSURE LIMITS

A-4.1.1 ACUTE INHALATION

Table A-4.1-1 Acute Inhalation Exposure Limits for Benzene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	24-hour MRL	30	Haematological / Immunological	Mice	Rozen et al 1984	ATSDR 2013; 2007
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	6-hour REL	1,300	Reproductive / developmental toxicity	Rats	Coate et al 1984	OEHHA 1999a; 2014a
TCEQ	1-hour ReV	580	Haematological/ Immunological	Mice	Rozen et al 1984	TCEQ 2007
US EPA	-	-	-	-	-	US EPA 2002

- not available

The ATSDR recommend an acute (24-hour) MRL of 30 µg/m^3 for benzene (ATSDR 2013). This MRL is based on an observed decrease in mitogen-induced lymphocyte proliferation following the exposure of mice to benzene vapours for 6 hours per day over a 6-day period (Rozen et al 1984). The study LOAEL of 10.2 ppm (33 mg/m^3) was adjusted from intermittent to 24-hour exposure and converted to an human equivalent concentration (HEC) of 2.55 ppm (8 mg/m^3) using US EPA (1994) methodology for the extrarespiratory effects of a category 3 gas. The 24-hour $LOAEL_{HEC}$ was divided by a 300-fold uncertainty factor to account for use of a LOAEL (10), extrapolation from animals (3) to humans and human variability (10) (ATSDR 2007).

The OEHHA (2014a) recommend an acute REL of 1,300 for 6-hour exposure to benzene. This REL was derived from a study of developmental toxicity in rats conducted by Coate et al. (1984). The study addressed the most sensitive noncancer endpoint associated with benzene inhalation which was lowered fetal body weights in offspring following dam exposure for 6 hours/day on gestational days 6 to 15 (OEHHA 1999a). It is noted the OEHHA reference exposure levels for benzene are currently under review and a 1-hour REL based on haematological effects in mice (Keller and Snyder et al. 1988) is being proposed (OEHHA 2014b).

The TCEQ (2007) developed a 1-hour ReV of 580 µg/m^3 for benzene using the same study and LOAEL identified by the ATSDR. The hematotoxic effects observed in the Rozen et al. 1984 study were supported by two additional studies in mice (Dempster and Snyder 1991; Corti and Snyder, 1996). The TCEQ (2007) converted the LOAEL of 10.2 ppm (33 mg/m^3) to a 1-hour HEC of 18.5 ppm (59 mg/m^3) which was then

divided by a 100-fold uncertainty factor to account for use of a LOAEL (3), extrapolation from animals to humans (3) and human variability (10).

The US EPA do not recommend an acute exposure limit for benzene but do cite a variety of animal studies examining the acute effects of benzene inhalation which confirm that acute exposure to high benzene concentrations results in hematotoxic effects, with a greater sensitivity observed in mice over rats (US EPA 2002).

The TCEQ 1-hour ReV of 580 µg/m^3 was selected for the current assessment of acute exposure to benzene as the effect of benzene on lymphocyte response in mice was supported by several studies and the 1-hour exposure duration selected by the TCEQ was considered the most appropriate for the response observed.

A-4.1.2 CHRONIC INHALATION

Table A-4.1-2 Chronic Inhalation Exposure Limits for Benzene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	MRL	9.8	Haematological	Human	Lan et al 2004	ATSDR 2013; 2007
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	RsC	3	Leukemia	Human	Rinsky et al 1987	Health Canada 2010
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	REL RsC	60 0.3	Haematological Leukemia	Human	Tsai et al 1983 Rinsky et al 1981	OEHHA 1999b; 2011; 2014a
RIVM	CR (adjusted)	2	Leukemia	Human	Adopted from WHO 2000	RIVM 2001
TCEQ	ReV ESL	280 4.5	Haematological Leukemia	Human	Rothman et al 1996 Rinsky et al 1981; 1987	TCEQ 2007
US EPA	RfC RsC	30 1.3 to 4.5	Haematological Leukemia	Human	Rothman et al. 1996 Rinsky et al 1981; 1987	US EPA 2003; 2000
WHO	RsC	1.7	Leukemia	Human	Crump and Allen, 1984; Rinsky et al. 1987; Paustenbach et al. 1992	WHO 2000

- not available

IARC (2014) has classified benzene as carcinogenic to humans (Group 1). With the exception of ATSDR, all the regulatory agencies reviewed have established chronic inhalation guidelines based on epidemiological evidence of an association between chronic occupational exposure to benzene and leukemia mortality rates. The ATSDR, OEHHA, TCEQ and US EPA have also established chronic inhalation guidelines based on haematological/immunological effects (i.e., lymphocyte response).

The ATSDR (2013) recommend a chronic MRL of 9.8 µg/m^3 for benzene. The MRL was derived from a study of workers in Chinese shoe manufacturing industries (Lan et al 2004) which reported an exposure-response relationship between benzene exposure levels (measured by individual vapour monitors) and decreased lymphocyte (B cell) count in workers exposed an average of 6.1 years (ATSDR 2007). A $BMCL_{0.25sd}$ of 0.10 ppm (0.33 mg/m^3), representing the lower 95% confidence limit for a 0.25 standard deviation reduction below the control mean B cell count, was identified as the point of departure (POD) for the chronic MRL. The $BMCL_{0.25sd}$ was adjusted for continuous exposure and an uncertainty factor of 10 applied for human variability to result in a chronic MRL of 0.003 ppm (0.0098 mg/m^3).

Health Canada (2010) derived a TC_{05} of 15 mg/m^3 for benzene based on the incidence of mortality from leukemia in a cohort of rubber hydrochloride (pliofilm workers) (Rinsky et al. 1987). The exposure concentration associated with a 5% increase in mortality from acute myelogenous leukemia (TC05) was derived using cancer potencies based on exposure estimates of Crump and Allen (1984) as described in Health Canada (1993). When divided by 5,000 the TC_{05} translates to an RsC of 3 µg/m^3 for a 1 in 100,000 incremental increase in mortality from acute myelogenous leukemia.

An REL of 60 µg/m^3 was derived by OEHHA (2014a) for chronic exposure to benzene. This REL was based on haematological effects following occupational exposure of a cohort of 454 male petroleum refinery workers exposed to benzene (personal monitors) over an average for 7.4 years (Tsai et al. 1983). Again, the OEHHA reference exposure levels for benzene are currently under review and a chronic REL based on haematological effects in Chinese shoe workers (Lan et al 2004), is being proposed (OEHHA 2014b).

The OEHHA (2011) also recommend a unit risk factor of 0.000029 per µg/m^3 for benzene based on mortality from leukemia in pliofilm workers as reported by Rinsky et al (1981) using a weighted cumulative exposure/relative risk procedure by CDHS (1984). This unit risk factor translates to a RsC of 0.3 µg/m^3 for a 1 in 100,000 incremental increase in mortality from leukemia.

