Appendix 31: Qualitative Human and Ecological Health Assessment

# **APPENDIX 31**

Qualitative Human and Ecological Health Assessment





# EAGLE GOLD PROJECT

Qualitative Human and Ecological Health Assessment

#### FINAL REPORT



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### ABBREVIATIONS AND ACRONYMS

	Agency for Toxic Substances and Disease Registry
BC MoE	British Columbia Ministry of Environment
CAC	Criteria Air Contaminants
CCME	Canadian Council of Ministers of the Environment
CEAA	Canadian Environmental Assessment Act
COHb	carboxyhemoglobin
COPC	contaminants of potential concern
EA	environmental assessment
EC	Environment Canada
ERED	US EPA's Environmental Residue-effects Database
FNNND	First Nation of Na-Cho Nyäk Dun
HC	Health Canada
HHERA	human health and ecological risk assessment
HLF	heap leach facility
LAA	Local Assessment Area
la e Kau	
log Kow	log octanol-water partition coefficient
	log octanol-water partition coefficientNational Ambient Air Quality Objectives
NAAQO	
NAAQO NND	National Ambient Air Quality Objectives
NAAQO NND PbB	National Ambient Air Quality Objectives
NAAQO NND PbB PEL	National Ambient Air Quality Objectives Na-Cho Nyäk Dun blood lead
NAAQO NND PbB PEL PM <sub>10</sub>	National Ambient Air Quality ObjectivesNational Ambient Air Quality ObjectivesNa-Cho Nyäk Dunblood leadblood lead
NAAQO NND PbB PEL PM <sub>10</sub> PM <sub>2.5</sub>	National Ambient Air Quality Objectives Na-Cho Nyäk Dun blood lead CCME Probable Effects Level Particulate matter <10 µm
NAAQO NND PbB PEL PM <sub>10</sub> PM <sub>2.5</sub> PQRA	National Ambient Air Quality Objectives Na-Cho Nyäk Dun blood lead CCME Probable Effects Level Particulate matter <10 μm Particulate matter <2.5 μm
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NAAQO NND PbB PEL PM <sub>10</sub> PM <sub>2.5</sub> PQRA RAA RAIS sp	National Ambient Air Quality Objectives Na-Cho Nyäk Dun blood lead CCME Probable Effects Level Particulate matter <10 μm Particulate matter <2.5 μm Preliminary Quantitative Risk Assessment Regional Assessment Area Risk Assessment Information System

TSP	total suspended particulate
US EPA	United States Environmental Protection Agency
WHO	World Health Organization
WRSAs	waste rock storage areas
YESAA	Yukon Environmental and Social Assessment Act

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# 1 INTRODUCTION

Stantec was retained by Victoria Gold Corp. (VIT) to prepare a qualitative human health and ecological risk assessment (HHERA) for the Eagle Gold Project (the Project), using methods consistent with environmental assessment (EA) standards under Yukon and federal legislation. The Project is a proposed open pit gold mine within the Dublin Gulch watershed located 85 km by road northeast of the Village of Mayo, Yukon. The closest permanent residences to the proposed Project are 65 km by road in Keno City, now home to a small permanent population which increases with the return of seasonal residents. A range of activities occur in the vicinity of the proposed mine, including extensive traditional land use by the First Nation of Na-Cho Nyäk Dun (FNNND) (e.g. hunting, fishing, berry harvesting), trapping, placer mining, tourism, and recreation.

## 1.1 Purpose of the HHERA

This report examines the potential effects of the Project on human and ecological health, specifically related to potential chemical releases to the environment. The HHERA considers chemical releases to air, soil, and water, and includes qualitative assessments of both baseline conditions and potential Project-related inputs. A quantitative assessment may be warranted and recommended if the results of the current qualitative assessment or future monitoring indicate contaminant inputs greater than levels anticipated to pose a risk to human or ecological health. The HHERA will form part of the supporting documentation completed as part of the Project Proposal.

# 2 STUDY METHOD

This section outlines the scope and process used to assess potential effects of chemical releases from the Project on human and ecological health.

## 2.1 Regulatory Setting

While a HHERA is not explicitly required under the Yukon Environmental and Social Assessment Act (YESAA), the assessment is required to consider the potential for the Project to result in effects on human health. Specifically, Section 42(1)(g) of the YESAA identifies health of Yukon Indian persons and other residents of Yukon as a matter to be considered. The HHERA fulfills this regulatory requirement.

The HHERA follows established methods published or endorsed by Health Canada (HC), Environment Canada (EC), the Canadian Council of Ministers of the Environment (CCME) and the United States Environmental Protection Agency (US EPA), which are widely accepted and have been used successfully throughout Canada in similar environmental assessments.

Guidance for preparing this HHERA was taken from:



- Federal Contaminated Sites Risk Assessment in Canada, Part I: Guidance on Human Health Risk Preliminary Quantitative Risk Assessment (PQRA) (HC 2004a)
- Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part A) (US EPA 1989a)
- A Framework for Ecological Risk Assessment (CCME 1996)
- Canadian Handbook on Health Impact Assessment (HC 2004c).

### 2.2 Risk Assessment Framework

The purpose of HHERA is to evaluate the potential for adverse health outcomes from both short-term (acute) and long-term (chronic) exposures resulting from contaminants released to air, land, and water. HHERA is an evaluation process used to describe the nature and magnitude of these potential health risks.

All chemicals (from natural and anthropogenic sources) have the potential to cause environmental effects. However, the magnitude of environmental effects (i.e., risk) depends on the receptor (person or wildlife) being exposed, the route of exposure, and the hazard (inherent toxicity) of the chemical. For risk to exist, three critical elements need to be present: receptor, hazard and exposure. That is:

- A chemical or compound must be released to the environment at a sufficient concentration.
- A pathway must exist for the chemical to travel to a potential receptor and be absorbed into that receptor.
- A dose must be received, and the chemical must have some adverse toxicological effect (i.e., temporary or permanent damage).

For example, a receptor could be exposed to a chemical, but if that chemical is essentially hazardless (low toxicity) and present at only very low levels, then no unacceptable risk would be expected. Alternatively, an extremely hazardous chemical may be present, but if there is no way for a receptor to be exposed to that chemical, then that receptor is not at risk. If any one of these components is missing, then there is no risk to receptors. The key elements of a HHERA are presented schematically in Figure 2.2-1.



Figure 2.2-1: Key Elements of Human Health and Ecological Risk Assessment

The risk assessment framework consists of the following components, described in Figure 2.2-2:

- Problem Formulation
- Exposure Assessment
- Toxicity Assessment
- Risk Characterization
- Uncertainty Analysis.

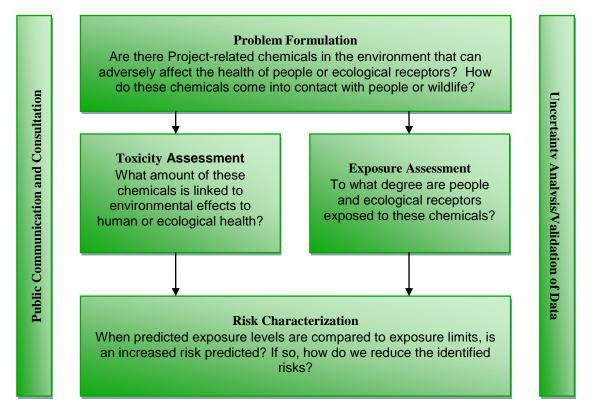


Figure 2.2-2: Risk Assessment Framework

### 2.2.1 **Problem Formulation**

During the problem formulation stage information is gathered and interpreted, focusing the study on the primary issues of concern for the Project. The gathered data provide information regarding the physical layout and characteristics of the assessment area, possible exposure pathways, potential human and ecological receptors, contaminants of potential concern (COPC), and any other specific issues of concern to be addressed.



The key tasks requiring evaluation within the problem formulation step include:

- Characterization of the Project and the assessment area, including habitat and land use
- Identification of COPC associated with Project-related emissions
- Identification of the potentially affected environmental media
- Receptor identification and characterization
- Identification of exposure pathways and routes.

### 2.2.2 Exposure Assessment

People and ecological receptors can come into contact with chemicals in a variety of ways, depending on their daily activities and land use patterns. The means by which receptors contact a chemical in an environmental medium is referred to as an exposure pathway. The means by which a chemical enters the body from the environmental medium is referred to as an exposure route. The exposure assessment incorporates information about Project-related chemical emissions, activities and land use in the area, receptor characteristics, and the exposure pathways identified during the problem formulation phase of the HHERA.

Generally, receptors (human or ecological) can be exposed to chemicals in the environment by:

- Directly inhaling them
- Coming into direct dermal contact with them, or
- Ingesting them along with food or water.

The exposure assessment predicts the rate of exposure (i.e., the quantity and rate at which a chemical is received) of the selected receptors to the COPC via the various exposure scenarios and pathways identified in the problem formulation step. The rate of exposure to chemicals from many pathways is usually expressed as the amount of chemical taken in per body weight per unit time (e.g. microgram ( $\mu$ g) chemical/kilogram (kg) body weight/day).

The magnitude of the exposure of receptors to chemicals in the environment depends on the interaction of a number of variables, including the:

- Concentration of chemicals in various environmental media
- Physical-chemical characteristics of the COPC, which affect their environmental fate and transport and determine such factors as efficiency of absorption into the body
- Influence of site-specific environmental characteristics, such as geology, soil type, topography, hydrology, hydrogeology, local meteorology, and climatology on a chemical's behaviour within environmental media
- Physiological and behavioural characteristics of the receptors (e.g. respiration rate, soil/dust intake, time spent at various activities and in different areas).

In the case of a qualitative risk assessment, the exposure assessment simply identifies the people and the wildlife and the potential routes of exposure that they may be subjected to.

### 2.2.3 Toxicity Assessment

The toxicity assessment involves the selection of toxicity reference values (TRVs), also referred to as exposure limits, for COPC. Toxicity is the potential for a chemical to produce any type of damage, permanent or temporary, to the structure or functioning of any part of the receptor's body. The toxicity of a chemical depends on the amount of the chemical taken into a receptor (referred to as the "dose") and the duration of exposure (i.e., the length of time that a receptor is exposed to a chemical). For each COPC, there is a specific dose and duration of exposure necessary to produce a toxic environmental effect in a given receptor. This is referred to as the "dose-response relationship" of a chemical. The toxic potency of a chemical (i.e., its ability to produce any type of damage to the structure or function of any part of the body), is dependent on the inherent properties of the chemical itself (i.e., its ability to cause a biochemical or physiological response at the site of action), as well as the ability of the chemical to reach the site of action. This dose-response principle is central to the HHERA methodology.

In a qualitative risk assessment only a brief description of chemical toxicity is provided. In the event a more detailed quantitative risk assessment is undertaken, specific TRVs are required to be sourced from reputable government agencies or peer reviewed literature.

### 2.2.4 Risk Characterization

The risk characterization integrates the exposure and toxicity assessments to provide a conservative estimate of health risk for the receptors assessed in the various exposure scenarios. Potential risks are characterized through a comparison of the estimated or predicted exposures from all pathways (from the exposure assessment) with the identified exposure limits (from the toxicity assessment) for COPC.

If the results of the assessment indicate the potential for adverse health risks related to Project emissions, this may lead to a need for development of site-specific management options and/or criteria.

In the case of a qualitative assessment, discussion on the relative increase of chemicals in the environment and route(s) of exposure are explored and a discussion for the need of a quantitative assessment of risk to humans and the environment is made.

### 2.2.5 Uncertainty Analysis

Uncertainties are inherent in the risk assessment process. Generally, uncertainties are addressed by incorporating conservative assumptions in the analysis. As a result, risk assessments tend to overstate the actual risk. Although many factors are considered in preparation of a risk analysis, results are generally only sensitive to a few of these factors. The uncertainty analysis is included to demonstrate that assumptions used are conservative, or that the analysis result is not sensitive to the key assumptions.

# 3 ASSESSMENT AREA AND SCENARIOS

### 3.1 Assessment Area

The assessment area for the Project HHERA (see Figure 3.1-1) is based on the boundaries defined for the Soil Quality, Water Quality and Air Quality assessments with consideration of boundaries established for wildlife, vegetation, and fish and fish habitat assessments,(see Project Proposal Section 6) as well as information from the FNNND Traditional Knowledge and Use Study (Stantec 2010).

