



**PRESTON SAND & GRAVEL  
TOWNSHIP OF NORTH DUMFRIES**

**SCREENING LEVEL HUMAN HEALTH RISK  
ASSESSMENT (SLHHRA) OF AIR QUALITY  
IMPACTS OF THE PROPOSED HENNING PIT**

**Prepared For: Preston Sand and Gravel Company Limited**

December 13<sup>th</sup>, 2013

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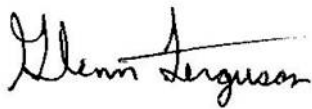
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**SCREENING LEVEL HUMAN HEALTH RISK ASSESSMENT OF  
AIR QUALITY IMPACTS OF THE PROPOSED HENNING PIT**

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## GLOSSARY OF TERMS

This glossary is intended to provide the reader with working definitions of many of the technical terms and acronyms which appear in the risk assessment. Various government agencies and organizations may have different definitions for these terms; thus the definitions are intended only to provide guidance to the reader as to how these terms are used within this report. Sources for these definitions include a variety of documents prepared by the Ontario Ministry of the Environment (MOE), United States Environmental Protection Agency (US EPA), New York State Department of Health, Health Canada, U.S. Federal Drug Administration (USFDA), International Programme on Chemical Safety (IPCS), Agency for Toxic Substances and Disease Registry (ATSDR), and the World Health Organization (WHO).

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### **90<sup>th</sup> Percentile**

The 90<sup>th</sup> percentile of a set of measurements is the value below which 90% of the results fall.

### **AAQC**

See *Ambient Air Quality Criteria*.

### **Acute**

Occurring over a short time. An acute or short-term exposure can result in short term or long-term health effects. An acute effect happens within a short time after an exposure (*i.e.*, may be minutes or days).

### **Agency for Toxic Substances and Disease Registry (ATSDR)**

As an agency of the U.S. Department of Health and Human Services, the mandate of ATSDR is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and disease related to toxic substances. Further information can be found on <http://www.atsdr.cdc.gov/>.

### **Ambient**

Environmental or surrounding conditions. Ambient air is usually outdoor air (as opposed to indoor air).

### **Ambient Air Quality Criteria (AAQC)**

Air quality criteria established by the MOE for specific chemicals and substances above which there is the risk of potential for adverse effects. These effects can be health-based, or protective of other important endpoints (*e.g.*, corrosion, odour, staining, *etc.*). In Ontario, these criteria are usually developed for annual averages or on a 24-hour basis in  $\mu\text{g}/\text{m}^3$ , though criteria are also available for 1-hour and 10-minute exposure durations for select chemicals.

### **Asthmatics / asthma**

Asthmatics are people who suffer from asthma, a disease involving episodes (“asthma attacks”) where the breathing tubes become constricted making it difficult to breath.

### **ATSDR**

See *Agency for Toxic Substances and Diseases Registry*.

**Background level**

A typical or average level or concentration of a chemical or substance in the environment, without any contribution from the proposed project.

**Benchmark**

A reference point (such a regulatory standard or guideline) against which a measurement can be compared (as a noun), or the act of comparing a measurement to a reference value (as a verb).

**Canadian Council of Ministers of the Environment (CCME)**

A group comprised of 14 environment ministers from the federal, provincial and territorial governments which promotes effective intergovernmental cooperation and coordinated approaches to inter-jurisdictional issues such as air pollution and toxic chemicals. Although the CCME establishes nationally-consistent environmental standards, strategies and objectives, it has no authority to enforce them on individual jurisdictions.

**Carcinogen / Carcinogenic**

A substance or chemical that can cause cancer. Knowledge that a chemical or substances can cause cancer is usually obtained from laboratory studies in animals. Only infrequently do we know that a substance definitely causes cancer in humans. Sometimes the cancer effect is dependent on the type of exposure.

**CCME**

See *Canadian Council of Ministers of the Environment*.

**Chemical of Concern (COC)**

A chemical of concern is a contaminant identified as of interest for evaluation as part of the risk assessment.

**Chronic**

Occurring over a long period of time, several weeks, months or years, depending on the exposed species.

**COC**

See *Chemical of concern*.

**Concentration**

The proportion of one substance contained in a given amount of a specific media. The unit is a concentration unit which has two components: the numerator (*i.e.*, quantity of chemical present) and the denominator (*i.e.*, quantity of the media or volume of solution in which the chemical is present). For example, a NO<sub>2</sub> concentration of 10 µg/m<sup>3</sup> represents 10 µg of NO<sub>2</sub> present within 1 cubic metre of air.

**Concentration Ratio (CR)**

The concentration is similar to the *hazard quotient*, where the exposure and exposure limit are expressed as concentrations (rather than as doses). See *Hazard quotient*.

**Contaminant**

A substance that is present in an environment where it does not belong.

**CR**

See *Concentration ratio*.

***Cumulative ground-level air concentrations***

Predicted total ground-level air concentrations, based upon adding the modelled contribution from the proposed Project to the measured regional background air concentrations, including other nearby operations.

***Data gap***

Refers to a type of data which is unavailable or limited, and which would likely reduce uncertainty in the risk assessment if it were to be available or if the data set were more complete.

***Dermal***

Referring to the skin. For example, dermal absorption means absorption through the skin.

***Dose***

The amount of chemical or substance taken in or absorbed by an exposed individual. Dose often takes body weight into account. For example, to receive equivalent doses of medicine, children are given smaller amounts than adults. The unit is mg/kg for example. The dose rate is the frequency that the dose is applied, such as "mg/kg body weight per day". Acute toxicity usually refers to single doses, while chronic toxicity refers to given dose rates.

***Dose-response relationship***

The relationship between the amount of a substance absorbed (*i.e.*, dose) and the resulting changes in body function or health (*i.e.*, response).