The RIVM (2001) has established a CR of 20 µg/m^3 for benzene assuming an excess cancer risk of 1 in 10,000. This was divided by 10-fold to determine an air concentration of 2 µg/m^3 for an excess cancer (leukemia) risk of 1 in 100,000 for comparison with other agencies. The RIVM (2001) adopted the lower limit of the EU (1999) cancer risk estimates for chronic exposure to benzene, which is equivalent to the unit risk recommended by the WHO (2000).

An ReV of 280 µg/m^3 is recommended for benzene by the TECQ (2007). This guideline is based on hematotoxic effects (reduced lymphocyte count) in Chinese workers occupationally exposed to benzene for an average of 6.3 years (Rothman et al 1996). The critical effect of decreased lymphocyte count is supported by the results of Lan et al (2004) for workers in Chinese shoe manufacturing industries exposed to benzene for an average of 6.1 years (TCEQ 2007). The TCEQ (2007) derived a benchmark concentration (BMC) of 8.4 mg/m^3 (adjusted for continuous exposure) from the Rothman et al (1996) study to which an uncertainty factor of 30 was applied to account for human variability (10) and a lack of data for reproductive/developmental effects (3).

The TCEQ (2007) also recommend a chronic ESL ($^{chronic}ESL_{linear(c)}$) of 4.5 µg/m^3 for an excess lifetime cancer risk of 1 in 100,000 following chronic exposure to benzene. This air concentration was derived using the cancer potency estimates of Crump and Allen (1994) for acute myelogenous leukemia in the pliofilm cohort described by Rinsky et al. (1981; 1987).

The US EPA (2003) recommends a RfC of 30 µg/m^3 for benzene. This RfC was based on the effect of reduced absolute lymphocyte count in Chinese workers reported in the Rothman et al (1996) study. A BMC of 8.2 mg/m^3 was calculated and adjusted by an uncertainty factor of 300 to account for human variability (10), extrapolating from a LOAEL to a NOAEL (3), extrapolating from subchronic to chronic exposure (3) and database uncertainties (3) (US EPA 2002).

The US EPA (2000) also recommends unit risk factors for benzene based on the incidence of acute myelogenous leukemia reported in workers exposed to benzene. Air concentrations recommended for benzene at a 1 in 100,000 cancer risk level range from 1.3 to 4.5 µg/m^3 and were determined from the pliofilm cohort described by Rinsky et al. (1981; 1987) using risk calculations recommended by Paustenbach et al. (1993); Crump and Allen (1984); Crump (1994) and U.S. EPA (1998).

The WHO (2001) recommend an air quality of guideline of 1.7 µg/m^3 for an excess lifetime cancer (leukemia) risk of 1 in 100,000 following chronic exposure to benzene. This guideline was derived from a range of studies reporting risk estimates for mortality from leukemia in the pliofilm cohort of workers (Crump and Allen, 1984; Rinsky et al. 1987; Paustenbach et al. 1992).

The lowest air concentration recommended by the ATSDR (2007) for haematological/immunological effects (9.8 µg/m^3) was selected for the assessment of non-carcinogenic effects following chronic inhalation exposure to benzene.

The range of air concentrations identified by the US EPA (2003) for a 1 in 100,000 cancer risk level is supported by similar exposure limits derived by Health Canada, RIVM, TCEQ and WHO for the same response (i.e., leukemia). An important distinction of the Health Canada guideline was the identification of the exposure concentration associated with mortality from, rather than incidence of, leukemia. For the purpose of this assessment, the lowest air concentration recommended by the US EPA (1.3 µg/m^3) was selected for the evaluation of potential carcinogenic effects following chronic inhalation exposure to benzene. Benzene was included in the chemical group for leukemia following chronic inhalation exposures.

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A-5 BENZO[A]PYRENE

A-5.1 INHALATION EXPOSURE LIMITS

A-5.1.1 ACUTE INHALATION

IARC (2014) has classified benzo[a]pyrene (B[a]P) as carcinogenic to humans (Group 1). Studies on the carcinogenic potential of B[a]P and mixtures of polycyclic aromatic hydrocarbons (PAHs) following chronic inhalation or oral exposures are outlined in ATSDR (1995); Health Canada (2010); RIVM (2001); and US EPA (1994).

Table A-5.1-1 Acute Inhalation Exposure Limits for B[a]P

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	-	-	-	-	-	OEHHA 2014
TCEQ	-	-	-	-	-	TCEQ 2014
WHO	-	-	-	-	-	WHO 2000

- not available

The effects of acute inhalation exposure to B[a]P have not been characterized and no acute exposure limits with supporting documentation were identified. As a C20 aromatic hydrocarbon B[a]P has extremely low volatility and inhalation of the chemical in isolation from particulate matter is unlikely. Controlled inhalation and intratracheal instillation studies in animals have demonstrated the carcinogenicity of B[a]P over long-term (chronic) exposure periods as described below.

A-5.1.2 CHRONIC INHALATION

Table A-5.1-2 Chronic Inhalation Exposure Limits for B[a]P

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	RsC	0.32	Respiratory tract tumours	Hamsters	Thyssen et al., 1981	Health Canada 2010
METRO VANCOUVER	-	-	-	-	-	MV 2011

Agency	Exposure Limit Type	Exposure Limit Value (µg/m³)	Critical Organ or Effect	Species	Study	Source
OEHHA	RsC	0.009	Respiratory tract tumours	Hamsters	Thyssen et al., 1981	OEHHA 2011
RIVM	-	-	-	-	-	RIVM 2001
TCEQ	-	-	-	-	-	TCEQ 2014
US EPA	-	-	-	-	-	US EPA 1994
WHO	RsC	0.00012	Lung cancer	Human	Redmond, 1976; US EPA 1984	WHO 2000; 1987

- not available

Health Canada (2010) developed a unit risk factor of 0.031 per µg/m^3 for B[a]P. This unit risk factor was determined using multistage modeling of the tumour incidence in the respiratory tract of hamsters exposed by inhalation (nose only) to B[a]P for 4.5 hours/day, 7 days/week during the first 10 weeks of the study and 3 hours/day, 7 days/week for the remainder of the study (up to 96 weeks) (Thyssen et al. 1981). This unit risk factor translates to an RsC of 0.32 µg/m^3 based on a 1 in 100,000 excess lifetime cancer risk.

A unit risk factor of 0.0011 per µg/m^3 was derived for B[a]P by the OEHHA (2011). Similar to Health Canada, the linearized multistage model was fit to respiratory tract tumour data in hamsters as reported by Thyssen et al. (1981). The OEHHA (2011) further calculated an oral risk factor (0.43 per mg/kg body weight/day) based on the exposure conditions described in the study and the inhalation rate and body weight of hamsters. A human equivalent unit risk factor for the inhalation pathway was then determined by applying an interspecies surface area correction factor, based on bodyweight and surface area, to the oral risk factor. The resulting OEHHA (2011) unit risk factor corresponds to an RsC of 0.009 µg/m^3 for a 1 in 100,000 excess lifetime cancer risk. It is noted that while the bodyweight scaling approach selected by the OEHHA (2011) is consistent with US EPA (2005) cancer risk assessment guidance for oral exposures, the EPA does not recommend this approach for determining human equivalent exposures for the inhalation pathway.