The Air Quality assessment includes reporting of chemical concentrations at ground level for numerous potential receptor locations, including identified sensitive receptor locations, such as active placer mining areas (see Figure 3.1-1). The HHERA discusses the potential effects of the overall maximum predicted ground level concentrations of air contaminants, as well as potential effects of ground level concentrations at sensitive receptor locations.

FNNND traditional use areas (e.g. fishing, hunting, trapping and berry picking locations) are considered sensitive receptor locations. These locations have not been identified specifically in the assessment to protect First Nations traditional knowledge; however, the current assessment boundaries adequately evaluate potential health effects of the Project associated with traditional use activities.

### 3.2 Receptor Locations

Specific locations were assessed as part of the HHERA. The selection of the receptor locations incorporated land use, air modeling results, and input from other disciplines' studies (Figure 3.1-1).

Receptor locations were selected with consideration of:

- Land use
- Presence of existing communities
- Likelihood or known presence of ecological receptors (e.g. proximity to Project, bodies of water, wetlands)
- Locations of known recreational, subsistence, and traditional use (e.g. fishing, placer mining)
- Habitat for local wildlife species.

### 3.3 Assessment Scenarios

Five main scenarios were evaluated as part of this HHERA:

- 1. **Existing Conditions**—the baseline phase considers existing chemical conditions in air, soil, water, and biota (e.g. fish tissue) in the assessment area
- 2. **Construction**—the construction phase includes all construction and site preparation work that occurs prior to the start up of mining and heap leach activities. It includes earthworks,

pre-stripping of the open pit, construction of major water management structures, preparation and construction of the heap leach facility (HLF) liner, and construction of buildings and facilities to support operations of the mine. The duration of the construction phase is approximately 1.7 yrs (January 2012 to August 2013).

- 3. **Operations**—the operations phase is the period of time from the start of mining operations to the end of mining and ore processing, including the gradual development and growth of the waste rock storage areas (WRSAs), the HLF and the Open Pit. The operations phase is 7.3 years, from September 2013 to December 2020.
- 4. **Closure and Reclamation**—the closure and reclamation phase is the period of time where mining operations have ended and closure and reclamation activities are taking place. There is a one year period when heap leaching is still occurring to extract and recover residual gold in the ore, but no new ore is placed on the heap leach facility. The HLF then goes through rinsing and detoxification, drain down, and reclamation. The re-contouring and reclamation of the WRSAs and development of any aquatic habitat enhancements occur in the closure and reclamation phase as well. Decommissioning and demolition of site facilities and infrastructure will occur during this phase as well. The closure and reclamation phase is 10 years, from January 2021 to December 2030.
- 5. **Post-closure Monitoring**—this period represents conditions in the assessment area after reclamation is complete. Although environmental monitoring will occur throughout all phases of the Project, post-closure monitoring will be dependent on when reclamation activities are completed. At this time, we have assumed the post-closure phase will begin in January 2031.

Project Scenario	Phase	Description			
Existing Conditions	Baseline	<ul> <li>Qualitative assessment of existing conditions in the assessment area</li> <li>Health risks assessed using measured concentrations of</li> </ul>			
		chemicals of potential concern (COPC) in surface soil, surface water, sediment, fish tissue, and vegetation			
Construction	Construction	<ul> <li>Qualitative assessment of the potential health risks associated with air emissions during construction and commissioning of the Project</li> </ul>			
Operations	Project (Baseline and Project)	<ul> <li>Qualitative assessment of COPC emissions from the Facility in combination with existing/baseline conditions.</li> </ul>			
		<ul> <li>Assessment at the point of maximum ground level concentration as well as individual HHERA receptor locations</li> </ul>			
	Future Conditions	<ul> <li>Qualitative evaluation of mine emissions in combination with future sources of emissions</li> </ul>			
Closure and Reclamation	Closure	<ul> <li>Qualitative assessment related to the removal of infrastructure and rehabilitation of the area</li> </ul>			
Post-closure Monitoring	Post-closure	<ul> <li>Qualitative assessment of return of conditions after reclamation</li> </ul>			

 Table 3.4-1:
 Summary of Scenarios Assessed in the HHERA

### 3.3.1 Baseline

To effectively assess potential risks attributable to Project-related emissions, baseline chemical concentrations in air, soil, water, and biota must be established. Given the isolated and relatively undisturbed setting of the proposed Project, existing chemical conditions are expected to reflect those of the natural geology.

### 3.3.2 Construction

During the construction phase there will be a requirement for the operation of diesel generators (power supply) and the operation of heavy machinery for mine site construction. Emissions of Criteria Air Contaminants (CACs) from these activities have the potential to alter air quality and subsequently affect human health. Although ecological receptors may be exposed to these CACs from vehicle and diesel exhaust, it is not standard practice to examine potential affects to ecological receptors from these contaminants as they would not affect the overall viability of the ecosystem.

The pre-production site preparation and clearing of overburden at the location of the open pit all have the potential to increase the dust load at the site, potentially increasing the soil metal concentrations around other areas of the property. Physical clearing activities and infrastructure development would not result in any chemical emissions that would affect human health, or they could be mitigated using established methods and practices.

### 3.3.3 Operations

Activities related to open pit mining (e.g. blasting), ore crushing, conveyance processing, and stockpiling pose a potential risk to both human and ecological health, given their potential for dust generation and subsequent metal deposition onto surrounding soils. It is possible that any increase in soil metal loading could subsequently be taken up by vegetation and also wildlife/country foods. There will also be a potential for increase loading of metals into the watershed. Any loading could have the potential for impacting potable water and ultimately be taken up into fish tissue.

Similar to the construction phase of the Project, any power generation or vehicle emissions have the potential to generate CACs that may affect human health.

### 3.3.4 Closure and Reclamation

During the closure phase there will be a requirement for heavy machinery to be operated for grading, infrastructure removal, and reclamation activities. These activities pose a potential risk to human health.

### 3.3.5 Post-closure Monitoring

During this phase environmental chemical concentrations will be monitored to ensure that there is not an ongoing potential risk to humans and the environment.

# 4 **PROBLEM FORMULATION**

### 4.1 Receptors

Human and ecological receptors that could potentially be exposed to Project-related emissions are outlined below.

### 4.1.1 Humans

Potential Project effects on human health were considered with respect to First Nations and members of the public who use the HHERA assessment area for traditional, subsistence, and recreational purposes. The FNNND harvest berries, Labrador tea, wild onion, fish, moose, grouse and ptarmigan from the assessment area. Placer mining, trapping, and recreational activities also occur within the assessment area.

As air currents have the potential to carry airborne CACs from the Project to human and ecological receptors, air quality was also assessed at Elsa where there is a camp for the operation of the Bellekeno Mine. Given that Keno City, the closest permanent settlement to the Project, is greater than 65 km away, it is outside the zone of any influence from potential Project effects on air quality.

As potential health effects to Project workers are governed by occupational health and safety laws and codes of practice in Yukon, potential effects on workers at the Project site itself were not considered in the health assessment.

### 4.1.2 Ecological Receptors

Potential Project effects on ecological health were considered due to the presence of wildlife and fish in the assessment area, and the importance of maintaining healthy and sustainable wildlife populations in the region. Ecological health is also important to First Nations people and other land users who harvest plants, fish, and wildlife in the vicinity of the Project. Detailed lists of wildlife and fish species present in the region are described in the Wildlife (Appendix 12), and Fish and Fish Habitat (Appendix 5) baseline reports, respectively.

Mammalian species potentially present in the assessment area include moose (*Alces alces*), woodland caribou (*Rangifer tarandus caribou*), black bear (*Ursus americans*), grizzly bear (*Ursus arctos*), grey wolf (*Canus lupus*), red fox (*Vulpes vulpes*), snowshoe hare (*Lepus americanus*), wolverine (*Lagopus* sp.), and American marten (*Martes americana*). Game bird species include spruce grouse (*Falcipennis canadensis*), dusky grouse (*Dendragapus obscures*), ruffed grouse (*Bonasa umbellus*), and three species of ptarmigan (*Lagopus* sp.). Raptors, songbirds, and waterfowl species are also present. The wood frog (*Rana sylvatica*) is the only amphibian species likely to occur, and no reptile species are expected to inhabit the area. Arctic grayling (*Thymallus arcticus*) and slimy sculpin (*Cottus cognatus*) were the only two fish species caught during the 2009 baseline fisheries assessment.



### 4.2 Chemicals and Elemental Metals of Potential Concern

Selection of the chemicals and metals of potential concern (COPC) to be evaluated is a critical step in any risk assessment. It is standard practice in HHERA to limit the number of chemicals evaluated to those representing the greatest potential to affect health. It is preferable to comprehensively evaluate a smaller number of chemicals that represent the greatest potential concern, than it is to conduct a less detailed risk assessment on a larger number of chemicals that are of lesser potential concern. The COPC selection process is designed such that if no unacceptable health risks are predicted for the chemicals evaluated, then health risks would not be expected for any of the chemicals not included in the evaluation (i.e., those that are present at lower environmental concentrations, emitted at lower rates, or possessing a lower toxic potency). A number of screening methods can be used to narrow a list of chemicals for further analysis. These include:

- Relative toxic potency determinations using emission rates and exposure limits
- Comparison of relative bioaccumulation and persistence in the environment, based on the log octanol-water partition coefficient (log Kow) and soil half-life values
- Identifying chemicals viewed as a concern by regulatory authorities for the industry in question
- Identifying chemicals perceived as a concern by the public.

The following is a summary of the evaluation process for the derivation of COPC for the Project.

### 4.2.1 Sources of Emissions

The following key issues have the potential to change the chemical environment associated with the proposed mine and subsequently affect health:

- Emissions of CACs from Project activities (e.g. operation of heavy machinery) have the potential to affect human and ecological health.
- Dust generation and deposition of metals to surface soils from mining activity have the potential to increase soil metal concentrations and affect foods for humans and terrestrial ecological receptors.
- Release, seepage, or discharge of groundwater and surface water and associated metals to the watershed post-closure have the potential to increase the metal body burden of fish and thus potentially affect human health and terrestrial ecological receptors through consumption.

# 4.2.2 Chemicals and Metals of Potential Concern Carried forward in the HHERA

COPCs for the Project were identified based on existing conditions, Project specifications, and a review of chemicals that are related to gold mine operations. Chemicals and metals typically associated with mine activities include metals in dust/ore and criteria air pollutants (e.g. SO<sub>2</sub>, NO<sub>2</sub>, and particulates).

The list of COPC evaluated in this assessment is provided in Table 4.2-1.

COPC	
Criteria Air Contaminants	
Sulphur Dioxide (SO <sub>2</sub> )	Particulate Matter (PM <sub>2.5</sub> )
Nitrogen Dioxide (NO <sub>2</sub> )	Particulate Matter (PM <sub>10</sub> )
Metals	
Antimony	Lead
Arsenic	Mercury
Barium	Molybdenum
Beryllium	Nickel
Boron	Silver
Cadmium	Selenium
Chromium	Thallium
Cobalt	Tin
Copper	Vanadium
Cyanide (sodium cyanide)	Zinc

Table 4.2-1: COPC Considered for Human and Ecological Health

#### 4.2.2.1 Criteria Air Pollutants Discussion of Toxicity

CACs that are associated with Project activities are detailed below. They are primarily related to various combustion sources including open burning, construction equipment, rock moving equipment, diesel generators, and motor vehicles. They include:

- Particulate matter <2.5 µm (PM<sub>2.5</sub>)
- Particulate matter <10 µm (PM<sub>10</sub>)
- Total suspended particulates (TSP)
- Nitrogen oxides (NO<sub>x</sub>)
- Sulphur dioxide (SO<sub>2</sub>)
- Carbon monoxide (CO).

#### **Particulate Matter**

The inhalation of ambient air is the only pathway where receptors can be directly exposed to emissions of airborne particulate matter (PM). Concurrently, the size of the airborne particles to which people are exposed is one of the most important aspects in determining the potential for health risk resulting from PM exposure. Size is directly related to where particles will be deposited in specific parts of the respiratory tract. Particles larger than approximately 10  $\mu$ m in aerodynamic diameter (>PM<sub>10</sub>) are deposited almost exclusively in the nose, throat, and upper respiratory tract, and tend to be coughed out over a very short period of time. This size range is considered



outside the inhalable range for people, since these particles are too large to be deposited in the lung. Particles greater than  $PM_{10}$  are therefore not considered as critical as the fractions less than  $PM_{10}$  since they are not absorbed into the body.