***Effect***

Change in the state or dynamics of an organism, system, or (sub)population caused by the exposure to an agent.

***Endpoint***

An adverse effect on a living system (from single organisms to entire ecosystems) which is studied in an experiment, or an adverse effect whose prevention or minimization is the basis of a benchmark. See also *Effect* and *Benchmark*.

***Emissions***

Materials released to the environment from a source. Emissions may be released from localized sources (such as an industrial smokestack), diffuse sources (such as a landfill site) or mobile sources (such as an automobile or locomotive).

***Exceedance***

Refers to predicted ground-level air concentrations which are greater than, or exceed, the corresponding regulatory benchmark for that averaging period.

***Exposure***

Exposure is any contact with a chemical by swallowing, breathing or direct contact (such as through the skin or eyes). Exposure may be either short term (acute) or long term (chronic). Exposure can vary greatly, and is often associated with specific activities or behaviours of people or ecological organisms. It is quantified as the amount of a substance that can be absorbed, or the amount available for inhalation or ingestion.



**Exposure assessment**

A process that estimates or measures the amount of a chemical or substance that enters or comes into contact with a person or ecological organism. An exposure assessment also takes into consideration the length of time and the nature of a population exposed to a chemical.

**Exposure pathway**

The pathway a chemical, substance or agent may take to reach or cause exposure of humans or other living organisms. Pathways link a source of a chemical, substance or agent (*i.e.*, soil) to its eventual entry into the body.

**Exposure route**

The route through which a substance can enter the body.

**Exposure scenario**

A combination of facts, assumptions, and inferences that define a discrete situation where potential exposures may occur. These may include the source, the exposed population, the time frame of exposure, microenvironment(s), and activities. Scenarios are often created to aid exposure assessors in estimating exposure under varying conditions.

**Guideline**

Recommended limit for some parameter or substance in a specific medium and/or environment. For example, health guidelines are upper limits of exposure, below which adverse health effects are absent or minimized.

**Hazard**

Inherent property of an agent or situation having the potential to cause adverse effects when an organism, system, or (sub)population is exposed to that agent or situation.

**Hazard assessment**

The hazard assessment involved identifying and understanding potential health outcomes that can result from exposure to each COC and the conditions under which the outcomes might be observed. The hazard, or toxicity, assessment methodology is based on the fundamental dose response principle. That is, the response of biological systems to chemical exposures increases in proportion to the concentration of a chemical in critical target tissues where adverse health outcomes may occur.

**Health Canada (HC)**

The Canadian Federal department responsible for helping Canadians maintain and improve their health.

**Health assessment**

A process to determine the health impacts related to particular events or circumstances, such as the release of a chemical, substance or agent into the environment. It includes a health interpretation of all the information known about the situation. The information may include some or all of the following: site investigation (environmental sampling and studies), exposure assessment, risk assessment, biological monitoring and health effects studies. The information is used to advise people how to prevent or reduce their exposures, to determine if remedial actions are necessary, or the need for additional studies. The types of studies carried out in a health assessment can include studies of the environment (soil measurements, chemical availability, *etc.*) or studies of the people living in the environment (epidemiological studies or biological monitoring studies).

**HHRA**

See *Human health risk assessment*.

**Human health risk assessment (HHRA)**

A risk assessment focused on estimating potential human health risks to a defined set of individuals from exposure to a particular agent or agents. The HHRA process includes four basic steps: problem formulation (hazard identification), exposure assessment, hazard assessment, and risk characterization.

**Ingestion**

Taking a substance into the body by swallowing it, whether incidentally or purposely.

**Inhalation**

Breathing or inhaling air, and the substances it contains, into the lungs.

**Interstitialization**

The process by which ultrafine particles are penetrate through the pulmonary alveoli, and are sequestered in the interstitial space within the lung tissue.

**Mode of Action**

The mode of action of a substance is defined as the general recognition of the broad biochemical pathways (such as DNA synthesis, protein synthesis, cholesterol synthesis) which are inhibited or affected by a substance. The mode of action is distinguished from the mechanism of action of a substance, which is defined as the mechanism by which a toxicologically active substance produces an effect on a living organism or in a biochemical system. The mechanism of action is usually considered to include an identification of the specific targets to which a toxicologically active substance binds or whose biochemical action it influences.

**Modelling**

The process by which scientists consider many scenarios of exposure for the purpose of determining the associated health risks. A selected scenario may be preferred for a given site when information is known about the site and about the behaviour of the chemical or substance. In most cases modelling involves the use of mathematical equations to inter-relate the factors critical to the process being studied. These mathematical equations have been developed through studies of factor inter-relationships. Models are used to predict events expected in the future, or that have occurred in the past, when direct measurements are not feasible. Models can be used to assist in designing studies to obtain direct measurements of the processes of concern.

**MOE**

See *Ontario Ministry of the Environment*.

**NAAQOs**

See *National Ambient Air Quality Objectives*.

**NAPS**

See *National Air Pollution Surveillance network*.

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**National Air Pollution Surveillance (NAPS) network**

The NAPS network, a joint initiative of Environment Canada and various provinces (including Ontario), territories and some municipal governments, to monitor ambient concentrations of key COC (primarily SO<sub>2</sub>, NO<sub>2</sub>, ozone, fine particulate matter, and carbon monoxide) at key monitoring stations across Canada. These measurements are used by the provinces to report the Air Quality Index and by Environment Canada to report the Air Quality Health Index (AQHI).

**National Ambient Air Quality Objectives (NAAQOs)**

NAAQOs are the benchmark against which Canada assess the impact of anthropogenic activities on air quality and ensures that current emission control policies are successfully protecting human health and the environment. The federal government sets NAAQOs on the basis of recommendations from the Federal-Provincial Working Group on Air Quality Objectives and Guidelines consisting of representatives from both Health Canada and Environment Canada, as well as their relevant provincial counterparts.