The US EPA (1994) does not currently recommend an inhalation unit risk estimate for B[a]P, however, the potential inhalation toxicity of B[a]P is currently under review by the US EPA with a draft human health assessment released August 2013 for independent peer review and public comment (US EPA 2013).

The WHO (1987; 2000) selected B[a]P as an indicator of the carcinogenic potential of PAH mixtures in air and developed a unit risk factor of 0.0087 per µg/m^3 using a linearized multistage model and epidemiological data for mortality due to lung cancer in workers exposed to mixtures of PAH in coke-oven emissions (Redmond 1976; US EPA 1984). Using this unit risk factor, a B[a]P air concentration of 0.00012 µg/m^3 would be associated with a 1 in 100,000 increased risk of mortality as a result of lung cancer. The WHO (1987; 2000) guideline for B[a]P represents an index of PAH mixtures from coke oven emissions and similar

combustion processes. The WHO (2000) noted that although the PAH composition in coke-oven emissions may not correlate to PAH in ambient air, epidemiological studies involving other PAH mixtures have determined similar cancer risks and a unit risk within the same order of magnitude was determined for B[a]P from animal data (i.e., Heinrich et. al 1994).

The WHO (2000) guideline for B[a]P was considered the most appropriate for the assessment of a mixture of carcinogenic PAH in Project emissions. B[a]P was included in the chemical group for lung tumours following chronic inhalation exposures.

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A-6 1,3-BUTADIENE

A-6.1 INHALATION EXPOSURE LIMITS

A-6.1.1 ACUTE INHALATION

IARC (2014) has classified benzo[a]pyrene (B[a]P) as carcinogenic to humans (Group 1). Studies on the carcinogenic potential of B[a]P and mixtures of polycyclic aromatic hydrocarbons (PAHs) following chronic inhalation or oral exposures are outlined in ATSDR (1995); Health Canada (2010); RIVM (2001); and US EPA (1994).

Table A-6.1-1 Acute Inhalation Exposure Limits for 1,3-Butadiene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
ATSDR	-	-	-	-	-	ATSDR 2012
OEHHA	1-hour REL 8-hour REL	660 9	Developmental Ovarian atrophy	Mice	Hackett et al. 1987; NTP 1993	OEHHA 2014; 2013
TCEQ	6-hour ReV	3,700	Developmental	Mice	Hackett et al. 1987	TCEQ 2008
US EPA	24-hour RfC	15	Developmental	Mice	Hackett et al. 1987	US EPA 2002

- not available

The OEHHA (2014), TCEQ (2008) and US EPA (2002) have all developed acute inhalation exposure guidelines for 1,3-butadiene based on a study of developmental toxicity in mice (Hackett et al., 1987). Hacket et al. (1987) examined the reproductive and developmental effects of 1,3-butadiene on pregnant CD-1 mice and their offspring. The mice were exposed via inhalation to 0, 40 ppm (88.4 mg/m^3), 200 ppm (442 mg/m^3) or 1,000 ppm (2,210 mg/m^3) 1,3-butadiene for 6 hours/day on gestational days 6 to 15 and sacrificed on gestational day 18 (Hackett et al., 1987).

The OEHHA (2013) 1-hour REL of 660 µg/m^3 (0.297 ppm) was based on lowered fetal body weights in male offspring following dam exposure for 6 hours/day on gestational days 6 to 15 (Hackett et al. 1987). A $BMCL_{05}$ of 17.7 ppm was identified for lowered male fetal weights using values reported by Green (2003) following a re-analysis of the Hackett et al. (1987) data. A dosimetric adjustment factor was used to calculate an HEC of 29.7 ppm (65.6 mg/m^3) to which an uncertainty factor of 100 was applied to account for use of an animal study (3) and variability in human response (30) (OEHHA 2013).

The OEHHA (2013) also developed an 8-hour limit of 9 µg/m^3 (0.0042 ppm) for 1,3 butadiene based on a chronic NTP (1993) bioassay study that reported ovarian atrophy in female mice exposed via inhalation for 6 h/day, 5 d/wk over 103 weeks. This guideline was not selected for the current assessment of acute inhalation exposure as it was based on a response to chronic inhalation exposure and is intended for repeated 8-hour exposures.

The US EPA (2002) subchronic RfC of 15 µg/m^3 (0.007 ppm) was also based on decreased fetal bodyweights in mice (Hackett et al. 1987). The US EPA (2002) used benchmark modeling to identify an LEC_{05} of 2.9 ppm (6.4 mg/m^3) for a 24-hour exposure period and applied an uncertainty factor of 400 to account for extrapolation from an animal study (3), variability in human response (10), use of a lowest effect level (4) and database deficiencies (3).

The TCEQ (2008) 6-hour ReV of 3,700 µg/m^3 (1.7 ppm) was based on the maternal toxicity of 1,3 butadiene in mice (Hackett et al. 1987). A $BMCL_1$ of 51.3 ppm (113.4 mg/m^3) was determined for decreased maternal extragestational weight gain as a result of daily 6 hour exposures on gestational days 6 to 15. An uncertainty factor of 30 was applied to account for use of an animal study (3) and variability in human response (10).

The REL of 660 µg/m^3 recently developed by OEHHA (2014) using a re-analysis of the Hackett et al. (1987) data was selected for the assessment of 1-hour exposures to 1,3-butadiene. The lowest guideline of 15 µg/m^3 recommended by the US EPA was also selected for assessment of 24-hour exposures to 1,3-butadiene. Use of these two guidelines in the acute inhalation assessment was considered protective of 6-hour exposures to 1,3-butadiene.

It is noted that the limited data available suggests that mice are more sensitive to the developmental effects of butadiene compared to rats or humans due to a greater rate of metabolism of butadiene to the reactive metabolites responsible for butadiene toxicity (OEHHA 2013; ATSDR 2012; TCEQ 2008)). The ATSDR has not developed an acute inhalation exposure limit for 1,3-butadiene due to the lack of available data to account for the significant differences in the metabolism of 1,3-butadiene between species and the concern that exposure limits based on responses observed in mice may overestimate the potential risks to human health (ATSDR 2012).

A-6.1.2 CHRONIC INHALATION

Table A-6.1-2 Chronic Inhalation Exposure Limits for 1,3-Butadiene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m³)	Critical Organ or Effect	Species	Study	Source
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
ATSDR	-	-	-	-	-	ATSDR 2012
HEALTH CANADA	RsC (based on TC01)	1.7	Leukemia	Human	Delzell et al., 1995	Health Canada 2000
OEHHA	RsC RfC	0.06 2.2	Lung neoplasms Ovarian atrophy	Mice	Melnick et al. 1990 NTP 1993	OEHHA 2013; 2011
RIVM	RsC	0.3	Leukemia	Human	Health Canada, 2000; Delzell et al., 1995	RIVM 2009
TCEQ	RsC ReV	20 33	Leukemia Ovarian atrophy	Human Mice	Delzell et al., 1995, others NTP 1993	TCEQ 2008
US EPA	RsC RfC	0.3 2	Leukemia Ovarian atrophy	Human Mice	Health Canada, 2000; Delzell et al., 1995 NTP 1993	US EPA 2002
WHO	-	-	-	-	-	WHO 2000

- not available

1,3-Butadiene has been classified as carcinogenic to humans (Group 1) by the International Agency for Research on Cancer (IARC 2008; 2013). An increased incidence of leukemia in workers exposed to 1,3-butadiene in styrene butadiene rubber plants has been reported by Delzell et al. (1995). Health Canada (2000), the RIVM (2009), TCEQ (2008) and US EPA (2002) have all considered the results of this occupational study in the development of chronic inhalation exposure limits. The OEHHA (2011) developed a chronic inhalation exposure limit for 1,3-butadiene based on the occurrence of lung tumours in mice as reported by Melnick et al. (1990).