Fine particles are defined as having an aerodynamic diameter of <2.5  $\mu$ m and ultrafine particulate matter have been defined as particles which are less than 0.1  $\mu$ m in aerodynamic diameter. Fine and ultrafine particulate matter have the potential to cause adverse health effects due to their small size, high surface area, and their ability to reach the alveoli and penetrate the deepest part of the lung structure. Unlike PM<sub>10</sub>, fine and ultrafine particulate matter are not likely to be coughed out. Ultrafine particles (also commonly referred to as nanoparticles) also have a greater tendency than larger particles to carry bound chemical components into the lung (e.g. gases, organic compounds and metals). Due to their small size these particles also tend to be present in greater numbers, and they possess a greater total surface area than larger particles of the same mass. As a result, ultrafine particles have the capacity to produce potentially serious respiratory and cardiovascular complications.

Thus, when examining the potential impacts of exposure to PM on human populations, the emphasis in an HHRA is typically focused on the fine and/or ultrafine fractions, as opposed to the broader size fraction represented by total suspended particulate (TSP), which comprises particles ranging up to 44  $\mu$ m. PM<sub>10</sub> is also widely used to evaluate potential health issues, since this size of particle can also travel deep into the lung. When both sets of data are available (PM<sub>10</sub> and PM<sub>2.5</sub>), the PM<sub>2.5</sub> data tends to carry more weight in determining the potential for health risks, due to the finer size of the particles.

#### **Nitrogen Oxides**

Recent epidemiological studies and evidence from toxicological and human clinical studies provide evidence of a relationship between exposures to  $NO_2$  and respiratory health effects. Those effects reported for humans include respiratory symptoms of increasing severity that are linked to the level of exposure. Studies have linked exposure to  $NO_2$  to decreased pulmonary function in asthmatic populations.

Nitrogen-containing air pollutants, in particular  $NO_2$ , have been reported to cause leaf damage. Most important is the combination of  $NO_2$  with other air pollutants such as sulphur dioxide and ozone that cause a higher degree of phytotoxicity. Symptoms such as chlorosis, browning or bleaching between the leaf veins, especially near margins have been reported (Malhotra and Blauel 1980) in deciduous trees species at acute  $NO_2$  concentrations. Nitrogen oxides can affect vegetation indirectly, via chemical reactions in the atmosphere, or directly after being deposited on vegetation, soil or water. The indirect pathway refers to the atmospheric chemistry of NO and  $NO_2$  which are of key importance in the production and removal of tropospheric ozone ( $O_3$ ) (Mansfield 2002). The direct impact of airborne nitrogen is due to toxic effects, eutrophication, and acidification.

#### **Sulphur Dioxides**

Recent epidemiological studies and evidence from toxicological and human clinical studies suggest a relationship between short-term (24-hr average) exposures to SO<sub>2</sub> and respiratory health effects

(e.g. increased airway resistance; reduced air conductance). However, limited or insufficient data exist to provide reasonable certainty about the effects of ambient concentrations of  $SO_2$  on lung function, airway hyper-responsiveness, or inflammation.

High concentrations of sulphur dioxide can produce acute injury in vegetation, which is first observed on the plant foliage. The plant foliage is more sensitive than stems, buds or reproductive parts (Legge and Krupa 2002). The most prevalent phytotoxicity symptoms occur in the form of foliar necrosis, even after relatively short duration exposure. The acute effects are generally, less important in the field than chronic effects, which result from long-term exposure to much lower concentrations, with periodic intermittent and random peak levels (Krupa 1996). Long-term exposure may induce chronic injury symptoms such as marginal or interveinal chlorosis in broad leaf plants and premature fall colouration and premature loss of leaves (Legge, et al., 1998). Often reduced growth and yield and increased senescence have been reported without the development of visible foliar injury symptoms.

#### **Carbon Monoxide**

Health effects of carbon monoxide exposure are largely cardiovascular-related, due to the formation of carboxyhemoglobin, which impairs the oxygen carrying capacity of the blood (World Health Organization 1999).

Carbon monoxide diffuses rapidly across alveolar, capillary, and placental membranes. Approximately 80 – 90% of the absorbed carbon monoxide binds with hemoglobin to form carboxyhemoglobin (COHb), which is a specific biomarker of exposure in blood. The affinity of hemoglobin for carbon monoxide is 200 – 250 times that for oxygen. The binding of carbon monoxide with hemoglobin to form COHb reduces the oxygen-carrying capacity of the blood and impairs the release of oxygen from hemoglobin to extravascular tissues. These are the main causes of tissue hypoxia produced by carbon monoxide at low exposure levels. The World Health Organization (WHO) provides CO guidelines that are based on the Coburn-Foster-Kane exponential equation, which takes into account all the known physiological variables affecting carbon monoxide uptake.

#### 4.2.2.2 Metals Discussion of Toxicity

Mining involves extraction of ore bodies that are rich in minerals (inorganic elements). Through the mining, crushing, conveying, processing, and waste disposal processes, fugitive emissions of dust have the potential to load to existing soil concentrations on the property. The Surficial Geology, Terrain, and Soils Baseline (Appendix 6) provides estimates of changes in metal concentrations in surface soil from air deposition loading. Those chemicals that were determined to have a loading that would be above soil quality guidelines, or those inorganic elements that were of potential concern in the baseline HHERA were carried forward in the Project effects assessment.

#### Arsenic

Elemental arsenic (As) occurs naturally within the earth's crust, and may be redistributed as airborne dust particles, through run-off or by leaching (ATSDR 2007). Human exposure to arsenic typically



occurs through consumption of food products and soils, drinking water, dermal contact, and through inhalation of airborne particles (ATSDR 2007).

One of the most common health risks of chronic oral exposure to inorganic arsenic is skin damage, including the occurrence of warts and an overall darkening of the skin, potentially leading to skin cancer (ATSDR 2007). In addition, oral exposure to inorganic arsenic may result in nausea, vomiting, and diarrhoea after repeated exposure to lower doses (ATSDR 2007). Neurotoxicity is also associated with arsenic exposure, leading to peripheral neuropathy of the sensory and motor nerves (RAIS 1992).

Both organic and inorganic forms of arsenic can be found in food. While the levels of each depend on the type of food, inorganic arsenic is not usually found at high levels. Higher levels of arsenic are generally found in fish and shellfish, but in the organic form, which is not of concern to human health.

#### Cadmium

Cadmium (Cd) is found in the earth's crust associated with zinc, lead, and copper ores. Cadmium exists in air as particles or vapours and can be transported long distances where it will deposit onto soils and water surfaces. It generally binds strongly to organic matter in soil. In water cadmium either adsorbs to sediments, or migrates in water. Cadmium can accumulate in aquatic organisms and plants. The primary source of cadmium exposure is from the food supply. Most of the cadmium that enters the body is stored in the kidneys and liver. The body can change most cadmium to a form that is not harmful, but too much cadmium can overload the capacity of the liver and kidney. Eating food or drinking water with very high cadmium levels severely irritates the stomach, leading to vomiting and diarrhoea, and sometimes death. Eating lower levels of cadmium over a long period of time can lead to a build-up of cadmium in the kidneys. If the levels reach a high enough level, the cadmium in the kidney will cause kidney damage. Exposure to lower levels of cadmium for a long time can also cause bones to become fragile and break easily. Cadmium is a probable human carcinogen (ATSDR 2008a).

#### Copper

Copper (Cu) is a metal that occurs naturally in the environment, and also in plants and animals. It is considered an essential nutrient for human sustainment at low levels, however high levels of exposure can cause harmful effects such as irritation of the nose, mouth and eyes, vomiting, diarrhoea, stomach cramps, nausea and, occasionally, death (ATSDR 2004). The US EPA's IRIS program (1991) determined that existing studies are inadequate to assess the carcinogenicity of copper.

#### Chromium

Chromium (Cr) is a naturally occurring element that is often found complexed with oxygen, iron, or lead. Although chromium has nine different oxidation states, it is often found either in its trivalent (III) or hexavalent (VI) states. Total chromium represents a mixture of these compounds.

The health effects of chromium compounds are greatly dependent on their speciation. Chromium (III) is an essential nutrient; helping the body effectively use sugar, protein, and fat. Although it can be toxic, this generally occurs at doses far higher than toxic doses of chromium (VI) (ATSDR 2008b).

Inhalation of chromium (VI) (or very high doses of chromium [III]) can cause irritation of the lining of the nose, resulting in nose ulcers (due to cellular necrosis) and runny nose, as well as causing breathing problems such as asthma, cough, shortness of breath, and wheezing (ATSDR 2008b). Ingestion of chromium (VI) has lead to irritation and ulcers in the stomach and small intestine, as well as anemia, in animal studies. Sperm damage and damage to the male reproductive system has also been observed in animal studies following exposure to chromium (VI) (ATSDR 2008b). Dermal contact with chromium (VI) can cause skin ulcers. Allergic reactions, consisting of severe redness and swelling of the skin, have been seen in people sensitive to either chromium (III) or chromium (VI) (ATSDR 2008b). Occupational exposures to chromium (VI) compounds have been associated with increased risks of respiratory system cancers (ATSDR 2008b). Epidemiological studies of workers exposed to chromium (VI) compounds in the plating and chromate pigment industries have consistently shown an association between occupational inhalation exposures and respiratory tract cancers (primarily nasal and bronchogenic cancers) (ATSDR 2008b). These studies have been used by both the US EPA and Health Canada to develop cancer slope factors for inhalation exposures to chromium (VI) (HC 2004b; US EPA 2008).

The US EPA on their Integrated Risk Information System (IRIS) reports that no data were located in the available literature that suggested that Cr (VI) is carcinogenic by the oral route of exposure (US EPA 2008). Based on the lack of evidence of carcinogenic activity for chromium (VI) by ingestion, the US EPA and Health Canada have determined that there is not enough information to derive a cancer slope factor for chromium (VI) via the oral route of exposure (US EPA 2008; HC 2004b).

#### Lead

Lead (Pb) is a naturally occurring element found in the earth's crust. While most of the lead found in the environment is the result of anthropogenic activities (including aging plumbing systems and leadbased paints), there are significant natural sources as well, including volcanoes, forest fires, sea spray, and weathering of lead-containing minerals (EC 1996). The different forms of lead found in the environment are governed by factors such as temperature, pH, and the presence of humic materials. Elemental lead occurs rarely in the ambient environment; the most common form of lead in the environment is Pb<sup>2+</sup>. Particulate-bound lead emitted from mining operations, smelters, and combustion sources occurs primarily in the form of lead-sulphur compounds such as PbSO<sub>4</sub>, PbO·PbSO<sub>4</sub>, and PbS (US EPA 1986). In the ambient atmosphere, lead exists primarily in the form of particulate-bound PbSO<sub>4</sub> and PbCO<sub>3</sub>, and is deposited onto soil and water surfaces in this form (ATSDR 2007).

The toxic effects of lead in humans are widely believed to be the same regardless of the route of entry, and are correlated to blood lead (PbB) in the vast majority of studies (ATSDR 2007). The effects from chronic exposure to lead in humans and experimental animals are primarily neurobehavioural, renal, hematological (stippling of red blood cells due to aggregation of ribosomes),



reproductive, and developmental (ATSDR 2007). Well characterized human health effects include neurotoxicity and renal toxicity, which can be severe at blood lead levels greater than 120  $\mu$ g/dL (US EPA 1986). Severe lead exposure in children (PbB above 380  $\mu$ g/dL) can cause coma, convulsions, and even death.

The most commonly reported and well-studied effects of environmental lead exposure are: 1) adverse effects on neurological function and neurobehavioural development in children; and 2) reduced growth rate. However, it remains unclear if lead causes such effects in adults (US EPA 2004). The effects in children often manifest as decreased IQ and memory, decreased gestation period, and retarded growth rate.

Epidemiological studies of occupationally exposed adults were not able to demonstrate an increase in cancers among an exposed population compared to a control group. The US EPA (2004) lists lead as a Group 2B, probable human carcinogen, based on sufficient animal evidence but did not recommend derivation of a quantitative estimate of oral carcinogenic risk due to a lack of understanding of the toxicological and pharmacokinetic characteristics of lead. Health Canada (1992) classified lead as Group IIIB—possibly carcinogenic to humans (inadequate data in humans, limited evidence in animals) according to the classification scheme of the Environmental Health Directorate of Health and Welfare Canada (CCME 1999).

#### Manganese

Manganese (Mn) occurs naturally in rocks and soil combined with other substances, such as oxygen, sulphur, and chlorine. Manganese is essential for good health. Humans are exposed to manganese primarily through the diet. Air also contains low levels of manganese. The most common health problems in workers exposed to high levels of manganese involve the nervous system. These health effects include behavioural changes and other nervous system effects. The inhalation of a large quantity of dust or fumes containing manganese may cause irritation of the lungs which could lead to pneumonia. The EPA concluded that existing scientific information cannot determine whether or not excess manganese can cause cancer (ATSDR 2008c).