**NOAEL**

See *No observed adverse effect level*.

**No observed adverse effect level (NOAEL)**

The highest dose in an experiment which did not cause an adverse effect.

**NO<sub>x</sub>**

See *Oxides of Nitrogen*

**Ontario Ministry of the Environment (and Energy) (MOE / MOEE)**

Provincial body responsible for development, implementation, and enforcement of regulations, as well as various programs and initiatives, which address environmental issues having local, regional and/or global effects.

**Oral**

By mouth. Oral exposure refers to exposure by swallowing a material. Also see *Ingestion*.

**Oxides of Nitrogen (NO<sub>x</sub>)**

Oxides of nitrogen (NO<sub>x</sub>), including nitric oxide (NO) and nitrogen dioxide (NO<sub>2</sub>) gases, are produced by both natural and human activities, primarily formed by the reaction of atmospheric oxygen and nitrogen during high temperature combustion processes.

**PAH**

PAH is an acronym for *polycyclic aromatic hydrocarbons*. All PAH contain only carbon and hydrogen, and are produced as by-products of incomplete combustion. They are commonly found in soot, and are emitted during the combustion of fossil fuels and wood.

**Particulate matter (PM)**

A general term that refers to dust, soot, and smoke that is emitted from such sources as factories, vehicles, and fires. A numeric subscript indicates the upper limit of the particles of interest (*i.e.*, PM<sub>10</sub> refers to particulate matter less than 10 microns in aerodynamic diameter).

**Parts per billion (ppb)**

Units of concentration (*i.e.*, µg/kg, ng/g, *etc.*)

**Parts per million (ppm)**

Units of concentration (*i.e.*, µg/g, mg/kg, *etc.*)

**Physico-chemical properties**

The physical and chemical characteristics of a substance. Examples of physico-chemical properties include boiling point, melting point, colour, odour, solubility, vapour pressure, *etc.*

**PM**

See *Particulate matter*.

**PM<sub>10</sub>**

Particulate matter which is less than 10 µm in diameter. This size of particulate is small enough so as to be easily inhaled into the lungs. This is the primary particulate size fraction evaluated for potential health impacts by the HHRA.

**PM<sub>2.5</sub>**

Particulate matter which is less than 2.5 µm in diameter. This size of particulate is small enough so as to be easily inhaled deep into the lower lungs (*i.e.*, alveolar), and potentially absorbed directly into the blood stream.

**ppb**

See *Parts per billion*.

**ppm**

See *Parts per million*.

**Problem Formulation**

Initial stage of the risk assessment, where information is gathered and interpreted to plan and focus the risk assessment.

**RA**

See *Risk assessment*.

**Receptor**

An individual (person, plant, animal) that could come into contact with hazardous substances. In the context of air dispersion modelling, a sensitive receptor can also refer to a specific geographical location at which ground-level air concentrations are predicted.

**Reference concentration (RfC)**

An estimated air concentration of a specific chemical or substance which is likely to be without risk of deleterious effects to people, animals or plants, even if the exposure continues over a lifetime. Typically expressed in mg/m<sup>3</sup> or µg/m<sup>3</sup>.

**Reference dose (RfD)**

An estimate of a rate of exposure of people, animals or plants that is likely to be without risk of deleterious effects, even if the exposure continues over a lifetime. Reference doses are adjusted for sensitive sub-groups of the population. Typically expressed in mg/kg bodyweight/day or µg/kg bodyweight/day.

**Respiratory Tract**

The respiratory tract consists of the nose, mouth, windpipe, lungs and related structured required for breathing.

**RfC**

See *Reference concentration*.

**RfD**

See *Reference dose*.

**Risk**

Risk, in the context of a human health risk assessment, is the likelihood of injury, disease or death that will be caused by an action or condition.

**Risk Assessment (RA)**

A process that estimates the likelihood or chance that people or the environment may experience adverse effects from a particular series of events or circumstances, such as exposure to chemicals, substances or agents. The four steps of a risk assessment are:

- problem formulation (also known as hazard identification);
- toxicity/effects assessment;
- exposure assessment; and,
- risk characterization.

Note: Likelihood is a quantitative term related to "probability", "chance" or to "risk".

**Risk characterization**

Final phase of the risk assessment, where the exposure and effects/toxicity information are combined to evaluate potential impacts, and provide a qualitative or quantitative characterization of health risk.

**Risk management**

The process of deciding how to reduce or eliminate possible adverse effects on people's health and the environment by considering the risk assessment, engineering factors (*i.e.*, can engineering procedures or equipment do the job, for how long and how well?) and social, economic and political concerns.

**Route of exposure**

The way in which a person or other organism in the natural environment may come in contact with a chemical, substance or agent. These are typically through inhalation, ingestion, or *via* dermal absorption. For example, drinking (ingestion) and bathing (skin contact) are two different routes of exposure to chemicals that may be found in water. See "Exposure."

**Safe**

In common language, safe means free from harm or risk. In scientific language, any exposure to most chemicals, substances or agents have some risk, although that risk may be extremely small. Therefore, scientifically, safe means at very low or negligible risk.

**Scenario**

A hypothetical situation evaluated in a HHRA.

**Screening Level Human Health Risk Assessment (SLHHRA)**

A screening level human health risk assessment (SLHHRA) is a qualitative or quantitative evaluation of risk typically based on a "worst-case" exposure scenario rather than verifiable site-specific conditions. As an initial scoping of potential risk, the SLHHRA approach relies on available data to provide conservative estimates of exposure and risk based on a worst-case scenario, so that exposures and risks are not underestimated. The intent is to determine whether there is the potential for adverse health impacts under these worst-case exposure scenarios for the relevant COC emitted from the proposed Project, identify any data gaps limiting the assessments ability to appropriately estimate exposures and risk, and eliminate any

COC and pathways of exposure which are not a concern moving forward for any further analyses.