Health Canada (2000) compiled exposure-response data for workers from 6 styrene butadiene rubber plants (Delzell et al. 1995) and used regression analyses to identify a butadiene concentration of 1.7 mg/m^3 associated with a 1% (0.01) excess probability of mortality as a result of leukemia (TC_{01}). By extrapolation the air concentration associated with a 1 in 100,000 or 0.00001 leukemia mortality risk would be 1.7 µg/m^3.

The US EPA (2002) considered the Health Canada (2000) analyses of the Delzell et al (1995) data as well as age-specific data on leukemia incidence rates for 1994-1998 from SEER (Surveillance, Epidemiology and End Results) program of the National Cancer Institute to estimate the incidence of (rather than mortality from) leukemia as a result of chronic inhalation exposure to butadiene. An inhalation unit risk of 0.3 µg/m^3 at the 1 in 100,000 risk level was recommended by the US EPA (2002) for chronic exposure to butadiene. This RsC was adopted by RIVM as the chronic inhalation limit value for the evaluation of 1,3-butadiene in air (2009).

The TCEQ (2008) have recommended a risk specific concentration of 20 µg/m^3 for 1 in 100,000 (1x10^{-5}) excess cancer risk associated with chronic inhalation of butadiene. This exposure limit was also based on the Delzell et. al. (1995) study but incorporated exposure estimates, epidemiological studies and dose-response modeling not available at the time of the Health Canada and US EPA assessments. Relative risks were determined using Texas specific rates of leukemia mortality and survival for up to 70 years exposure, whereas the US EPA considered 85 years exposure (TERA 2010).

The OEHHA (2011) recommended a risk specific concentration of 0.06 µg/m^3 at the 1x10^{-5} excess cancer risk for butadiene. In contrast to Health Canada, the OEHHA (2011) considered the available epidemiological data to be insufficient for unit risk calculation. The RsD was instead derived from chronic inhalation studies in mice (Melnick et al. 1990) which reported the occurrence of malignant neoplasms in the lung.

The US EPA (2002) RsC of 0.3 µg/m^3 for 1x10^{-5} excess risk of leukemia incidence was selected for the evaluation of chronic inhalation exposure to 1,3-butadiene. This guideline was selected over the Health Canada guideline as it was based on an incidence rate for leukemia rather than leukemia mortality rates. The US EPA guideline was selected over the TCEQ guideline as it was more conservative and considered national leukemia incidence rates and a longer exposure term. The OEHHA recommended the lowest guideline for the carcinogenicity of 1,3 butadiene based on the response in mice, however the consensus of four agencies on the use of an occupational study did not support the selection of the OEHHA guideline. 1,3-butadiene was included in the chemical group for leukemia following chronic inhalation exposures.

Chronic inhalation exposure limits for the non-carcinogenic effects of 1,3-butadiene have also been developed by the OEHHA (2014), TCEQ (2008) and US EPA (2002). All of these agencies developed non-cancer guidelines based on the NTP (1993) study of reproductive effects (ovarian atrophy) in mice following up to 2 years inhalation exposure to 1,3-butadiene.

The OEHHA (2013) identified a $BMCL_{05}$ HEC of 0.66 mg/m^3 (0.30 ppm) for ovarian atrophy from the NTP (1993) study. This was adjusted by an uncertainty factor of 300, to account for uncertainty in response between species (30) and sensitive individuals (10), resulting in a chronic REL of 2.2 µg/m^3 (0.001 ppm).

Similarly, the TCEQ (2008) determined a $BMCL_{05}$ HEC of 1.02 mg/m^3 (0.462 ppm) for ovarian atrophy based on the NTP (1993) study. An uncertainty factor of 30 was applied to account for sensitive individuals (10) and an incomplete database (3), resulting in a chronic ReV of 33 µg/m^3 (0.015 ppm). An interspecies uncertainty factor was not applied as an HEC was determined from the POD.

The US EPA (2002) determined a $BMCL_{10}$ HEC of 1.9 mg/m^3 (0.88 ppm) for ovarian atrophy based on the NTP (1993) study. This was adjusted by an uncertainty factor of 1,000, to account for uncertainty in response between species (3), an incomplete database (3), sensitive individuals (10) and extrapolation to a level below the 10% effect level (similar to a LOAEL-to-NOAEL extrapolation), resulting in a chronic RfC of 2 µg/m3 (0.001 ppm).

The lowest recommended exposure limit of 2 µg/m3 was selected for the current assessment of the non-carcinogenic effects of 1,3-butadiene, based on the US EPA (2002) RfC and supported by the OEHHA (2013) REL.

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A-7 DIESEL PARTICULATE MATTER

A-7.1 INHALATION EXPOSURE LIMITS

A-7.1.1 ACUTE INHALATION

Table A-7.1-1 Acute Inhalation Exposure Limits for DPM

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	-	-	-	-	-	OEHHA 2014
TCEQ	-	-	-	-	-	TCEQ 2014
WHO	-	-	-	-	-	WHO 1996

- not available

No acute inhalation exposure limits were identified for DPM from the agencies reviewed; therefore the assessment of DPM was limited to chronic exposures.

A-7.1.2 CHRONIC INHALATION

Table A-7.1-2 Chronic Inhalation Exposure Limits for DPM

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	-	-	-	-	-	Health Canada 2010
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	REL RsC	5 0.03	Respiratory system Lung cancer	Rats Human	Ishinishi et al. 1988 Garshick et al. 1987; 1988	OEHHA 1998 OEHHA 2011
RIVM	-	-	-	-	-	RIVM 2001
TCEQ	-	-	-	-	-	TCEQ 2014
US EPA	RfC	5	Respiratory system	Rats	Ishinishi et al. 1988	US EPA 2014; 2003
WHO	-	-	-	-	-	WHO 1996

- not available

Diesel engine exhaust contains thousands of chemicals which can complicate measurements of exposure. The carbonaceous fraction of diesel particulate, also known as elemental carbon, has been identified as a marker for diesel engine exhaust exposure. Elemental carbon represents a large fraction of the particulate mass of diesel exhaust and can be quantified at low levels. In the case of occupational studies, the diesel engine represents the only significant source for elemental carbon in the workplace (Birch and Cary 1996).