#### Mercury

Mercury (Hg) occurs naturally in the environment and exists in several forms. These forms can be organized under two headings: inorganic mercury and organic mercury (e.g. methyl mercury). Inorganic mercury compounds occur when mercury combines with elements such as chlorine, sulfur, or oxygen. These mercury compounds are also called mercury salts.

The most common organic form of mercury is methylmercury. Methylmercury is of particular concern because it can bioconcentrate in certain edible freshwater and saltwater fish, and marine mammals to levels that are many times greater than levels in the surrounding water. Methylmercury is primarily the product of microorganisms (i.e., bacteria and fungi), rather than from anthropogenic sources (ATSDR 1999a).

Mercury toxicity has a large effect on the nervous system. While all forms of mercury can be toxic, methylmercury and metallic mercury vapours are especially harmful because mercury in these forms

can reach the brain, permanently damaging it. This can lead to irritability, shyness, tremours, changes in vision or hearing, and memory problems (ATSDR 1999b). Exposure to high levels of metallic, inorganic, or organic mercury can also result in damage to the kidneys as well as developing fetuses (in the case of maternal exposure) (ATSDR 1999b).

Acute (short-term) exposure to metallic mercury vapour can cause lung damage, nausea, vomiting, diarrhoea, increased blood pressure and heart rate, skin rashes, and eye irritation (ATSDR 1999b).

#### Nickel

Nickel (Ni) is a naturally occurring metal existing in various mineral forms. It may be found throughout the environment including rivers, lakes, oceans, soil, air, drinking water, plants, and animals. Soil and sediment are the primary receptacles for nickel, but mobilization may occur depending on the physico-chemical characteristics of the soil (ATSDR 2005a). Some evidence suggests that nickel may be an essential trace element for mammals (Goyer 1991). As with most metals, the toxicity of nickel is dependent on the route of exposure and the solubility of the nickel compound (Coogan, et al., 1989). In general, the more soluble nickel compounds have a greater toxicity than less soluble forms; however, at the site of tissue deposition, the less-soluble compounds are more likely to be carcinogenic (ATSDR 2005a).

The most common form of nickel toxicity in humans is allergic reactions, generally resulting in skin rashes at the site of contact, but less frequently resulting in other skin rashes or asthma attacks. People generally become sensitive to nickel after prolonged contact with the skin (such as in the case of jewellery). Once sensitized, people can react to low levels of nickel in the air, food or water. Approximately 10 - 20% of people are sensitive to nickel (ATSDR 2005b).

Chronic inhalation exposure to higher levels of nickel can lead to chronic bronchitis and reduced lung function (ATSDR 2005b). Ingesting large amounts of nickel can lead to stomach ache and negative effects on the blood and kidneys (ATSDR 2005b). Animal studies have shown lung and nasal cavity damage as a result of nickel inhalation. Ingestion of large amounts of nickel has caused lung disease in dogs and rats. In rats and mice, effects on the stomach, blood, liver, kidneys immune system, reproductive system, as well as developmental affects, have been documented following the ingestion of large amounts of nickel (ATSDR 2005b).

The carcinogenic activity of nickel is dependent upon the specific species of nickel present. A recent paper by Silvara and Rohan (2007) reviewed the role of nickel and other trace elements in the genesis of cancer and found that more epidemiological studies are needed to establish any link between nickel and cancer.

#### Selenium

Selenium (Se) is a naturally occurring element that is considered to be an essential micronutrient for humans. The primary route of exposure is dietary with the majority of the selenium being excreted via urine (US EPA 1991; HC 1992; IOM 2000; ATSDR 2003).



Despite being an essential nutrient, exposure to higher levels of selenium can cause adverse health effects. These effects are dependent on the route and duration of exposure. Short-term oral exposure to high levels of selenium can cause nausea, vomiting, and diarrhoea. Chronic oral exposure can lead to a disease called selenosis, whose symptoms include hair loss, nail brittleness, and neurological abnormalities such as numbness and odd sensations in the extremities (ATSDR 2003). Short-term inhalation exposure to high levels of selenium or selenium dioxide can cause respiratory tract infection, bronchitis, difficulty breathing, and stomach pains. Chronic inhalation exposure can cause respiratory irritation, bronchial spasms, and coughing (ATSDR 2003).

Animal studies indicate the very high levels of selenium can affect sperm production and the female reproductive system; however, this has not been determined for human exposure (ATSDR 2003).

Selenium is classified as Group 3, not classifiable as a carcinogen, by IARC (1998). Several studies and investigations have remained inconclusive as to the carcinogenic potential of selenium (HC 1992). Following the IARC classification, the United States Environmental Protection Agency (US EPA 1993) classified one form of selenium, selenium sulphide, a main ingredient in anti-dandruff shampoo, as a probable human carcinogen. Selenium sulphide, however, is not readily absorbed through the skin, does not readily dissolve in water, and binds tightly with soil, therefore limiting the potential routes of exposure (ATSDR 2003).

#### Zinc

Zinc (Zn) is the 23rd most abundant element in the earth's crust and is found in air, soil, water and all foods. Zinc is an essential element, necessary for sustaining all life. It stimulates the activity of approximately 100 enzymes, supports a healthy immune system, is needed for wound healing, helps maintain the sense of taste and smell, and is needed for DNA synthesis. Zinc also supports normal growth and development during pregnancy, childhood, and adolescence. The recommended daily allowance of zinc is 15 mg for adult males, 12 mg for adult females, 10 mg for children older than 1 year, and 5 mg for infants 0 - 12 months old (NRC 1989).

Although zinc is essential to human health, levels 10 – 15 times higher than the amount needed for good health can be toxic to humans (ATSDR 2005c). Ingestion of large quantities of zinc, over a short period of time, can lead to stomach cramps, nausea, and vomiting. Chronic exposure to zinc via ingestion can cause anemia and decrease "good" cholesterol in the body. Rats who consumed large amounts of zinc became infertile, but this has not been demonstrated in humans (ATSDR 2005c). Inhalation of large amount of zinc (dust or fumes) can cause a short-term disease called metal fume fever. Long term effects of zinc inhalation are not known. Dermal contact with zinc acetate and zinc chloride is likely a skin irritant in people (ATSDR 2005c).

### 4.2.3 Chemical Screening

A quantitative toxicity assessment was not part of the scope of this HHERA. Where available, baseline and predicted chemical concentrations in air, soil, and water were compared to health-based guidelines, as listed below.

#### 4.2.3.1 Air Quality (Baseline and Predicted Levels)

National Air Quality Objectives (HC 2005; CCME 2000) were used to determine whether chemical concentrations resulting from air emissions pose a potential risk to human or ecological health. If chemical concentrations were greater than applicable health-based regulatory objectives, chemicals were assessed for their potential to cause health effects. If chemical concentrations were lower than the regulatory objectives, then concentrations of chemicals would not affect human or ecological health. Criteria air contaminants are typically assessed in terms of human health, rather than wildlife health, due to a lack of wildlife-specific toxicity data. In lieu of standards for wildlife, the standards for humans were applied to assess potential effects on wildlife.

Some CACs (nitrogen and sulphur dioxides) can also cause damage to vegetation, both directly and through acid deposition. Although not necessarily recognized in Canada, World Health Organization (WHO) guidelines for sulphur dioxide and nitrogen dioxide were used to provide an indication of potential Project effects on vegetation in the assessment area.

#### 4.2.3.2 Surface Soil (Baseline and Predicted Levels)

Yukon Contaminated Sites Regulation (Yukon Government 2002) guidelines for parkland were used to assess potential health risks to human and ecological receptors. Agricultural guidelines are also included for comparison.

#### 4.2.3.3 Water and Sediment Quality (Baseline and Predicted Levels)

In the absence of Yukon-specific guidance, CCME Water Quality Guidelines and Interim Sediment Quality Guidelines for the protection of aquatic life were used (CCME 1999, updated 2010). Where CCME sediment quality guidelines were not available, British Columbia Working Guidelines for Sediment were used (BC MoE 2006a). Health Canada's Guidelines for Drinking Water Quality were used to assess risks to human health (HC 2008).

#### 4.2.3.4 Fish Tissue (Baseline Levels)

Selenium and mercury concentrations were compared to British Columbia Ministry of Environment (BC MoE) and Health Canada guidelines, respectively, as no Yukon-specific guidance is available. The BC MoE guideline for selenium in fish tissue is designed to protect the aquatic life (i.e., fish), and the Health Canada guideline for mercury in fish tissue is protective of human health related to consumption of fish. In the absence of specific guidelines for other trace metal concentrations in fish tissue, analytical results were compared to the following:

- A report on metal levels in Arctic grayling muscle and liver tissue from 54 uncontaminated lakes in British Columbia (Rieberger 1992). Reported results are useful as indicators of metal concentrations in tissues of fish from lakes considered unaffected by human activity.
- The U.S. Environmental Protection Agency's Environmental Residue-Effects Database (ERED) on biological effects and contaminant concentrations in fish species (ERED 2008) for metals not listed by Rieberger (1992) or BC MoE (2006). Where data for Arctic grayling were not listed in ERED, species as similar as possible were used (in some cases data from



only one test species were available). Values from ERED are related to scientifically derived effects and are important for interpreting potential impacts of metals on fish from waterbodies affected by mining activities.

#### 4.2.3.5 Vegetation (Baseline Levels)

Metal concentrations measured in vegetation were compared to dietary guidelines established for domestic cattle (see Section 5.2.6).

### 4.3 **Potential Exposure Pathways**

Potential exposure pathways between COPC and human and ecological receptors include the following, summarized in Table 4.3-1:

- Humans—inhalation of criteria air contaminants
- Humans—uptake of metals from surface water used for drinking, incidental soil ingestion, dermal contact, and consumption of country foods (vegetation, fish, wildlife)
- Vegetation—uptake of metals from soil and groundwater
- Vegetation—direct contact with sulphur and nitrogen oxides and associated acids
- Fish and Aquatic Biota—uptake (i.e., absorption and ingestion) of metals from water and sediment, and ingestion of biota
- Wildlife—inhalation of criteria air contaminants
- **Wildlife**—uptake of metals from water, incidental ingestion of soil, dermal contact, and ingestion of vegetation, fish, other wildlife.

Exposure Pathway	Inclusion in HHERA	Rationale		
Humans				
Inhalation	✓	NND, subsistence, and recreational users frequent the area around the proposed mine and may be exposed to Project air emissions.		
Soil Ingestion	✓	NND, subsistence, and recreational users frequent the area around the proposed mine. Incidental soil ingestion may occur.		
Drinking water from creeks	✓	NND, subsistence, and recreational users in the vicinity of the proposed mine may drink water directly from creeks.		
Ingestion of country foods	✓	NND and others harvesting from the land in the vicinity of the Project may ingest chemicals associated with the Project that have been taken up into country foods (e.g. vegetation, fish, meat).		
Dermal contact	✓	NND, subsistence, and recreational users frequent the area around the proposed mine and may be exposed to Project-related chemicals through direct dermal exposure.		

Table 4.3-1: Potential Pathways of Exposure

Exposure Pathway	Inclusion in HHERA	Rationale
Ecological Receptors		
Inhalation	✓	A variety of wildlife species exist in the vicinity of the proposed Project.
Direct ingestion (from surface soil, sediment and surface water)	~	A variety of wildlife and fish species exist in the vicinity of the proposed Project.
Dermal contact/root uptake	✓	A variety of wildlife, fish, and plant species exist in the vicinity of the proposed Project.
Ingestion of plants and animals	~	A variety of wildlife species exist in the vicinity of the proposed Project. Contaminants may be passed up through the food chain.

Conceptual models for potential human and ecological exposure pathways are presented in Figures 4.3-1 and 4.3-2, respectively.

A quantitative exposure assessment was not part of the scope of work, but was considered if the qualitative investigation suggested potential health risks to human or ecological receptors.

# 5 DESCRIPTION OF BASELINE CONDITIONS

To effectively assess risks to potential human and ecological receptors, baseline chemical concentrations in air, soil, water, and biota must be established to evaluate risks attributable to Project-related emissions.

### 5.1 Baseline Conditions

The baseline assessment follows the risk assessment framework outlined in Section 2.2. Potential human and ecological health risks as a result of exposure to baseline chemical concentrations were assessed qualitatively.