**SDB**

Standards Development Branch. The branch of the MOE largely responsible for technical review of environmental risk assessments in Ontario.

**SLHHRA**

See *Screening Level Human Health Risk Assessment*.

**Threshold**

The dose or exposure below which an adverse effect is not expected.

**Total Suspended Particulates (TSP)**

A measure of the total number of particles of solid or liquid matter - such as soot, dust, aerosols, fumes and mist - found in a sample of ambient air. Typically assumed to be composed of suspended particulate that have aerodynamic diameters less than 40 µm.

**Toxicity**

A general term that can refer either to a substance's toxic potency, or the type(s) of effects that a substance can have (for example, ocular toxicity refers to effects on the eye; respiratory system toxicity refers to effects on the respiratory system).

**Toxicity assessment**

Step in the risk assessment process involving the evaluation of the toxicological properties and effects of a chemical, with special emphasis on establishment of dose response characteristics.

**Toxicity Reference Value (TRV)**

A toxicity reference value, or TRV, is an estimate of the dose (in this document, usually a daily dose over a long period of time) of a substance that is associated with a specific level of risk, or that is considered to be safe. Toxicity reference values are used to evaluate whether estimated or measured exposures are likely to cause adverse health effects. Toxicity reference values are also used to develop guidelines and standards, such as drinking water quality guidelines.

**TRV**

See *Toxicity reference value*.

**TSP**

See *Total suspended particulates*.

**UF**

See *Uncertainty factor*.

**µg**

Microgram (1 x 10<sup>-6</sup> grams).

**UFP**

See *Ultrafine Particulate Matter*.

**Ultrafine Particulate Matter (UPM)**

UFP constitute particulate matter smaller than 0.1 microns (or 100 nanometres) in size (*i.e.*, PM<sub>0.1</sub>). Due to their small size, UFPs are considered to be respirable particles and are able to

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travel deep within the lung with the potential to penetrate tissue and undergo interstitialization, and therefore are not easily removed from the body.

***Uncertainty analysis***

A detailed examination of the potential sources of variability and uncertainty within the data, and their influence on risk assessment results. See *Uncertainty Factor*.

***Uncertainty Factor (UF)***

One of several factors used in calculating the reference dose from experimental data. UFs are typically used to account for such uncertainties as: (1) the variation in sensitivity among humans (*i.e.*, intraspecies); (2) the uncertainty in extrapolating animal data to humans (*i.e.*, interspecies); (3) the uncertainty in extrapolating data obtained in a study that covers less than the full life of the exposed animal or human; (4) the uncertainty in using LOAEL data rather than NOAEL data (see LOAEL and NOAEL); and, (5) uncertainties associated with the adequacy of the database of experimental data.

***United States Environmental Protection Agency (US EPA)***

The federal agency responsible for developing and enforcing regulations to implement environmental laws enacted by Congress. EPA is responsible for researching and setting national standards for a variety of environmental programs, and delegates to states and tribes the responsibility for issuing permits and for monitoring and enforcing compliance.

***US EPA***

See *United States Environmental Protection Agency*.

***WHO***

See *World Health Organization*.

***World Health Organization (WHO)***

The United Nations agency which works in a variety of ways and with a variety of agencies internationally to attain the highest level of physical, mental, and social well-being for all people.

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## SCREENING LEVEL HUMAN HEALTH RISK ASSESSMENT OF AIR QUALITY IMPACTS OF THE PROPOSED HENNING PIT

### EXECUTIVE SUMMARY

#### Overview of the Study

Preston Sand & Gravel Company Limited (Preston) is proposing to develop an aggregate pit (Henning Pit) to be located in the Township of North Dumfries, Regional Municipality of Waterloo, Ontario. Preston is seeking a Class A licence (Category 3) under the Aggregate Resources Act, and a zoning by-law amendment under the Planning Act. Local residents (*i.e.*, Gerry Brown *et al.*) have filed an appeal, and the application has been referred to the Ontario Municipal Board (OMB) for consideration. The appellant cited issues with respect to whether dust and air emissions have been appropriately assessed so as to protect the health of nearby residents. RWDI was retained by Preston to undertake an assessment in support of the applications, to evaluate potential dust emissions and air quality associated with the proposed pit.

As part of the air quality assessment, Intrinsic Environmental Sciences Inc. (Intrinsic) was retained by Preston to assess the potential human health implications associated with modelled air emissions provided by RWDI in the local area of the proposed pit relating to the future facility emissions, as well as increased road traffic arising from the development. In addition, based on information provided by RWDI, Intrinsic evaluated the cumulative modelled air emissions in the area from both the proposed pit and existing background emissions.

To evaluate the potential impacts of increased dust emissions arising from the proposed development, a screening level human health risk assessment (SLHHRA) was completed. The SLHHRA can be used to determine the worst-case health implications of emissions to potentially sensitive individuals living, working, or playing in the surrounding community.

Overall, this project is being completed in addition to the requirements of the Local Air Quality regulation (Ontario Regulation 419/05), which does not consider cumulative, or background air quality, with respect to emissions from a subject facility. This regulation does not provide specific guidance on human health risk assessment methodology, nor specifically those required to complete a SLHHRA. The current human health risk assessment was conducted according to widely accepted risk assessment methodologies and guidance published and endorsed by regulatory agencies including the Ontario Ministry of the Environment (MOE), Health Canada, the Canadian Council of the Ministers of the Environment (CCME), and the United States Environmental Protection Agency (US EPA). The assessment is also limited to the operations evaluated as part of the RWDI Air Quality Assessment (RWDI, 2013).