The US EPA (2003) and OEHHA (1998) have established chronic exposure limits for diesel particulate matter (DPM). The US EPA (2014) RfC of 5 µg/m^3 is based on respiratory effects, including pulmonary inflammation and histopathological effects (fibrosis), in rats exposed to diesel exhaust for 16 hr/day, 6 days/week over 130 weeks (Ishinishi et al. 1988). A NOAEL of 460 µg DPM/m^3 was identified from the study. This exposure concentration was converted to a human $NOAEL_{HEC}$ of 144 µg DPM/m^3 using a mathematical model of DPM deposition and clearance and assuming that equal pulmonary surface loadings in rats and humans would be associated with similar effects (US EPA 2003). An uncertainty factor of 30 was applied to the $NOAEL_{HEC}$ to account for the response of sensitive individuals (10) and interspecies extrapolation (3). The OEHHA (1998) adopted the US EPA RfC of 5 µg/m^3 as their chronic REL for diesel exhaust.

The US EPA (2003) RfC of 5 µg/m^3 was selected for the evaluation of non-carcinogenic effects associated with exposure to DPM.

The National Toxicology Program (NTP) has classified diesel exhaust particulate as *reasonably anticipated to be a human carcinogen* based on studies in humans with supporting evidence from animal and mechanistic studies (NTP 2014). Diesel exhaust particles, which contain mutagenic and carcinogenic chemicals, are small enough to penetrate and persist in the lower lung region and were considered likely to account for observed human lung cancers; this is supported by evidence of a lack of lung tumours in rats exposed to diesel exhaust that was filtered to remove particles (NTP 2014). The International Agency for Research on Cancer (IARC 2012; 2014) has classified diesel engine exhaust as *carcinogenic to humans* (Group 1) based on sufficient epidemiological evidence for increased risk of lung cancer.

The OEHHA conducted a meta-analysis of studies reporting a relationship between lung cancer and exposure to diesel exhaust and determined that there was a positive association between occupational exposure to diesel exhaust and an increased risk of developing lung cancer (OEHHA, 2011). A unit risk factor of 0.0003 per µg/m^3 was recommended for particulate matter from diesel-fueled engines, based on the incidence of lung tumours reported in a case control study (Garshick et al. 1987) and a retrospective cohort study (Garshick et al. 1988) of US railway workers occupationally exposed to diesel exhaust. This unit risk factor translates to an RsC of 0.03 µg/m^3, assuming an acceptable lifetime cancer risk of 1 in 100,000.

The Health Effects Institute (HEI) organized a Diesel Epidemiology Expert Panel to review two sets of epidemiological studies on diesel exhaust available at the time, including the Garshick (1987; 1988) studies of railroad workers as well as studies of truck drivers (Steenland et al. 1990) (HEI 1999). The panel recommended against using the railroad worker data following a limited analysis of the exposure-response associations which, although suggesting lung cancer risk was greater in worker groups with higher exposure, also suggested that lung cancer risk decreased with increasing duration of employment (HEI 1999).

The US EPA reported that the weight of available evidence from epidemiology studies indicates that occupational exposure to diesel exhaust may pose a lung cancer risk (US EPA 2002; 2003). The studies on railroad workers (Garshick et al. 1987; 1988) and truck drivers (Steenland et al. 1990) were considered to have the best available exposure-response information for estimating cancer risk from occupational exposures, however the US EPA (2003) did not consider these data suitable to derive a cancer risk estimate for environmental exposures, stating there was too much uncertainty in the available data and outlining gaps that would require evaluation before a confident quantitative dose-response analysis and subsequent derivation of cancer unit risk can be performed. The US EPA did consider the supporting data for DPM carcinogenicity in animals but found the data, particularly for rats, not relevant to human exposures as the tumour incidences reported were non-linear and associated with exposure concentrations high enough to produce lung particle overload (US EPA 2003).

Among the evidence for the IARC (2012) classification of diesel engine exhaust as carcinogenic to humans was a recently conducted US National Cancer Institute/National Institute for Occupational Safety and Health study which reported an increased risk of death from lung cancer in non-metal miners (silica limestone, salt, trona and potash mines), following chronic occupational exposure to diesel emissions (Silverman et al. 2012; Attfield et al. 2012). The results from the nested case-control (Silverman et al. 2012) and cohort mortality (Attfield et al. 2012) studies of diesel exhaust exposure in non-metal miners provided evidence (robust exposure-response relationships) for an effect on lung cancer from diesel exhaust exposure in both underground mine workers as well as surface-only workers, suggesting that diesel exhaust may be hazardous in both confined and open spaces and represents a potential public health as well as an industrial health hazard.

This same group of authors (Silverman, Attfield and Garshick) released exposure-response estimates for diesel engine exhaust and lung cancer mortality (Vermeulen et al. 2014). Following a meta-regression analysis of lung cancer mortality and cumulative exposure to elemental carbon (EC), an excess of 21 lung cancer deaths per 10,000 individuals was predicted following lifetime environmental exposure (through 80 years of age) to 0.8 µg/m^3 EC (Vermeulen et al. 2014).

Elemental carbon or EC refers to the carbon-containing components of DPM and is considered the carbonaceous fraction of a diesel particle. The EC content of DPM from heavy duty diesel engines can vary widely (from 20 to 90%) but recent emissions profiles (based on the limited data available) suggests that EC comprises approximately 75% of DPM from heavy duty diesel engines (US EPA 2002). So an excess of 21 lung cancer deaths per 10,000 individuals following exposure to 0.8 µg/m^3 EC could be interpreted as an excess of 21 lung cancer deaths per 10,000 individuals following exposure to 1 µg/m^3 DPM (i.e., 0.8 µg/m3 EC/0.75) and a DPM air concentration 0.005 µg/m^3 would be associated with a 1 in 100,000 lifetime risk of lung cancer. This is a more conservative cancer risk estimate for DPM than the RsC (i.e., 0.03 µg/m^3) currently recommended by the OEHHA (2011). It is important to note that the risk estimate presented for EC by Vermeulen et al. (2014) is still preliminary and no agency has adopted it as an exposure limit. The extrapolation of this information to a DPM air concentration is provided only for comparison sake (i.e., to a cancer risk estimate available from a recognized regulatory agency).

Exposure characterization remains a source of significant uncertainty in determining the potential human cancer risks of diesel particulate matter in diesel exhaust. Concerns have been raised that lung cancer risks based on exposures to past diesel exhaust emissions do not represent lung cancer risks from exposure to current or future emissions. These concerns are based on the recent modifications to diesel engines which serve to filter out diesel particulate matter and modifications to diesel fuel, including ultra-low sulphur content fuel, which also lowers the particulate content in emissions.

Despite the uncertainty associated with the cancer risk estimate available for DPM, the carcinogenic effects of inhalation exposure to DEP were considered for the current assessment, based on the IARC (2012) decision and recent epidemiological evidence presented. The OEHHA (2011) RsC of 0.03 µg/m^3 was selected as it is the only regulatory guideline available for cancer risk. The data provided by Vermeulen et al. (2014) suggests that the OEHHA (2011) RsC is within an order of magnitude of a recent estimate for lung cancer risk associated with EC exposure. DPM was included in the chemical group for lung tumours following chronic inhalation exposures.