The assessment of potential health effects draws on information from other Valued Components, including air quality (criteria air contaminants and hazardous air pollutants), water quality (metals and chemical hazards), soil quality (metals), and fish tissue (metals). Baseline data have been collected for metals in soil, sediment, water, fish tissue, and vegetation. Complete data sets are provided by discipline in the Project Proposal's Section 4 (and associated Appendices). Data relevant to the HHERA are summarized below (Tables 5.2-1 to 5.2-6).

These data were sufficient for a qualitative evaluation of baseline conditions for the HHERA; however, some data gaps were identified. Firstly, no chemical information has been collected for wildlife tissue (e.g. moose). If significant Project effects are predicted with respect to metal inputs to the environment, then monitoring of wildlife tissue may be recommended. Secondly, no data are

available for berries or other vegetation typically consumed by humans. Sampling of berries in the assessment area may be suggested if significant Project effects are predicted in Section 6.

Baseline concentrations of air contaminants were not directly assessed, as there are no other facilities in the assessment area and no appropriate air quality monitoring stations to provide data. Baseline concentrations were assumed similar to other pristine areas, and to be below all regulatory health-based objectives.

### 5.2 Baseline Concentrations

### 5.2.1 Air Quality

Little is known of the existing air quality regime in the Project assessment area. Any existing air contaminants will have natural sources or be the result of long distance importation. Due to the remote location gaseous air-contaminants should be minimal. Any baseline air contaminants are likely to be  $PM_{2.5}$ , as only fine PM will survive long-range transport. In this context, the baseline Project site air quality should be pristine. Baseline air quality conditions are discussed further in the Project Proposal (Sections 1.1.7 and 6.6.1.2).

### 5.2.2 Surface Soil

Baseline metal concentrations were measured in overburden samples (0.5 to 6 m depth) and surface soil samples (0 to 0.5 m depth). Only concentrations of metals at depths between 0 and 1.5 m were considered in the HHERA (n=12), as plant roots and wildlife are unlikely be exposed to deeper soils.

Baseline results for soil are detailed in Appendix 6 (Surficial Geology, Terrain, and Soils). As certain samples were not representative of soil-map unit conditions, these data were not included in the soils or health assessments (e.g. a soil sample collected at a seep) (see Section 6.4 of the Project Proposal). Once appropriate baseline metals were determined for soil map units, only three metals were found to be naturally occurring in exceedance at the landscape level: chromium, nickel and arsenic. Although one baseline surface soil sample had a slightly elevated selenium concentration, this area is anticipated to be covered during operations and will not be accessible to potential receptors, and therefore, was not considered further.

Chromium, nickel, and arsenic were carried forward as COPCs in the HHERA (Table 5.2-1). For the purpose of the HHERA, metal concentrations in soil were compared to the Yukon Contaminated Sites Regulation (CSR) for Parkland. However, if more conservative CCME guidelines were exceeded when Yukon guidelines were met, these exceedances were noted and assessed (see Table 5.2-1).

COPC	Depth of Exceedance (m)	Maximum Concentration (mg/kg)	Guideline Concentration <sup>ь</sup> (mg/kg)	Proportion of Samples above Guideline (%) <sup>d</sup>	Relevant Pathway
		880	15	83	Groundwater used for drinking water
	0 – 1.1		20	83	Groundwater flow to surface water used by freshwater aquatic life
Arsenic			25	83	Livestock ingesting soil and fodder
			50	67	Toxicity to soil invertebrates and plants
			100	42	Human inadvertent intake of contaminated soil
Chromium	0 – 0.06	105	60	8	Groundwater used for drinking water; Groundwater flow to surface water used by freshwater aquatic life
			100	8	Human intake of contaminated soil
Nickel	0 – 0.06	54	50 <sup>°</sup>	8	Environmental health

#### Table 5.2-1: Baseline COPCs in Surface Soil<sup>a</sup>

NOTES:

<sup>a</sup>0 – 1.5 m depth

<sup>b</sup> Yukon CSR for Parkland

<sup>c</sup> CCME guideline for Parkland. Although Yukon criteria were not exceeded for nickel and selenium, the more conservative CCME guidelines were exceeded.

<sup>d</sup> *n* = 12

### 5.2.3 Surface Water

Baseline water quality sampling was conducted in the Lynx Creek, Haggart Creek, Dublin Gulch, and Eagle Creek watersheds each year 1993 through 1996 and 2007 through 2010. Samples were analyzed for general parameters, nutrients, dissolved organic carbon, cyanide, and total and dissolved metals. Baseline results for water quality are detailed in the Water Quality and Aquatic Biota baseline report. Only samples collected during 2007 through 2010 were considered in the HHERA. Baseline COPCs in surface water for samples collected during 2007 through 2009 are summarized in Table 5.2-2. Detailed water quality data for 2010 are presented in Appendix 16 (Environmental Baseline Report: Water Quality and Aquatic), and are summarized in Table 5.2-3. Data for 2010 are summarized separately, as results included a sampling event during spring freshet (May 2010), resulting in many more elevated baseline metal concentrations than during the other sampling events.



				,	
Parameter (total)	Maximum Concentration (mg/L)	Aquatic Life (AL) Guideline Concentration (mg/L)	Proportion of Samples Above AL Guideline (%) <sup>b</sup>	Drinking Water (DW) Guideline (mg/L)	Proportion of Samples Above DW Guideline (%) <sup>b</sup>
Aluminum	2.4	0.1 (total), pH ≥6.5	5	0.1 <sup>c</sup>	5
Arsenic	0.08	0.005	66	0.01	51
Cadmium	0.0004	0.00001 to 0.00006 <sup>a</sup>	4	0.005	0
Copper	0.007	0.002 to 0.004 <sup>a</sup>	0.7	≤1.0 <sup>c</sup>	0
Iron	4.8	0.3	1	≤0.3 <sup>c</sup>	1
Lead	0.0097	0.001 to 0.007 <sup>a</sup>	0.7	0.01	0
Cyanide	0.016	0.005	6	0.2	0

#### Table 5.2-2: Baseline COPCs in Surface Water (All Watersheds: 2007 – 2009)

NOTES:

<sup>a</sup> Varies with hardness, the range of metal values is presented for hardness of 20 - 180 mg/L

<sup>b</sup> n = 147

<sup>c</sup> aesthetic objective

Parameter (total)	Maximum Concentration (mg/L)	Aquatic Life (AL) Guideline Concentration (mg/L)	Proportion of Samples Above AL Guideline (%) <sup>b</sup>	Drinking Water (DW) Guideline (mg/L)	Proportion of Samples Above DW Guideline (%) <sup>b</sup>
Aluminum	5.2	0.1 (total), pH ≥6.5	43	0.1 <sup>c</sup>	43
Arsenic	0.08	0.005	75	0.01	44
Cadmium	0.0004	0.00001 to 0.00006 <sup>a</sup>	29	0.005	0
Copper	0.016	0.002 to 0.004 <sup>a</sup>	22	≤1.0 <sup>c</sup>	0
Iron	9.4	0.3	32	≤0.3 <sup>c</sup>	32
Lead	0.01	0.001 to 0.007 <sup>a</sup>	9	0.01	2.9
Cyanide	0.008	0.005	10	0.2	0

#### Table 5.2-3: Baseline COPCs in Surface Water (All Watersheds: 2010)

NOTES:

<sup>a</sup> Varies with hardness, the range of metal values is presented for hardness of 20 – 180 mg/L

<sup>b</sup> n = 68

<sup>c</sup> aesthetic objective

Total arsenic levels consistently exceeded CCME guidelines for protection of aquatic life in all watersheds sampled. For samples collected during 2007 through 2009, other metals listed in Table 5.2-2 and cyanide exceeded guidelines at only a few sites, and no exceedances were reported for Eagle Creek. In 2010, guideline exceedances occurred in all watersheds, and almost all exceedances occurred in samples collected in May, during high spring flows. Of 68 samples analyzed, 75% had arsenic concentrations exceeding the aquatic life guideline, and 44% exceeded the drinking water guideline.

A comparison of maximum concentrations with Canadian Drinking Water Guidelines showed that only aluminum, arsenic, and iron were above guideline levels. Cyanide concentrations in surface water were all below the drinking water guideline. Two samples collected from Eagle Creek in May 2010 had lead concentrations equal to the drinking water guideline of 0.01 mg/L. Arsenic did not exceed the drinking water guideline in Lynx Creek in any year. Only two samples from Haggart Creek and Eagle Creek exceeded the aesthetic objective for iron for the 2007 – 2009 data set, but in May 2010, the aesthetic objective was exceeded at most locations in the Dublin Gulch, Haggart, and Eagle Creek systems. A few samples in each of the watersheds exceeded the drinking water guideline for aluminum, with the exception of samples collected in May 2010, when most concentrations were above the guideline. It should be noted that drinking water guidelines for aluminum and iron are aesthetic objectives (e.g. taste of the water), rather than health effect levels.

It should also be noted that site-specific water quality objectives (SS WQO) for protection of aquatic life have been recommended for the Project (see discussion in Section 6.5.5 of the Project Proposal). CCME (2003) provides guidance in developing SS WQO. Baseline arsenic concentrations are up to ten times higher than CCME water quality guidelines year round in Dublin Gulch and occasionally in Haggart Creek (maximum values), indicating the need for site-specific objectives. Although SS WQO values have not been considered in this baseline assessment, these values have been considered in the water quality effects assessment (Section 6.5 of the Project Proposal).

### 5.2.4 Sediment

Sediment was sampled in various years over the years 1976 through 2010 in the Lynx Creek, Haggart Creek, Dublin Gulch, and Eagle Creek watersheds. Data for all years and sampling sites are presented in Appendix 16 (Water Quality and Aquatic Biota). A summary of data for sediment collected in 2007, 2009 and 2010 is presented in Table 5.2-4.

Metals data for the fine (<63  $\mu$ m) sediment fraction were similar to the water quality data in terms of high levels of arsenic at all sites and only periodic guideline exceedances for other metals. Arsenic levels were higher than the CCME Probable Effects Level (PEL) for protection of aquatic life in all samples analyzed. Nickel concentrations in sediment also exceeded the guideline at most sites.

Parameter (total metals)	Maximum Concentration (mg/kg dry weight)	Aquatic Life Guideline Concentration (mg/kg dry weight) ISQG/PEL <sup>a</sup>	Proportion of Samples Above AL Guideline (%) <sup>b</sup>
Arsenic	608	5.9/17	100 <sup>c</sup>
Cadmium	1.1	0.6/3.5	7
Chromium	61	37.3/90	6 <sup>c</sup>
Copper	53	35.7/197	10 <sup>c</sup>
Lead	120	35/91.3	17 <sup>c</sup>
Nickel	68	16/75 <sup>d</sup>	90 <sup>e</sup>
Zinc	130	123/315	8 <sup>c</sup>

 Table 5.2-4:
 Baseline COPCs in Sediment (2007 – 2010)

NOTES:

<sup>a</sup> CCME Interim sediment quality guideline (ISQG)/Probably Effects Level (PEL)

<sup>b</sup> n = 54

<sup>c</sup> Samples with exceedances include replicates collected from the same creek on the same day (upstream, mid, and downstream samples).

<sup>c</sup> Three of the samples were replicates from the same creek on the same day (upstream, mid, downstream).

<sup>d</sup> BC Working Sediment Guidelines

<sup>e</sup> Dublin Gulch was the only creek in which all three replicate samples were below the guideline (on two different sampling dates).

### 5.2.5 Fish Tissue

A total of 21 Arctic grayling and 53 slimy sculpin tissue samples were analyzed for metal concentrations in 2009. For Arctic grayling, all metals except mercury were analyzed in both muscle and liver tissue. Mercury was analyzed in muscle tissue only. For slimy sculpins (relevant to wildlife consumption), whole fish were analyzed for metals (except mercury).

A full description of fish tissue results is presented in Appendix 5 (Environmental Baseline Report: Fish and Fish). Metals that exceeded any of the available guideline or reference values in Arctic grayling tissue are presented in Table 5.2-5 below. As reference values for comparison of whole fish concentrations for sculpin are not available (with the exception of selenium), sculpin values are not listed in Table 5.2-5. Selenium concentrations in all fish tissues are summarized by watershed in Table 5.2-6.