#### **What chemicals were assessed?**

Based upon the primary components present in aggregate dust and diesel exhaust from the vehicles involved with the operations, and the contaminants addressed in the Air Quality Assessment Report, the following contaminants were selected as COC for the current screening level assessment: i) crystalline silica; ii) respirable particulate matter (PM<sub>10</sub>); iii) fine particulate matter (PM<sub>2.5</sub>); iv) oxides of nitrogen (NO<sub>x</sub>); and, v) benzo[a]pyrene (surrogate for the PAH group).



**What locations around the proposed Project were evaluated?**

To assess potential health risks related to the projected Project emissions, impacts to local air quality were evaluated at discrete locations in the surrounding community. As such, ground-level air concentrations were predicted at seven (7) off-site locations spaced around the footprint of the proposed Project: CRAND4, CRAND5, CRAND6, CRAND15, CHURCH12, RENTAL3, and PAULCAB14. These locations (see table below) are primary residences in the surrounding area, and one community church (*i.e.*, CHURCH12 which is the Cedar Creek Community Church). The geographical locations of each of these sensitive receptor locations are noted on Figures 3-1.

<b>Location of Sensitive Receptors selected for Assessment</b>			
<b>Receptor Name</b>	<b>Address</b>	<b>UTM X (m)</b>	<b>UTM Y (m)</b>
CRAND4	1986 Cedar Creek Road, RR4 Cambridge	548506.79	4798914.82
CRAND5	1970 North Dumphries Road, RR4, Cambridge	548628.26	4798736.40
CRAND6	1697 Cedar Creek Road, RR4, Cambridge	549222.23	4798777.88
CRAND15	1809 Dumphries Road, RR4, Cambridge	548511.36	4797503.76
CHURCH2	Cedar Creek Church	548029.94	4798642.77
RENTAL3	Rental Farm House	548253.49	4798921.47
PAULCAB14	N/A	548525.63	4797551.29

It should be noted that since ambient concentrations decline with distance from the source, it would be expected that potential receptor locations further away from the proposed Project would observe even lower impacts than those of the selected locations.

**What operating scenarios were considered in the assessment?**

To properly evaluate potential exposures to residents living and working in the area directly surrounding the proposed Project, the following exposure scenarios were assessed:

- Central Processing Plant in Phase 1 Area
  - Project Only (Henning Pit)
  - Cumulative Exposure (*i.e.*, Project plus existing background conditions)
- Auxiliary Processing Plant in Active Phase
  - Project Only (Henning Pit)
  - Cumulative Exposure (*i.e.*, Project plus existing background conditions)

The primary exposure pathway under evaluation in the current assessment was the inhalation of the COC by individuals living, working or playing in the residencies surrounding the proposed Project. Deposition onto soils was not a significant risk for the COCs selected for the current assessment, and as such, oral and dermal exposures were not evaluated.

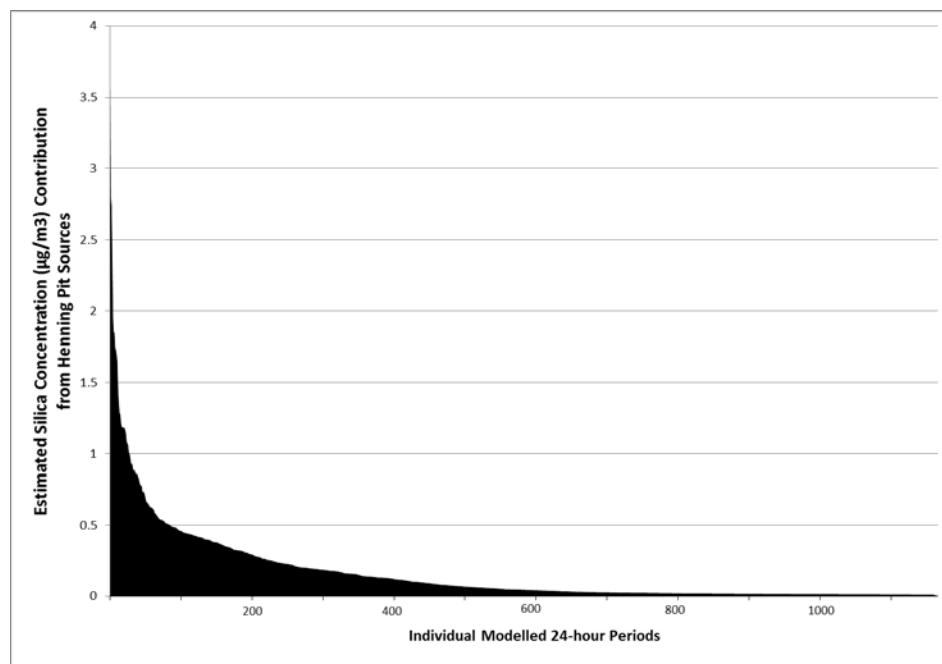
**What were the results of the assessment?**

Based on the results of the assessment, worst-case contributions from the proposed Henning Pit facility do not exceed the relevant regulatory benchmarks in any scenario, with the exception of minor 1-hour NO<sub>x</sub> exceedances in the Central Processing Plant scenario at most of the receptor locations.

However, it should be noted that the current assessment conservatively assumes that 100% emitted oxides of nitrogen are inter-converted to NO<sub>2</sub> (the most toxic of the oxides of nitrogen).

This assumption greatly over-estimates predicted concentrations of NO<sub>2</sub> as a result of emissions from diesel engines. Under typical conditions in the presence of ozone, only approximately 30% of NO (and other lesser oxides of nitrogen) are inter-converted to NO<sub>2</sub> (see discussion in Section 3.2.3.3). Even if one assumed 50% of the oxides of nitrogen are converted to NO<sub>2</sub>, ambient concentrations at the receptor locations arising from Project emissions would not exceed the relevant benchmark.