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A-8 FORMALDEHYDE

A-8.1 INHALATION EXPOSURE LIMITS

A-8.1.1 ACUTE INHALATION

Table A-8.1-1 Acute Inhalation Exposure Limits for Formaldehyde

Agency	Exposure Limit Type	Exposure Limit Value (µg/m³)	Critical Organ or Effect	Species	Study	Source
ATSDR	MRL 2-hour	50	Eye and nasal irritation	Human	Pazdrak et al. 1993	ATSDR 2013; 1999
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	Short-term IAQG 1-hour	123	Eye Irritation	Humans	Kulle et al. 1993	Health Canada 2006
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	REL 1-hour 8-hour	55 9	Eye irritation Respiratory irritation	Human	Kulle et al. 1987 Wilhelmsson and Holmstrom, 1992	OEHHA 2014; OEHHA 2008
TCEQ	ReV 1-hour	50	Eye and nasal irritation	Human	Pazdrak et al. 1993; Krakowiak et al. 1998	TCEQ 2008
WHO	30 min	100	Eye irritation	Human	Lang et al. 2008; Kulle et al. 1987	WHO 2010

- not available

The ATSDR (2013) recommend an acute inhalation MRL of 50 µg/m³ for formaldehyde. The MRL was based on a study by Pazdrak et al. (1993) which reported eye and nose irritation in human volunteers, including individuals with skin sensitivity to formaldehyde, following 2 hours exposure to 0.4 ppm (0.5 mg/m³) formaldehyde. A 10-fold uncertainty factor was applied to the exposure concentration to account for use of a LOAEL (3) and variability in human response (3) (ATSDR 1999).

Health Canada (2006) recommends an acute (1 hour) indoor air quality guideline of 123 µg/m³ for formaldehyde. This guideline represents one-fifth of the NOAEL of 1,230 µg/m³ for eye irritation in human clinical studies (Kulle 1993).

A 1-hour ReV of 50 µg/m^3 was recommended by the TCEQ (2008) for acute exposure to formaldehyde. Similar to the ATSDR (1999), this ReV was based on eye and nose irritation in human volunteers, including individuals with skin sensitivity to formaldehyde (Pazdrak et al. 1993) as well as individuals with asthmatic symptoms (Krakowiak et al 1998) following 2 hours exposure to 0.5 mg/m^3 formaldehyde. A 10-fold uncertainty factor was applied to the exposure concentration (0.5 mg/m^3) to account for use of a LOAEL (3) and variability in human response (3) (TCEQ 2008).

The OEHHA (2014) recommend a 1-hour REL of 55 µg/m^3 and an 8-hour REL of 9 µg/m^3 as acute exposure limits for formaldehyde. The 1-hour REL of 55 µg/m^3 (0.044 ppm) is based on a NOAEL of 0.5 ppm for mild to moderate eye irritation in nonasthmatic humans exposed to 0.5-3.0 ppm formaldehyde for a 3-hour period (Kulle et al. 1987). The OEHHA (2008) calculated a $BMCL_{05}$ of 0.44 ppm for eye irritation which was adjusted by an uncertainty factor of 10 to account for potential asthma exacerbation.

The OEHHA 8-hour REL of 9 µg/m^3 was based on an occupational study (Wilhelmsson and Holmstrom 1992) reporting nasal, eye and respiratory tract irritation in chemical plant workers exposed to a mean air concentration of 0.26 mg/m^3 formaldehyde over an average of 10 years (OEHHA 2008). The 8-hour REL was not considered for the acute exposure assessment as it was based on chronic exposure data and is intended for repeated 8-hour exposures.

The WHO (2010) recommended a short-term (30 minute) indoor air quality guideline of 100 µg/m^3 for formaldehyde. This guideline was derived from a NOAEL of 0.63 mg/m^3 for eye irritation (Lang et al. 2008; Kulle et al. 1987). The NOAEL was adjusted by a factor of 5, derived from the standard deviation of nasal pungency, resulting in a short term exposure guideline of 0.1 mg/m^3. The short-term guideline was also considered protective of long-term health effects associated with formaldehyde exposure, including cancer. The carcinogenic effects of formaldehyde (i.e., nasal carcinomas in rats) were attributed to increased cell proliferation as a result of cell damage from exposure to concentrations at and above 2.5 mg/m^3 (WHO 2010).

The lowest 1-hour guideline of 50 µg/m^3 (ATSDR, 1999; TCEQ 2008), based on eye and nasal irritation, was selected for the current assessment of acute inhalation exposure to formaldehyde. Formaldehyde was included in the chemical group for eye irritation following acute inhalation exposures.

A-8.1.2 CHRONIC INHALATION

Table A-8.1-2 Chronic Inhalation Exposure Limits for Formaldehyde

Agency	Exposure Limit Type	Exposure Limit Value (µg/m³)	Critical Organ or Effect	Species	Study	Source
ATSDR	MRL	10	Eye and respiratory irritation	Human	Holmstrom et al. 1989	ATSDR 2013; 1999
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	RsC	1.9	Nasal tumours	Rats	Monticello et al. 1996	Health Canada 2001
HEALTH CANADA	Long-term IAQG (8 hour average)	50	Asthma Hospitalization	Human	Rumchev et al. 2002	Health Canada 2006
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	REL RsC	9 2	Respiratory irritation	Human Rat	Wilhelmsson and Holmstrom, 1992 Kerns et al. 1983	OEHHA 2008 2011
RIVM	-	-	-	-	-	RIVM, 2001
TCEQ	ReV RsC	11 18	Respiratory irritation Cell proliferation[1]	Human Rat	Wilhelmsson and Holmstrom, 1992 Schlosser et al. 2003	TCEQ 2008
US EPA	RsC	0.8	Nasal tumours	Rat	Kerns et al. 1983	US EPA 1991

- not available
[1] Key precursor event to tumourigenesis

The ATSDR (2013) recommend a chronic MRL of 10 µg/m³ (0.008 ppm) for formaldehyde. This MRL was based on a LOAEL of 0.24 ppm as an average 8-hour TWA for mild irritation of the eye and respiratory tract and mild damage to nasal epithelium in chemical plant workers occupationally exposed to formaldehyde for an average of 10.4 years (Holmstrom et al. 1989). The LOAEL was adjusted by an uncertainty factor of 30 for use of a LOAEL for mild effects (3) and human variability (10). No adjustment was made for extrapolation to continuous exposure based on evidence provided by Wilmer et al. (1987) that formaldehyde exposure concentration was more important than the product of exposure duration and concentration for determining the severity of epithelial damage of the upper respiratory tract (ATSDR 1999).

The OEHHA (2014) recommend a chronic REL of 9 µg/m³ for noncarcinogenic effects following chronic exposure to formaldehyde. This exposure limit is based on the Wilhelmsson and Holmstrom (1992) study NOAEL (0.09 mg/m³) and uncertainty factor (10) identified for the 8-hour REL for nasal, eye and respiratory tract irritation in chemical plant workers exposed to formaldehyde (OEHHA 2008).