Parameter	Tissue	Reference or Guideline Value Exceeded	Source
Arsenic	liver, muscle	BC reference lakes	Rieberger (1992)
Aluminum	liver, muscle	BC reference lakes	Riebeger (1992)
Barium	liver	BC reference lakes	Riebeger (1992)
Cadmium	liver	BC reference lakes	Riebeger (1992)
Selenium	liver, muscle	Protection of aquatic life	BC MoE (2006)
Manganese	liver, muscle	BC reference lakes	Riebeger (1992)
Zinc	liver, muscle	BC reference lakes	Riebeger (1992)

 Table 5.2-5:
 Baseline COPCs in Arctic Grayling Tissue

Mean Selenium Concentration (mg/kg wet weight)									
Arctic Grayling Liver			Arctic Grayling Muscle		Whole Slimy Sculpin			Aquatic Life	
Haggart Creek	Lynx Creek	Dublin Gulch	Haggart Creek	Lynx Creek	Dublin Gulch	Haggart Creek	Lynx Creek	Ironrust Creek	Guideline
4.79	6.42	5.80	2.10	2.11	2.54	1.81	1.49	1.85	1.0

 Table 5.2-6:
 Baseline Selenium Concentrations in Fish Tissues

Mercury levels measured in all Arctic grayling muscle tissue were below Health Canada guidelines for human consumption of fish tissue. Baseline selenium concentrations in both Arctic grayling muscle and liver tissue were above the BC MoE guideline for protection of aquatic life. Selenium concentrations in slimy sculpin also exceeded this guideline.

#### 5.2.6 Vegetation

Vegetation species commonly browsed by wildlife (willow, sedge, blue-joint, northern rough fescue) were collected from nine locations in and around the Vegetation LAA and analyzed for trace metals. Metal concentrations for these 16 samples are detailed in Appendix 11 (Environmental Baseline Report: Vegetation). These data, along with metal concentrations in soils, were used to characterize baseline conditions in plants and soils and to assess soil suitability for reclamation.

Wild berries and other types of vegetation may be harvested by First Nations and other people in the area. These species were not selected for baseline sampling, as forage species used by wildlife were deemed sufficient surrogates for use in the qualitative human health assessment. VIT will include berry-producing species (or other vegetation species of concern to the local community) in the program designed to monitor effects of dustfall during Project operations.

As tolerances of wild ungulates for the trace metals are not known, dietary guidelines established for domestic cattle were used to predict effects on wild ungulates. This approach has been used in the assessment of mine projects in British Columbia and NWT previously. All metals were below toxic levels for dietary intake by cattle for all sites and for all vegetation species sampled based on dietary guidelines outlined in Puls (1994).

## 5.3 Baseline Exposure and Toxicity Assessment

Although concentrations in the assessment area of several metals exceed the soil quality, water quality, and sediment quality guidelines, there does not appear to be extensive human use of the area at this point, and it is unlikely that people are at significant risk from these exposures.

Environmental quality guidelines are meant to be very conservative to ensure protection of wildlife and vegetation. During the field programs, no evidence of stressed vegetation or wildlife was encountered.



### 5.4 Baseline Risk Characterization

Although baseline concentrations of several metals were elevated in soil, surface water, sediment, or fish tissue, arsenic was the only metal across all environmental media that consistently was above guidelines and reference values. One of the most important processes influencing the bioavailability of arsenic in soil is its sorption onto solids or particulates. Sorption is controlled by soil pH and the amount of clay, iron, aluminum, calcium, and phosphorus present. Uptake of arsenic through ingestion of contaminated forage is not considered an important route of exposure because concentrations of arsenic in terrestrial plants are generally low. Direct ingestion of arsenic from soil can be a major source of dietary arsenic, however, in cattle only about 1% of this is absorbed, with the remainder excreted directly (CCME 2001). Metal concentrations in all baseline vegetation samples collected from the assessment area were below levels considered toxic for cattle, used in this assessment as a surrogate for wild ungulates. Furthermore, there did not appear to be a relationship between arsenic concentrations in paired soil and vegetation samples, indicating that these vegetation species were not accumulating arsenic from the soil.

An assessment of baseline levels of metals in wildlife tissue, and the subsequent exposure of predatory wildlife and humans to metals via consumption of meat, cannot be made as data have not been collected to date.

The presence of healthy vegetation, fish, and wildlife in the assessment area indicates that ecological receptors are not likely adversely affected by elevated baseline metal concentrations in soil or in the aquatic environment. Areas that contain ore bodies often have mineralized soil associated with them, and thus have naturally elevated concentrations of some metals, to which the local ecosystem has adapted. Development of site-specific objectives (i.e., "acceptable" metal concentrations) for such sites is often appropriate. Metal levels in water are consistent with a mineralized area and likely also reflect previous disturbance of substrates during placer mining.

Potential exposure of humans to existing metal concentrations in the assessment area is not expected to cause adverse health effects. Although baseline levels of some metals in surface water exceed drinking water guidelines, humans are not expected to use these creeks as a regular source of drinking water. That said, it would be prudent to advise local First Nations and other people using the area to avoid drinking surface water for any appreciable period of time, based on the naturally occurring elevated arsenic concentrations.

Any changes from baseline conditions will be considered in the Project effects assessment (see Section 6 of the Project Proposal).

# 6 PROJECT EFFECTS ASSESSMENT

### 6.1 Chemical Screening

#### 6.1.1 Air Quality

#### 6.1.1.1 Criteria Air Contaminants

#### **Construction Phase**

Predicted maximum CAC concentrations are below the National Ambient Air Quality Objectives (NAAQO) except for the TSP and  $PM_{2.5}$ . The maximum 24-hour TSP and  $PM_{2.5}$  concentrations of 1,251 µg/m<sup>3</sup> and 35.1 µg/m<sup>3</sup>, respectively, are predicted at the south perimeter of the mine site in an area where the terrain rises rapidly. Plumes impinge the steep terrain in this area, and this phenomenon commonly leads to overestimates in dispersion modelling exercises. The area in which these exceedances are predicted to occur lies outside of the mine disturbance boundary and is also very small. The dispersion model also did not account for wet scavenging effects (natural dust suppression by rain and snow), which would decrease predicted ambient CAC concentrations. Given the above, these predicted CAC concentrations are not anticipated to pose a health risk.

Predicted  $SO_2$  concentrations are below the World Health Organization (WHO) guideline for protection of forested ecosystems. Modelled maximum ground-level concentrations for  $NO_2$  (24-hour) were predicted to be moderately in excess of these conservative WHO benchmarks. However, given that  $NO_2$  rapidly disperses, and predicted annual averages were below the WHO guideline, effects on vegetation are not expected to manifest in the assessment area and were not considered further.

Substance	Averaging Period	Maximum Predicted Concentration (µg/m³)	Regulatory Objective (µg/m³)
TSP	24-hour	1,251	120 <sup>a</sup>
	Annual	59.0	70 <sup>a</sup>
PM <sub>2.5</sub>	24-hour	35.1	30 <sup>b</sup>
	1-hour	137	400 <sup>a</sup>
NO <sub>2</sub>	24-hour	99.7	200 <sup>a</sup> 75 <sup>c</sup>
	Annual	12.2	100 <sup>a</sup> 30 <sup>c</sup>
	1-hour	1,195	35,000 <sup>a</sup>
CO	8-hour	988	15,000 <sup>a</sup>

Table 6.1-1:	Maximum Predicted CAC Concentrations during Project Construction
	maximum redicted OAO oblicentrations during reject oblist detion

Substance	Averaging Period	Maximum Predicted Concentration (µg/m <sup>3</sup> )	Regulatory Objective (µg/m <sup>3</sup> )
	1-hour	0.52	900 <sup>a</sup>
SO <sub>2</sub>	24-hour	0.31	300 <sup>ª</sup> 100 <sup>c</sup>
	Annual	0.03	60 <sup>a</sup> 20 <sup>c</sup>

#### NOTES:

Values in **bold** identify exceedance of applicable regulatory objectives.

<sup>a</sup> National Ambient Air Quality Objectives, NAAQO. Maximum Allowable Objective Level. (Government of Canada 2004).
 <sup>b</sup> Canadian Council of Ministers of the Environment (CCME 2000) Canada-wide Standard for Respirable Particulate Matter (PM<sub>2.6</sub>). This objective is referenced to the 98<sup>th</sup> percentile 24-h concentration, averaged over three consecutive years.

°WHO (2000) guidelines to protect forested ecosystems from SO<sub>2</sub> and NO<sub>2</sub>.

#### **Operations Phase**

Predicted maximum CAC concentrations are below the NAAQO, with the exception of TSP. Like the construction case, the 24-hour TSP maximum (338  $\mu$ g/m<sup>3</sup>) is found at the south perimeter of the mine site in an area where the terrain rises rapidly. Plumes impinge the steep terrain in this area. Like the construction case, this phenomenon may have produced overestimates in dispersion modelling exercises. Further, the area in which the exceedance occurs is very small, and the dispersion model did not account for wet scavenging effects (natural dust suppression by rain and snow), which would decrease predicted ambient CAC concentrations. As a result, this predicted concentration is not anticipated to pose a health risk.

Predicted  $SO_2$  and  $NO_2$  concentrations were similar to the construction phase, and are not considered a risk to vegetation in the assessment area.

CAC	Averaging Period	Maximum Predicted Concentration (µg/m³)	Regulatory Objective (µg/m <sup>3</sup> )
TOD	24-hour	397	120 <sup>a</sup>
TSP	Annual	38.4	70 <sup>a</sup>
PM <sub>2.5</sub>	24-hour	19.3	30 <sup>b</sup>
	1-hour	145	400 <sup>a</sup>
NO <sub>2</sub>	24-hour		200 <sup>ª</sup> 75 <sup>°</sup>
	Annual	15.8	100 <sup>a</sup> 30 <sup>c</sup>
<u> </u>	1-hour	1,311	35,000 <sup>a</sup>
CO	8-hour	1,085	15,000 <sup>a</sup>

Table 6.1-2: Maximum Predicted CAC Concentrations during Project Operations

CAC	Averaging Period	Maximum Predicted Concentration (µg/m <sup>3</sup> )	Regulatory Objective (µg/m <sup>3</sup> )	
	1-hour	0.40	900 <sup>a</sup>	
SO <sub>2</sub>	24-hour	0.21	300 <sup>a</sup> 100 <sup>c</sup>	
	Annual	0.02	60 <sup>a</sup> 20 <sup>c</sup>	

#### NOTES:

Values in **bold** identify exceedance of applicable regulatory objectives.

<sup>a</sup> National Ambient Air Quality Objectives, NAAQO. Maximum Allowable Objective Level.

<sup>b</sup> Canadian Council of Ministers of the Environment (CCME 2000) Canada-wide Standard for Respirable Particulate Matter (PM<sub>2.5</sub>). This objective is referenced to the 98<sup>th</sup> percentile 24-h concentration, averaged over three consecutive years. <sup>c</sup> WHO (2000) guidelines to protect forested ecosystems from SO<sub>2</sub> and NO<sub>2</sub>.

#### 6.1.1.2 Metals in Dustfall

Dustfall effects result from deposition of  $PM_{2.5}$  emitted by fugitive sources.  $PM_{2.5}$  generated by combustion sources is much smaller and remains airborne for much longer periods of time. Whereas TSP is removed from the atmosphere through gravitational settling,  $PM_{2.5}$  is susceptible to long range transport. Consequently,  $PM_{2.5}$  is not included as part of dustfall effects. As the construction phase is relatively short-lived, total dustfall will be smaller during construction than during the operations phase. As a result, dustfall is considered only during the operations phase of the Project.

A metal speciation profile is provided in Section 6.6 of the Project Proposal, and potential effects of metals in dustfall on soil quality are discussed in Section 6.1.2 below.

#### 6.1.2 Soil Quality

Dustfall results were used to predict loading of metals to surface soil. The potential loading of 18 metals into the environment were modeled (Table 6.1-3). Modeling is described in detail in Section 6.4.1.10 of the Project Proposal.

Table 6.1-3:	Metals Considered in Soil Loading
--------------	-----------------------------------

Metals								
Antimony	Arsenic	Barium	Beryllium	Cadmium	Chromium	Cobalt	Copper	Lead
Mercury	Molybdenum	Nickel	Selenium	Silver	Thallium	Tin	Vanadium	Zinc

For the purpose of this assessment, metals that exceeded health-based guidelines at baseline were considered further in the effects assessment if the predicted concentration in surface soil increased by ≥10% from baseline over eight years of mine operations, or if a metal that was below guidelines at baseline but was predicted to increase above guidelines during operations. Ten percent was chosen to reflect a true change in concentration from baseline to operations, rather than potential analytical



or sampling error. Only arsenic was found to have a 10% increase or greater in baseline conditions that exceeded soil quality guidelines and was carried forward in the effects assessment.