It is also important to remember that the assessed concentrations represent the worst-case maximum concentration predicted by the Air Quality assessment. This worst-case concentration prediction is a result of the aligning of worst-case operations, meteorological, and situational conditions. For example, the maximum predicted concentration of 24-hour silica exposures at the worst-case sensitive receptor location (CRAND5) arising from Project emissions was 3.8 µg/m<sup>3</sup>. However, if one reviews the predicted daily concentrations of silica at the CRAND5 sensitive receptor location over the five years modelled by the Air Quality assessment arising from the proposed Project (see Figure ES-1 below), only 2.3% of the days have 24-hour silica concentrations that exceed even 1 µg/m<sup>3</sup> and less than 0.35% of the days exceed the 2 µg/m<sup>3</sup> threshold for silica. Given the 24-hour health-based AAQC for silica is 5 µg/m<sup>3</sup>, contributions to ambient concentrations of silica from the proposed Project at the sensitive receptor locations are well below levels which would be of concern to human health (*i.e.*, silicosis).



**Figure ES-1 Estimated 24-Hour Silica Concentrations at CRAND5 from Henning Pit Sources over Five Years**

The same pattern is observed for all of the other COCs evaluated in the current assessment. For 24-hour PM<sub>2.5</sub> at the worst-case sensitive receptor location, the maximum predicted concentration arising from Project sources is 3.4 µg/m<sup>3</sup>. However, if one reviews the predicted daily concentrations of PM<sub>2.5</sub> at this worst-case sensitive receptor location over the five years modelled, only 1.5% of the days have 24-hour PM<sub>2.5</sub> concentrations that exceed even 1 µg/m<sup>3</sup> and less than 0.35% of the days exceed the 2 µg/m<sup>3</sup> threshold. Again, with a regulatory benchmark of 25 µg/m<sup>3</sup>, the contributions of fine particulate from the proposed Project are typically orders of magnitude below levels that could potential cause health concerns in the surrounding population.

Given the hourly and daily variability in COC concentrations, and the multitude of conservative assumptions used within the Air Quality Assessment, one would not expect that the predicted emissions from the proposed Project would result in any elevated unacceptable health concerns.

The Air Quality Assessment also predicted cumulative concentrations for each of the COCs assuming the contributions for the proposed Project are added to already existing contributions from the other existing pit facilities, as well as regional background concentrations. The cumulative assessment did indicate that the worst-case exposure conditions could result in ambient concentrations that exceeded the respective regulatory benchmark for some of the COCs. However, in each of these cases, the contribution from the proposed Project was minimal to negligible compared to already existing background sources.

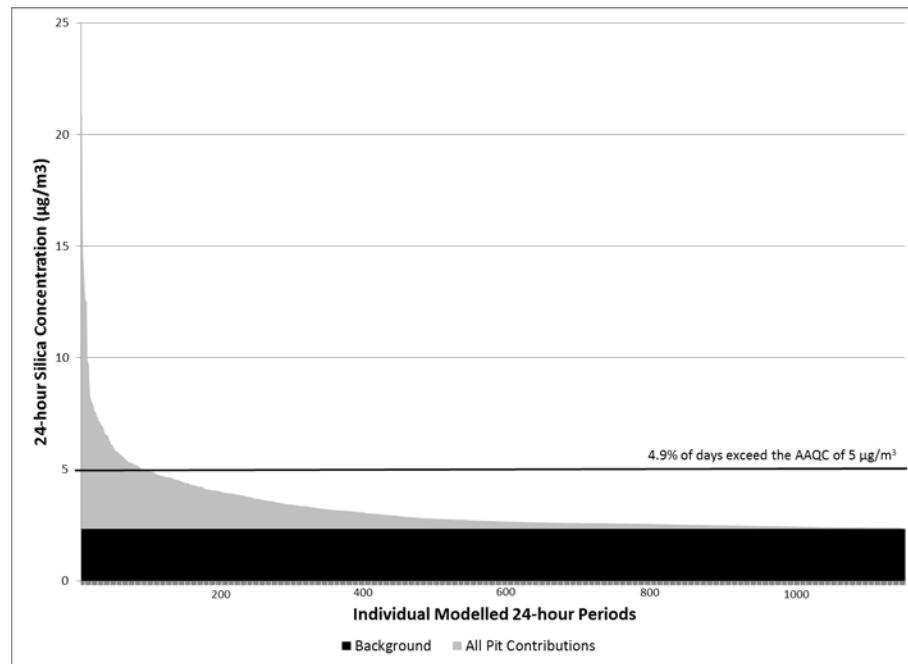
Furthermore, like the pattern observed for emissions from the proposed Project, cumulative concentrations also varied greatly on an hourly and daily basis, depending on a variety of operational, meteorological, and situational conditions.

Using 24-hour silica concentrations as an example again, the maximum predicted **cumulative** concentration of 24-hour silica exposures at the worst-case sensitive receptor location for cumulative exposures (PAULCAB4) was  $21.2 \mu\text{g}/\text{m}^3$ . Interestingly, the contribution of silica exposure from the proposed Project on that particular modelled day was  $0.079 \mu\text{g}/\text{m}^3$ , with the other existing pits (*i.e.*, St. Mary's and Lafarge pits) and related road traffic providing nearly all of the estimated silica concentrations on this worst-case day. This discrepancy is due to the location of the PAULCAB4 receptor in relation to both the proposed Project site and the adjacent pit operations, specifically the Lafarge facility.