The TCEQ (2008) recommend a chronic ReV of 11 µg/m³ for noncarcinogenic effects associated with chronic exposure to formaldehyde. This exposure limit is based on the Wilhelmsson and Holmstrom (1992) study NOAEL (0.09 mg/m³) for nasal, eye and respiratory tract irritation in chemical plant workers exposed to formaldehyde for 8 hours/day, 5 days/week over an average of 10 years. The NOAEL was adjusted for continuous exposure (0.032 mg/m³) and an uncertainty factor of 3 was applied to account for human variability (TCEQ 2008).

Health Canada (2006) recommended a long term indoor air quality guideline of 50 µg/m³ (based on an 8 hour average) for formaldehyde. This guideline is based on a study by Rumchev et al. (2002) that reported an association between formaldehyde concentrations in indoor air and hospitalization for asthma in children from six months to three years of age. An air concentration of 50 µg/m³ represents the lower end of the exposure concentration range associated with no significant increase of asthma hospitalization. Although an increase in rat nasal carcinomas was reported in studies of exposures to high formaldehyde concentrations, this was considered the result of proliferative regeneration in response to cytotoxicity. Negligible cancer risks were predicted from lifetime exposure to 50 µg/m³ as this air concentration was considered to be sufficiently low to prevent irritation and inflammatory responses (Health Canada 2006).

IARC (2014) has classified formaldehyde as *carcinogenic to humans* (Group 1) and the NTP (2014) has listed formaldehyde as *known to be a human carcinogen*. Although not completely understood, there is evidence for a genotoxic mode of action for nasal tumours and lymphohematopoietic cancers observed in human and animal chronic formaldehyde exposure studies (NTP 2014). Health Canada (2001), OHEHHA (2011), TECQ (2007) and US EPA (1991) have developed chronic inhalation exposure limits based on the carcinogenic potential of formaldehyde.

The US EPA (1991) identified an inhalation unit risk of 1.3×10^{-5} per µg/m³ from a study reporting nasal squamous cell carcinomas in rats following chronic (2 year) inhalation exposure to formaldehyde (Kerns et al. 1983). This unit risk is equivalent to an RsC of 0.8 µg/m³ assuming a 1 in 100,000 incremental cancer risk level. It is noted that the potential inhalation toxicity of formaldehyde is currently under review by the US EPA with a draft human health assessment released on June 2, 2010 for independent peer review and public comment (US EPA 2012).

The OEHHA (2011) derived an inhalation unit risk of 6×10^{-6} per µg/m³ using the Kerns et al. (1983) data for nasal squamous cell carcinomas in rats. The OEHHA unit risk is equivalent to an RsC of 2 µg/m³ for an incremental cancer risk of 1 in 100,000. The upper range of cancer risks predicted by the OEHHA (2011) using the rat bioassay data were determined to be consistent with lung cancer mortality risk estimates for workers (cohort of over 26,000) exposed to formaldehyde (Blair et al. 1986).

Health Canada (2001) determined a TC_{05} of 9.5 mg/m³ using data for the incidence of nasal squamous tumours in a more recent study in rats (Monticello et al. 1996). This air concentration is associated with a 5% (1 in 20) increase in tumour incidence over background. Dividing the TC_{05} by a factor of 5,000 results in an RsC of 1.9 µg/m³ for a 1 in 100,000 incremental cancer risk level.

The TCEQ (2008) recommend an RsC of 18 µg/m³ for formaldehyde assuming a 1 in 100,000 cancer risk level. This exposure limit was derived from Schlosser et al. (2003) who reported BMC and POD values for tumour incidence and cell proliferation in 3 data sets (including Kerns et al. 1983) describing these effects in rats following chronic formaldehyde inhalation. Nasal cell proliferation was the POD selected for guideline development as it represents a key event in formaldehyde-induced carcinogenesis. A POD_{HEC} of 0.44 ppm, representing the 95% $BMCL_{01}$, was determined for this endpoint. The RsC of 0.015 ppm (18 µg/m³) was developed by applying an uncertainty factor of 30 to the POD to account for extrapolation from animal data (3) and human variability (10).

An exposure limit of 9 µg/m³, recommended by the OEHHA and supported by the ATSDR and TCEQ limits, was selected for the evaluation of non-carcinogenic effects following chronic inhalation exposure to formaldehyde. Formaldehyde was included in the chemical groups for nasal irritation following chronic inhalation exposures.

An exposure limit of 2 µg/m³, recommended by the OEHHA was selected for the evaluation of carcinogenic effects following chronic inhalation exposure to formaldehyde. Although the US EPA provided the most conservative guideline (currently under review), the OEHHA conducted a more recent evaluation of the available data and considered the results of animal as well as human studies. Formaldehyde was included in the chemical group for nasal tumours following chronic inhalation exposures.

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A-9 NAPHTHALENE

A-9.1 INHALATION EXPOSURE LIMITS

A-9.1.1 ACUTE INHALATION

Table A-9.1-1 Acute Inhalation Exposure Limits for Naphthalene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	-	-	-	-	-	ATSDR 2013
BC MOE	-	-	-	-	-	BC MOE 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	-	-	-	-	-	OEHHA 2014
TCEQ	ESL	200	Odour	-	-	TCEQ 2014
WHO	-	-	-	-	-	WHO 2001

- not available

The TCEQ have recommended an interim ESL of 200 µg/m^3 for short–term exposure to naphthalene based on odour (TCEQ 2014). This guideline is not health-based and no supporting documentation was provided for the odour threshold identified.

No other acute inhalation guidelines were identified for public exposure to naphthalene; however the American Conference of Governmental Industrial Hygienists (ACGIH) recommends a short-term exposure limit (STEL) of 79 mg/m^3 for naphthalene based on the potential for eye and respiratory tract irritation (OSHA 2012). This STEL was established for occupational exposures up to 15 minutes duration. A 1-hour exposure limit was derived from the ACGIH STEL as follows: 79 mg/m^3 x 15 min = X mg/m^3 x 60 min. This assumes that the biological response to acute naphthalene exposure will be a constant that is a function of time and exposure concentration (i.e., Habers law). Using this assumption, a limit of 20 mg/m^3 was determined for 1-hour exposure to naphthalene. The STEL was developed for worker exposure and therefore a 10-fold uncertainty factor was applied to the 1-hour air concentration to account for sensitive individuals in the general population. The resulting exposure limit of 2 mg/m^3 (2,000 µg/m^3) was selected for the evaluation of acute inhalation exposure to naphthalene. Naphthalene was included in the chemical groups for eye irritants and respiratory irritants following acute inhalation exposures.