### 6.1.3 Water Quality

Water quality predictions have been used to guide Project design, mitigation, and monitoring, so that water quality guidelines for protection of human health and aquatic life, and site-specific water quality objectives, are met (see Section 6.5 of the Project Proposal). For mine operations, several design features and mitigation measures have been developed to reduce the potential for Project-related adverse effects on stream water quality, the most important of which is the mine water treatment plant (MWTP). The MWTP will reduce levels of nitrogen, phosphorus and metals. Effluent from the MWTP will be discharged to Haggart Creek through a diffuser pipe on the creek bed. Water quality in receiving streams will be protected using:

- Sediment control points
- Seepage collection ponds
- Feed and product ponds for MWTP
- Diversion ditches to keep non-contact water away from mine activities
- Heap design with double and triple liners, a leak detection and recovery system, and seepage collection
- Recycling of water from open pit and waste rock storage areas to the heap leach facility
- Detoxification facility for removal of cyanide from heap leach solution.

During closure and post-closure, remediation measures will be in place to protect water quality, including treatment wetlands for passive treatment of metals potentially from the waste rock storage areas and the heap leach facility. In addition, monitoring of site discharges is anticipated to continue for at least 15 years (to year 25). These remediation measures are detailed in Section 6.5 of the Project Proposal.

Two areas where contact surface water and ecological receptors (e.g. terrestrial wildlife) could potentially interact are at the open pit and Platinum Gulch. The open pit will have some waste rock left in it during Year 10, and a small pit lake will form, which will drain to Platinum Gulch. If monitoring indicates that further treatment of open pit water is needed, pit lake runoff will first flow into a sub-surface flow treatment wetland at the Platinum Gulch waste rock storage area. However, mitigation measures may be required pending water quality monitoring results, so that wildlife is discouraged from coming in contact with surface water.

Given implementation of the above mitigation measures and treatment technologies, water quality is not anticipated to pose a health risk to humans or ecological receptors, and therefore, is not considered further in this health effects assessment. Should monitoring during and after mine operations reveal that concentrations of metals are above health-based guidelines where human and/or ecological receptors may be exposed, a quantitative health risk assessment may be warranted.

### 6.2 Exposure Assessment

#### 6.2.1 Air Quality

With respect to air quality, 24-hour TSP concentrations are predicted to exceed the regulatory objective during the construction and operations phases. However, as  $PM_{2.5}$  concentrations are more indicative of particulate matter that poses a health concern for humans than TSP (TSP includes larger particulates), and elevated TSP is localized to a small area, this is not of concern to human or ecological health. The predicted 24-hour  $PM_{2.5}$  concentration during the construction phase slightly exceeds the regulatory objective; however, the construction phase is relatively short-lived (1.7 years) and  $PM_{2.5}$  exposure consequently is not anticipated to affect human health particularly given the isolated nature of the site.  $PM_{2.5}$  concentrations meet guidelines during the operations phase.

### 6.2.2 Soil Quality

Modeling of dust metal-concentrations indicate that only arsenic could potentially increase greater than 10% of baseline conditions. This exceedance would be in a limited area of the soils RAA (Table 6.2-1). Given the limited area of potential arsenic effects and the conservatism built into the modeling exercise, it is unlikely that there would be a significant increase in arsenic exposure for either humans or wildlife in the assessment area. However, long-term soil monitoring will be conducted in areas outside the Project footprint where arsenic levels are predicted to be further elevated during mine operations, as outlined in the Project Proposal (see Section 6.4).

Assessment Area Outside Footprint	Soil Map Unit	Soil Reclamation Suitability	Baseline Soil Arsenic Concentration (mg/kg)	Operations Soil Arsenic Concentration	Percent Increase	Area of Concentration Increase (ha) Outside Footprint <sup>a</sup>
				56.1	28.4	2.0
				48.5	11.1	43.0
	D3x	Unsuitable	43.7	52.9	21.0	9.6
				56.1	28.3	1.5
				52.8	20.8	3.7
LAA		Unsuitable	88.9	100.6	13.2	0.0
				102.5	15.3	2.1
	Dav			102.3	15.0	0.1
	D4x			99.2	11.5	6.6
				102.6	15.4	2.8
				98.3	10.6	0.6
Subtotal						72.0

Table 6.2-1: Predicted Soil Element Loading in the Soils LAA and RAA

Assessment Area Outside Footprint	Soil Map Unit	Soil Reclamation Suitability	Baseline Soil Arsenic Concentration (mg/kg)	Operations Soil Arsenic Concentration	Percent Increase	Area of Concentration Increase (ha) Outside Footprint <sup>a</sup>
		Unsuitable	88.9	100.6	13.2	9.1
	D4x			100.4	13.0	7.1
RAA				98.7	11.0	2.3
				99.2	11.5	0.0
				102.6	15.4	0.0
Subtotal	18.5					
Total						90.5

NOTES:

<sup>a</sup> Within the mine footprint, the Eagle Pup and Platinum Gulch WRSAs and the Open Pit will receive arsenic loading greater than 10% (13.4 ha) and are not included as part of the assessment as these areas will be capped with soil and reclaimed. D3x: Histic Dystric Turbic Cryosol/ Histic Dystric Static Cryosol

D4x: Histic Eutric Turbic Crvosol/ Histic Eutric Static Crvosol

## 6.3 Toxicity Assessment

Arsenic compounds are considered both carcinogenic and non-carcinogenic for human exposure. One of the most common health risks of chronic oral exposure to inorganic arsenic is skin damage, potentially leading to skin cancer (ATSDR 2007). Oral exposure to inorganic arsenic may also result in neurotoxicity (RAIS 1992), and nausea, vomiting, and diarrhoea after repeated exposure to lower doses (ATSDR 2007). As the Project is not anticipated to appreciably increase levels of arsenic exposure above baseline conditions for humans and ecological receptors (see Section 6.2), a toxicity assessment was not conducted.

However, if a quantitative risk assessment is found to be warranted in the future, specific toxicity reference values (TRVs) obtained from reputable government agencies (e.g. US EPA) or peer reviewed literature will be used for comparison with predicted exposure concentrations.

## 6.4 Risk Characterization

Given that water quality (with mitigation) and air quality are not anticipated to be adversely affected by mine operations, and, with the exception of arsenic, metal loading to soil is predicted to be minimal, the proposed mine is not anticipated to adversely affect human or ecological health.

As loading of arsenic to surface soil during mine operation is limited spatially, it is not anticipated to pose a risk to human or ecological health. Additionally, the area supports healthy vegetation and wildlife populations even though existing (baseline) arsenic concentrations are elevated above health-based guidelines, indicating that organisms in the region have adapted and that site-specific, rather than generic regulatory guidelines are appropriate for the site.

# 6.5 Uncertainty Analysis

Uncertainties are inherent in the HHERA process. The most effective ways to decrease uncertainty are to collect site-specific data and to quantitatively assess potential risks.

With the exception of wildlife tissue (e.g. moose) and berries, baseline site-specific chemical data were collected for the Project (i.e., soils, forage vegetation, surface water, sediment, fish tissue), allowing for accurate evaluation of existing metal concentrations in these media.

A qualitative, rather than quantitative, health assessment was conducted for the Project, introducing uncertainty around potential receptor uptake of metals from soil, water, and food. Exposure predictions and comparison to toxicity reference values could not be made. Instead, the likelihood of receptors being exposed to adverse concentrations of arsenic was evaluated, based on soil and water quality modeling (predicted concentrations and spatial extent), use of the area by potential receptors (potential for exposure), and the inclusion of mitigation measures (removal of either the toxicity or the exposure pathway). While quantitative assessments introduce a new set of uncertainties (assumptions), a high degree of conservatism is built in, ensuring that no risk exists when a 'no risk' conclusion is made.

# 7 COMMITMENTS

The contaminant-related commitments made in the air quality, soil quality, and water quality assessments are adequate and will need to be implemented to protect human and ecological health receptors in the assessment area (Section 6 of the Project Proposal). In particular, monitoring of metals in surface water and soils during mine operations, and of surface water post-closure, will be required to ensure that concentrations do not pose a health risk to humans or fish and wildlife.

# 8 CLOSURE

Stantec has prepared this report for the sole benefit of VIT for the purpose of documenting baseline conditions in anticipation of an environmental assessment under the *Yukon Environmental and Socio-Economic Assessment Act* (YESAA). The report may not be relied upon by any other person or entity, other than for its intended purposes, without the express written consent of Stantec and VIT. Any use of this report by a third party, or any reliance on decisions made based upon it, are the responsibility of such third parties.

The information provided in this report was compiled from existing documents and data provided by VIT, and by field data compiled by Stantec. This report represents the best professional judgment of our personnel available at the time of its preparation. Stantec reserves the right to modify the contents of this report, in whole or in part, to reflect any new information that becomes available. If any conditions become apparent that differ substantially from our understanding of conditions as presented in this report, we request that we be notified immediately to reassess the conclusions provided herein.



#### 9 REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR). 1999a. Toxicological Profile for Mercury. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=115&tid=24. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 1999b. ToxFAQs for Mercury. April 1999. Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=113&tid=24. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2003. Toxicological profile for selenium. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=153&tid=28. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological profile for copper. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=206&tid=37. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2005a. Toxicological profile for nickel. Available at: http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=245&tid=44. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2005b. ToxFAQs for Nickel. Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=244&tid=44. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2005c. Toxicological profile for zinc. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=302&tid=54. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological profile for arsenic. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=22&tid=3. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2007. ToxFAQs for Lead. Available at: http://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=93&tid=22. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2008a. Toxicological profile for cadmium. Draft for Public Comment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=48&tid=15. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2008b. Toxicological profile for chromium. Draft for public comment. Atlanta, Georgia, US Department of Health and Human Services, Public Health Service. Available at: http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=62&tid=17. Accessed: September 2010.
- Agency for Toxic Substances and Disease Registry (ATSDR). 2008c. Toxicological profile for manganese. Draft for Public Comment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. Available at:

http://www.atsdr.cdc.gov/ToxProfiles/TP.asp?id=102&tid=23. Accessed: September 2010.

- BC Ministry of Environment (BC MoE). 2006a. A Compendium of Working Water Quality Guidelines for British Columbia.
- BC Ministry of Environment (BC MoE). 2006b. British Columbia Approved Water Quality Guidelines.
- Canadian Council of Ministers of the Environment (CCME). 1999. *Canadian Environmental Quality Guidelines*. 1999 and Updates.
- Canadian Council of Ministers of the Environment (CCME). 2000. *Canada-wide Standard for Respirable Particulate Matter (PM) and Ozone*. Available at: http://www.ccme.ca/assets/pdf/pmozone\_standard\_e.pdf. Accessed: August 2010.
- Canadian Council of Ministers of the Environment (CCME). 2001. Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health. Arsenic. In: *Canadian Environmental Quality Guidelines*, 1999 (updated 2001), Canadian Council of Ministers of the Environment, Winnipeg, MB.
- Canadian Council of Ministers of the Environment (CCME). 2003. *Guidance on the Site-Specific* Application of Water Quality Guidelines in Canada:. Procedures for Deriving Numerical Water Quality Objectives. Available at: http://ceqg-rcqe.ccme.ca/.
- Coogan, T.P., D.M. Latta, E.T. Snow, and M. Costa. 1989. Toxicity and carcinogenicity of nickel compounds, In: *Critical Reviews in Toxicology*, Vol 19. McClellan, R.O., ed., CRC Press, Boca Raton, FL. pp. 341-384.
- Cormier, S.A., S. Lomnicki. W. Backes and B. Dellinger. 2006. Origin and Health Impacts of Emissions of Toxic By-Products and Fine Particles from Combustion and Thermal Treatment of Hazardous Wastes and Materials. *Environmental Health Perspectives* 114: 810-817.
- Environment Canada. 1996. Canadian soil quality guidelines for lead: environmental supporting document - Final draft. December 1996. Environment Canada, Guidelines Division, Ecosystem Science Directorate, Ottawa, ON. Cited in CCME 1999.
- Government of Canada. 2004. *National Ambient Air Quality Objectives (NAAQO).* Available at: http://www.hc-sc.gc.ca/ewh-semt/air/out-ext/reg\_e.html#3.
- Goyer, R. 1991. Toxic effects of metals. In M.O. Amdur, J.D. Doull and C.D. Klaassen (ed.). *Casarett and Doull's Toxicology*. 4th ed. New York: Pergamon Press. 623-680.
- Health Canada (HC). 1992. Guidelines for Canadian Drinking Water Quality Technical Documents: Lead.
- Health Canada (HC). 2004a. Federal Contaminated Sites Risk Assessment in Canada, Part I: Guidance on Human Health Risk Preliminary Quantitative Risk Assessment (PQRA). Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/contamsite/part-partie\_i/index-eng.php Accessed: August 2010.
- Health Canada (HC). 2004b. Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values. Environmental Health Assessment Services, Safe Environments Programme. Available at: http://www.hc-sc.gc.ca/ewhsemt/pubs/contamsite/part-partie\_ii/index-eng.php. Accessed: August 2010.
- Health Canada (HC). 2004c. Canadian Handbook on Health Impact Assessment.