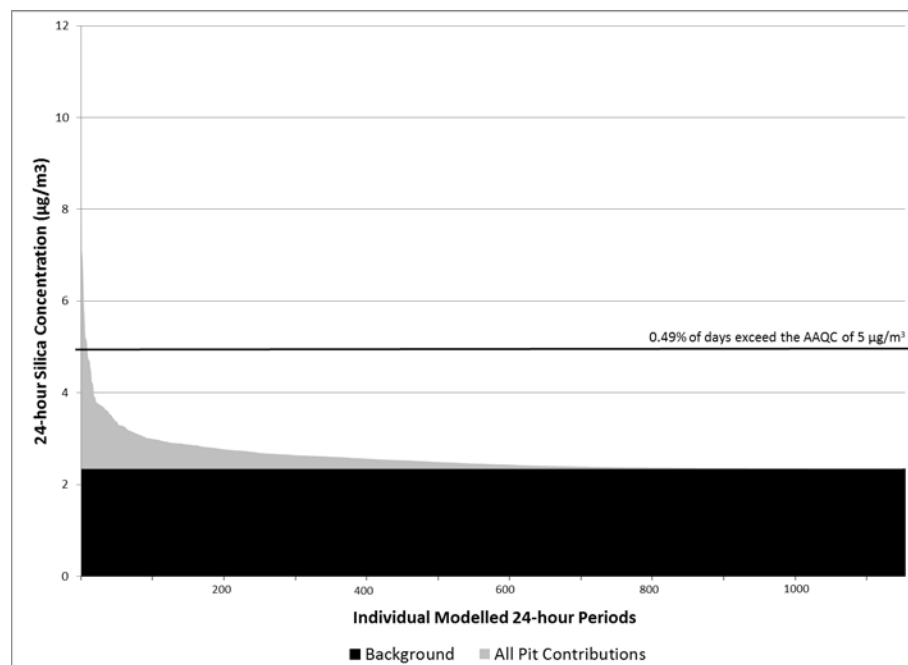
If one reviews the predicted daily **cumulative** concentrations of silica at the PAULCAB4 sensitive receptor location over the five years modelled by the Air Quality assessment (see Figure ES-2), one again sees a similar pattern as observed in the Project only emission scenarios. In this case, less than 5% of the days have 24-hour silica concentrations from cumulative sources that exceed the  $5 \mu\text{g}/\text{m}^3$  regulatory benchmark.

A similar review of the predicted daily **cumulative** concentrations of silica at the CRAND5 receptor (*i.e.*, the worst-case location for emissions from the proposed Project) shows that the maximum cumulative exposure was  $9.7 \mu\text{g}/\text{m}^3$  of which only the  $3.8 \mu\text{g}/\text{m}^3$  of silica noted previously was related to emissions from the proposed Project. Figure ES-3 shows the rapid decline in ambient daily silica concentrations around CRAND5, with less than 0.5% of the days have 24-hour silica concentrations from cumulative sources that exceeded the  $5 \mu\text{g}/\text{m}^3$  regulatory benchmark.

Chronic silicosis usually develops after 10 or more years of prolonged and consistent inhalation exposure to crystalline silica. The 24-hour regulatory benchmark of  $5 \mu\text{g}/\text{m}^3$  is intended to provide a threshold with a built-in margin of safety to prevent the potential for developing chronic silicosis over an extended period of continuous exposure. Given exceedances of the  $5 \mu\text{g}/\text{m}^3$  threshold are predicted to be a very infrequent event even under worst-case conditions, and the degree of conservatism built into the assumptions used to predict silica concentrations in the Air Quality Assessment, it is not expected that current emissions would result in the potential to develop silicosis in individuals living around the proposed Project site.



**Figure ES-2 Estimated 24-Hour Silica Concentrations at PAULCAB4 from Cumulative Sources over Five Years**



**Figure ES-3 Estimated 24-Hour Silica Concentrations at CRAND5 from Cumulative Sources over Five Years**

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This conclusion is further confirmed when one compares predicted annual exposures of silica at each of the sensitive receptor locations with the chronic regulatory benchmark for silica. In this case, none of the predicted Project Only or cumulative concentrations of silica over a long-term basis exceeds the health-based regulatory benchmark protective of silicosis, at any of the sensitive receptor locations. As such, though occasional short-term elevated concentrations of silica could be observed around the proposed Project (largely due to existing background and extraction activities), ambient concentrations over the longer term period are unlikely to reach levels that would result in potential health concerns related to silica exposures.

***What were the conclusions of the overall assessment?***

Based on the results of the assessment, and given the considerable conservatism built into the Air Quality Assessment, no unacceptable health risks related to emissions from the proposed Project would be expected. In fact, estimated emissions from the proposed Project typically represent a minimal to negligible component of the overall cumulative exposures for each of the COCs predicted for the sensitive receptor locations around the proposed Project site. Furthermore, results of the assessment also indicate that cumulative exposures to the evaluated chemicals are not expected to result in unacceptable health risks in the surrounding community given the degree of conservatism built into the assessment assumptions and scenarios.

However, to ensure ambient dust concentrations are kept at a minimal level, it is recommended that the dust suppression and related mitigation measures outlined in the Air Quality Assessment report be considered and implemented for the proposed Project where feasible.

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## SCREENING LEVEL HUMAN HEALTH RISK ASSESSMENT OF AIR QUALITY IMPACTS OF THE PROPOSED HENNING PIT

### 1.0 INTRODUCTION

Preston Sand & Gravel Company Limited (Preston) is proposing to develop an aggregate pit (Henning Pit) to be located in the Township of North Dumfries, Regional Municipality of Waterloo, Ontario. Preston is seeking a Class A licence (Category 3) under the Aggregate Resources Act, and a zoning by-law amendment under the Planning Act. Local residents (*i.e.*, Gerry Brown *et al.*) have filed an appeal, and the application has been referred to the Ontario Municipal Board (OMB) for consideration. The appellant cited issues with respect to whether dust and air emissions have been appropriately assessed so as to protect the health of nearby residents. RWDI was retained by Preston to undertake an assessment in support of the applications, to evaluate potential dust emissions and air quality associated with the proposed pit.