A-9.1.2 CHRONIC INHALATION

Table A-9.1-2 Chronic Inhalation Exposure Limits for Naphthalene

Agency	Exposure Limit Type	Exposure Limit Value (µg/m^3)	Critical Organ or Effect	Species	Study	Source
ATSDR	MRL	4	Nasal lesions	Rat	Abdo et al., 2001; NTP 2000	ATSDR 2013; 2005
BC MOE	-	-	-	-	-	BC MOE 2013
HEALTH CANADA	TC	3	Nasal lesions	Mouse	NTP, 1992; US EPA 1998	Health Canada 2010
HEALTH CANADA	Long-term IAQG (24 hour average)	10	Nasal lesions	Rats	NTP 2000	Health Canada 2013
METRO VANCOUVER	-	-	-	-	-	MV 2011
OEHHA	RfC	9	Nasal lesions	Mouse	NTP 1992	OEHHA 2000
RIVM	-	-	-	-	-	RIVM, 2001
TCEQ	ESL	50	-	-	-	TCEQ 2014
US EPA	RfC	3	Nasal lesions	Mouse	NTP, 1992	US EPA 2014; 1998
WHO	Long-term IAQG	10	Nasal lesions	Rats	Various	WHO 2010

- not available

The ATSDR (2005) recommend a chronic inhalation MRL of 4 µg/m^3 for naphthalene. This MRL was based on the occurrence of nasal lesions as reported in two chronic inhalation studies in mice (NTP 1992) and rats (Abdo et al. 2001; NTP 2000). Mice were exposed to naphthalene concentrations of 0,10 or 30 ppm for 6 hours/day, 5 days/week over 104 weeks (NTP 1992). Rats were exposed to 0,10, 30 or 60 ppm naphthalene for 6 hours/day, 5 days/week over 105 weeks (Abdo et al. 2001; NTP 2000). Nasal lesions were observed in both species at the lowest exposure level (LOAEL of 10 ppm or 52 mg/m^3). A $LOAEL_{HEC}$ of 1.04 mg/m^3 (0.2 ppm) was determined for rat nasal lesions, after adjusting for continuous exposure and using EPA (1994) inhalation dosimetry for a category 1 gas to derive a human equivalent concentration (from rat to human) (ATSDR 2005). An uncertainty factor of 300 was applied to the $LOAEL_{HEC}$ to account for use of a LOAEL (10), extrapolation from rats to humans, with dosimetric adjustment (3) and human variability (10). The ATSDR MRL was not selected for the current assessment based on their use of inhalation dosimetry for a category 1 gas when there is evidence to suggest that naphthalene is a category 3 gas, as described below.

An RfC of 9 µg/m^3 is recommended by the OEHHA (2000) for non-carcinogenic effects following chronic inhalation exposure to naphthalene. This RfC was based on the NTP (1992) LOAEL of 52 mg/m^3 (10 ppm) for the occurrence of nasal lesions in mice exposed to naphthalene 6 hours/day, 5 days/week over 104 weeks. This LOAEL was adjusted for continuous exposure (9 mg/m^3) and a 1000-fold uncertainty factor was applied to account for use of a LOAEL (10), extrapolation from mice to humans without dosimetric adjustment (10) and human variability (10). In keeping with the US EPA (1998) IRIS approach, the OEHHA (2000) treated naphthalene as a category 3 gas, based on its low water solubility, low direct reactivity and data to suggest that the toxic effects of naphthalene on the respiratory tract are the result of a reactive oxygenated metabolite that may be formed in the liver or respiratory tract.

The TCEQ have recommended an interim ESL of 50 µg/m^3 for long–term exposure to naphthalene based on health (TCEQ 2014), although no supporting documentation was provided for this ESL.

The US EPA (1998) developed an RfC of 3 µg/m^3 for naphthalene. Similar to the OEHHA (2000), this guideline was based on a LOAEL of 10 ppm (52 mg/m^3) for nasal lesions in mice chronically exposed to naphthalene (NTP 1992). The US EPA (1998) determined a $LOAEL_{HEC}$ of 9 mg/m^3 after adjusting for continuous exposure and following inhalation dosimetry guidance for a category 3 gas. An uncertainty factor or 3000 was applied to account for use of a LOAEL (10), extrapolation from mice to humans (10), human variability (10) and deficiencies in the database, including lack of a 2-generation reproductive toxicity study and lack of chronic inhalation data for other animal species (3).

Health Canada (2010) also established a TC of 3 µg/m^3 for the noncarcinogenic effects of naphthalene based on the US EPA (1998) RfC.

The WHO (2010) and Health Canada (2013) both established indoor air quality guidelines of 10 µg/m^3 for chronic exposure to naphthalene. The WHO (2010) indoor air quality guideline is based on a LOAEL of 53 mg/m^3 for nasal lesions in rats chronically exposed (105 weeks) to naphthalene (NTP 2000). This LOAEL was adjusted to account for continuous exposure (6/24 hours × 5/7 days) and an uncertainty factor of 1000 was applied to the LOAEL to account for extrapolation from rats to humans (10), human variability (10) and use of a LOAEL rather than a NOAEL. This annual average guideline is considered to be protective of the carcinogenic risks of naphthalene exposure (WHO 2010).

The Health Canada (2013) indoor air quality guideline was also established based on the NTP (2000) chronic inhalation study in rats. Similar to the WHO, Health Canada adjusted a LOAEL of 52 mg/m^3 for continuous exposure and applied an uncertainty factor of 1000 to account for extrapolation from rats to humans (10), human variability (10) and deficiencies in the database. This guideline is considered to be protective of nasal cytotoxicity which can lead to nasal tumour development in rats following chronic naphthalene exposure. The minimum recommended sampling time for this guideline is 24 hours (Health Canada 2013).

IARC (2014) has classified naphthalene as possibly carcinogenic to humans (Group 2B) and the NTP (2014) has stated naphthalene is *reasonably anticipated to be a human carcinogen*. The supporting evidence for the NTP classification was provided in an NTP (2000) study in which chronic exposure to naphthalene produced highly malignant and extremely rare tumours of the lining of the nose of rats. The mechanism for naphthalene carcinogenesis is not clear but formation of a specific stereoisomer of naphthalene oxide (1R,2S-) as well as oxidative damage and DNA breakage may play a role (NTP 2014).

The OEHHA (2011) recommend a unit risk value of 0.034 per mg/m^3 for carcinogenic effects following chronic inhalation exposure to naphthalene. This corresponds to an RsC of 0.3 µg/m^3 assuming 1 in 100,000 (1x10^{-5}) excess lifetime cancer risk. Unit risk factors were developed for naphthalene using benchmark dose methodology and tumour incidence data for female mice, male rats and female rats (NTP 1992; 2000). The selected unit risk factor was for the male rat (NTP 2000), the species most sensitive to naphthalene exposure via inhalation (OEHHA 2011).

Health Canada (2013) and WHO (2010) recently developed an indoor air quality guideline of 10 µg/m^3 for chronic exposure to naphthalene, which was considered protective of nasal cytotoxicity and tumour development. Both agencies attributed nasal and lung tumor development to a progression of effects from tissue damage in the nasal cavities and lungs as a result of high exposure concentrations, rather than a direct acting carcinogenic effect.

The majority of agencies have developed chronic inhalation exposure limits based on the non-carcinogenic effects (nasal lesions) of naphthalene reported in mice and rats. For the purpose of this assessment the lowest recommended RfC of 3 µg/m^3 (US EPA, Health Canada) was selected for the assessment of noncarcinogenic effects following chronic inhalation exposure to naphthalene. Naphthalene was included in the chemical group for nasal irritation following chronic inhalation exposures.

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