Health Canada (HC). 2005. *National Ambient Air Quality Objectives*. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/air/naaqo-onqaa/index-eng.php. Accessed: August 2010.

Health Canada (HC). 2008. Guidelines for Canadian Drinking Water Quality.

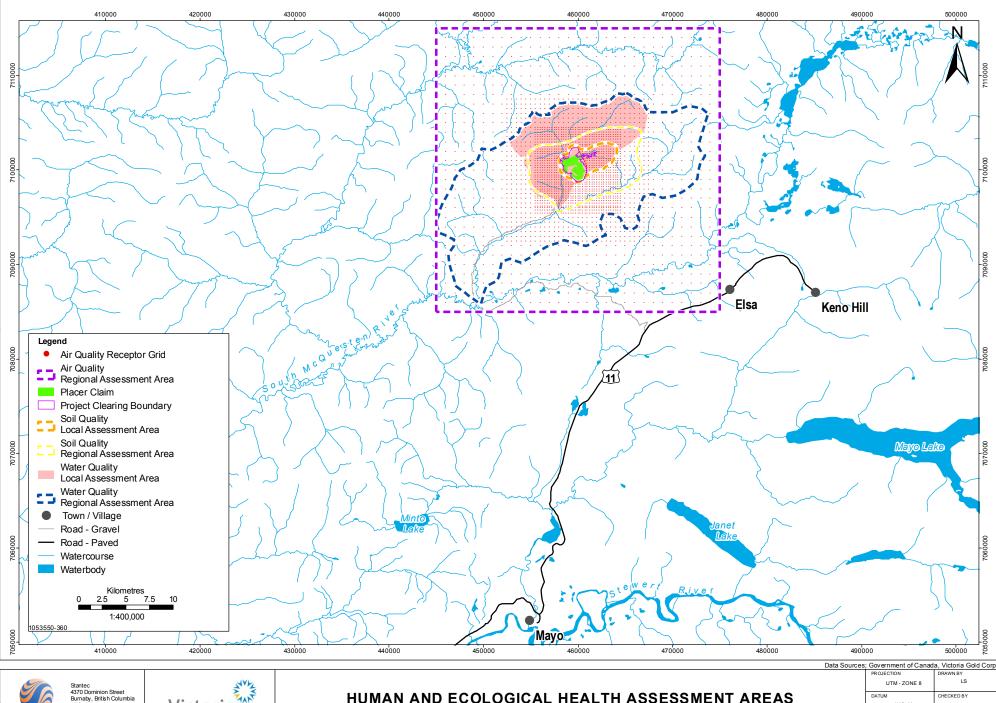
- Institute of Medicine (IOM). 2000. *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Cartenoids*. Food and Nutrition Board of the Institute of Medicine. National Academy Press: Washington, D.C.
- International Agency for Research on Cancer (IARC). 1998. Selenium and Selenium Compounds. Available at: http://www.inchem.org/documents/iarc/vol09/selenium.html. Accessed: August 2010.
- Kruppa, S. V. 1996. The role of atmospheric chemistry in the assessment of crop growth and productivity. In M. Yunus and M. Iqbal (ed.). *Plant Response to Air Pollution*. Chichester, UK: John Wiley & Sons. 35-73.
- Legge, A.H. and S. V. Krupa. 2002. Effects of sulphur dioxide. In J. Bell and M. Treshow (ed.). *Air Pollution and Plant Life*. Chichester, UK: John Wiley & Sons. 135-162.
- Legge, A.H., H. Jager and S.V. Kruppa. 1998. Sulphur Dioxide. In R.B. Flagler (ed.). *Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas*, 2nd ed. Air and Waste Management Association, Pittsburgh, PA.
- Malhotra, S.S. and R.A. Blauel. 1980. *Diagnosis of air pollutant and natural stress symptoms on forest vegetation in western Canada*. North. For. Res. Cent., Can. For. Serv., Environ. Can. Inf. Rep. NOR-X-228. 84 p.
- Mansfield, T.A. 2002. Nitrogen oxides: old problems and new challenges. In J. Bell and M. Treshow (ed.). *Air Pollution and Plant Life*. Chichester, UK: John Wiley & Sons. 120-133.
- National Research Council (NRC). 1989. *Recommended Dietary Allowances*, 10th ed. Washington, DC: National Academy Press.
- Puls, R. 1994. *Mineral Levels in Animal Health: Diagnostic Data*, 2nd Edition. Sherpa International, Clearbrook, BC.
- Rieberger, K. 1992. *Metal concentrations in fish tissue from uncontaminated BC lakes*. Ministry of Environment, Lands and Parks, BC.
- Risk Assessment Information System (RAIS). 1992. *Formal Toxicity Summary for Arsenic*. Available at: http://rais.ornl.gov/tox/profiles/arsenic.html#t3. Accessed: August 2010.
- Silvaro, S.A.N. and T. Rohan. 2007. Trace elements and cancer risk: a review of the epidemiologic evidence. *Cancer Causes & Control.* 18: 7–27.
- Stantec Consulting Ltd. (Stantec). 2010. *First Nation of Na-Cho Nyäk Dun Traditional Knowledge and Use. Final Report.* Prepared for Victoria Gold Corp., Vancouver, BC by Stantec Consulting Limited, Burnaby, BC, and First Nation of Na-Cho Nyäk Dun, YT. June 2010.
- United States Environmental Protection Agency (US EPA). 1986. *Air quality criteria for lead*. Research Triangle Park, NC, US Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and

Assessment Office. EPA 600/8-83-028F. Cited in: *Agency for Toxic Substances and Disease Registry (ATSDR)*. 2007. ToxFAQs for Lead. August 2007.

- United States Environmental Protection Agency (US EPA). 1988. Integrated Risk Information System (IRIS) Database Arsenic. Available at: http://www.epa.gov/iris/. Accessed: August 2010.
- United States Environmental Protection Agency (US EPA), 1991. Integrated Risk Information System (IRIS): Copper. Available at: http://www.epa.gov/iris/subst/0368.htm#carc. Accessed: August 2010.
- United States Environmental Protection Agency (US EPA), 1993. Integrated Risk Information System (IRIS): Selenium and Selenium Compounds. Available at: http://www.epa.gov/iris/subst/0472.htm. Accessed: August 2010.
- United States Environmental Protection Agency (US EPA). 2004. Integrated Risk Information System (IRIS) Database. Lead and compounds (inorganic). Available at: http://www.epa.gov/iris. Acessed: August 2010.
- United States Environmental Protection Agency (US EPA). 2008. *Environmental Residue-Effects Database (ERED)*. Available at: http://www.wes.army.mil/el/ered/. Accessed: August 2010.
- World Health Organization (WHO). 1999. *Carbon monoxide. Environmental Health Criteria 213.* Available at: http://www.inchem.org/documents/ehc/ehc/ehc213.htm#1. Accessed: August 2010.
- World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91. Available at: http://www.euro.who.int/\_\_data/assets/pdf\_file/0005/74732/E71922.pdf. Accessed: August 2010.
- Yukon Government. 2002. O.I.C. 2002/171. Contaminated Sites Regulation, Environment Act. Available at: http://www.gov.yk.ca/legislation/regs/oic2002\_171.pdf.

# 10 FIGURES

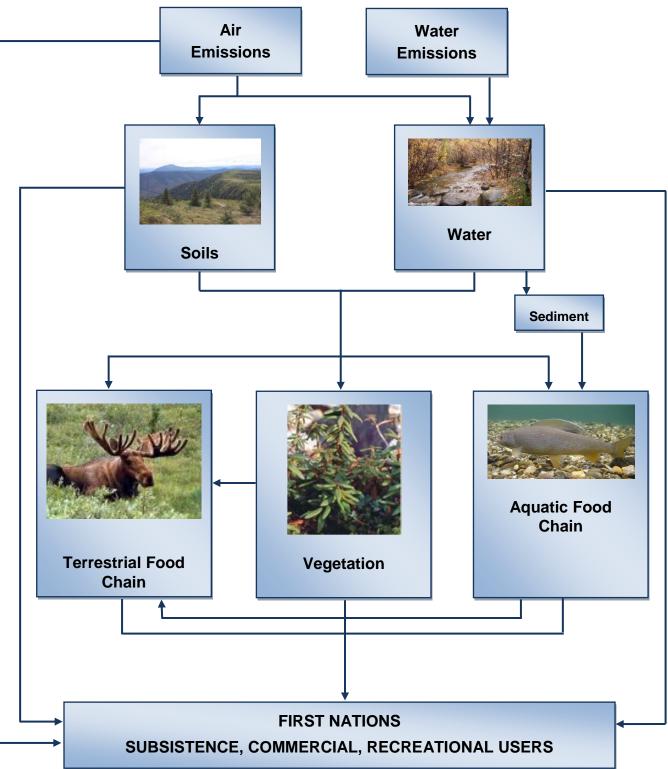
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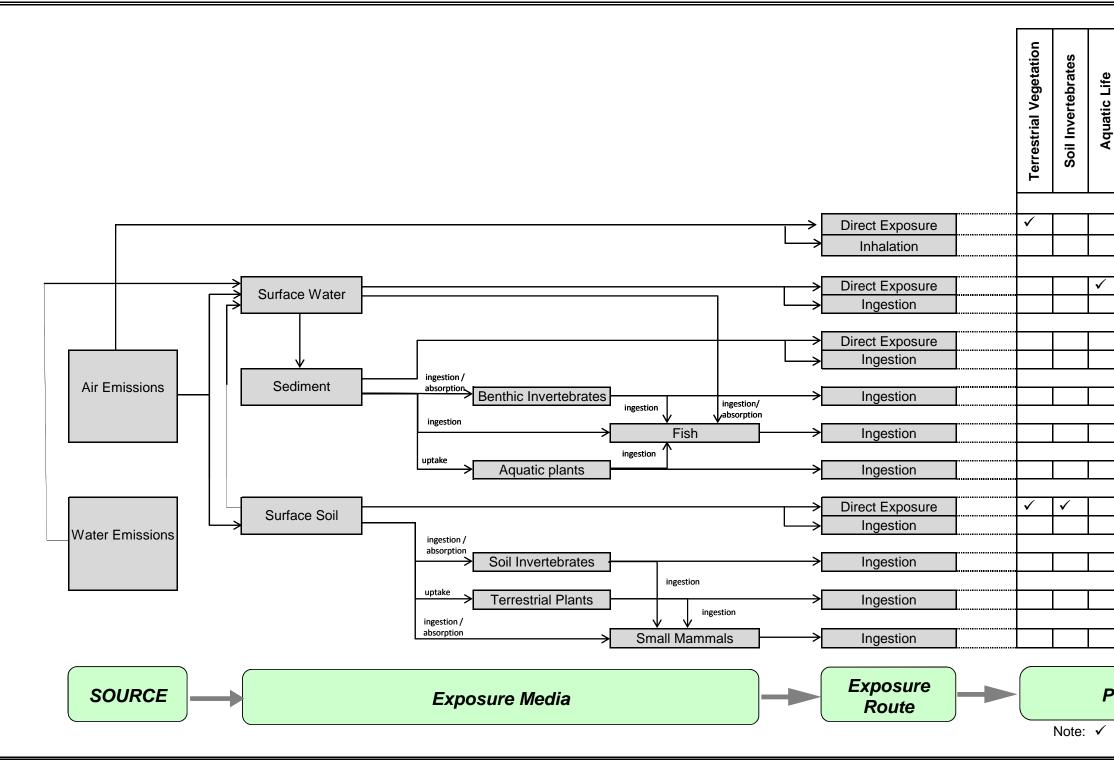


Figure 4.3-2: Exposure Pathway Model for Ecological Receptors

Aquatic Life	Sediment Receptors	Snowshoe Hare	Moose	Black Bear	Mallard	Red-Tailed Hawk	Grouse		
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