As part of the air quality assessment, Intrinsic Environmental Sciences Inc. (Intrinsic) was retained by Preston to assess the potential human health implications associated with modelled air emissions provided by RWDI in the local area of the proposed pit relating to the future facility emissions, as well as increased road traffic arising from the development. In addition, based on information provided by RWDI, Intrinsic evaluated the cumulative modelled air emissions in the area from both the proposed pit and existing background emissions.

To evaluate the potential impacts of increased dust emissions arising from the proposed development, a screening level human health risk assessment (SLHHRA) was completed. The SLHHRA can be used to determine the worst-case health implications of emissions to potentially sensitive individuals living, working, or playing in the surrounding community.

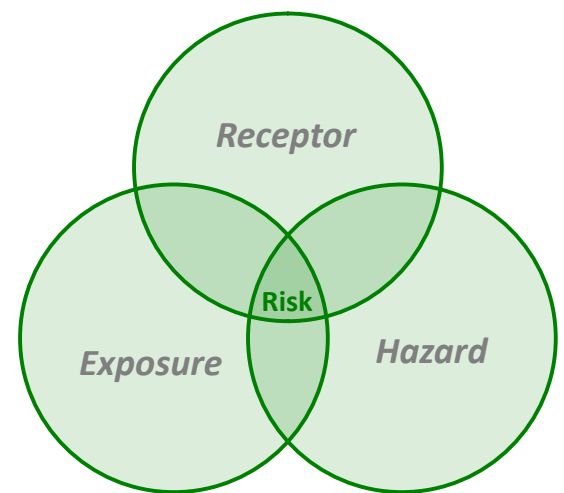
Overall, this project is being completed in addition to the requirements of the Local Air Quality regulation (Ontario Regulation 419/05), which does not consider cumulative, or background air quality, with respect to emissions from a subject facility. This regulation does not provide specific guidance on human health risk assessment methodology, nor specifically those required to complete a SLHHRA. The current human health risk assessment was conducted according to widely accepted risk assessment methodologies and guidance published and endorsed by regulatory agencies including the Ontario Ministry of the Environment (MOE), Health Canada, the Canadian Council of the Ministers of the Environment (CCME), and the United States Environmental Protection Agency (US EPA). The assessment is also limited to the operations evaluated as part of the RWDI Air Quality Assessment (RWDI, 2013).

## 2.0 PROPOSED APPROACH

In general, a human health risk assessment (HHRA) is a scientific study that evaluates the potential for the occurrence of adverse health effects from exposures of people (receptors) to chemicals of concern (COC) present in surrounding environmental media (e.g., air, soil, sediment, surface water, groundwater, food and biota, etc.), under existing or predicted exposure conditions. HHRA procedures are based on the fundamental dose-response principle of toxicology. The response of an individual to a chemical exposure increases in proportion to the chemical concentration in critical target tissues where adverse effects may occur. The concentrations of chemicals in the target tissues (the dose) are determined by the degree of exposure, which is proportional to the chemical concentrations in the environment where the receptor resides, works or visits.

All chemicals (anthropogenic and natural) have the potential to cause environmental effects in people and the ecosystem. However, it is the chemical concentration, the route of exposure, and the inherent toxicity of the chemical that determines the level of environmental effect and potential for unacceptable risk to the exposed receptor. As illustrated in the diagram to the right, if all three components are present (i.e., where the three circles intersect), the possibility of adverse risk exists.

The prediction of an individual's exposure to specific chemicals in the environment and the potential risks resulting from such exposures can be determined through the completion of a quantitative HHRA.



For the current investigation, a screening level human health risk assessment (SLHHRA) has been used to evaluate the potential impacts of projected increase in ambient concentrations of various chemicals of concentrations emitted from the proposed pit, as well as increased road traffic arising from the development. The SLHHRA will attempt to evaluate the potential impacts of both the proposed development, as well as cumulative risk based on the overall contribution of the proposed development to the existing background concentrations within the surrounding airshed. The following section provides a brief overview of the SLHHRA approach to evaluating potential health risk.

### 2.1 Screening Level Human Health Risk Assessment (SLHHRA)

A screening level human health risk assessment (SLHHRA) is a qualitative or quantitative evaluation of risk typically based on a "worst-case" exposure scenario rather than verifiable site-specific conditions. As an initial scoping of potential risk, the SLHHRA approach relies on available data to provide conservative estimates of exposure and risk based on a worst-case scenario, so that exposures and risks are not underestimated. The intent is to determine whether there is the potential for adverse health impacts under these worst-case exposure scenarios for the relevant COC emitted from the proposed Project, identify any data gaps limiting the assessments ability to appropriately estimate exposures and risk, and eliminate any COC and pathways of exposure which are not a concern moving forward for any further analyses.

Should the SLHHRA indicate the potential for the risk, the process provides an excellent foundation on which additional data gathering and analysis can be conducted, in support of a more detailed quantitative human health risk assessment (HHRA). Ultimately, due to the conservative approach and assumptions used, a SLHHRA cannot predict whether potential health risks will occur. Rather, a SLHHRA can only determine if significant human health risks are unlikely. In many cases, a detailed HHRA may be necessary to address the inherent conservatism and uncertainty built into the SLHHRA process, to permit a detailed quantification of actual human health risks related to airborne emissions from the proposed Project, should the SLHHRA indicate the potential for human health impacts.

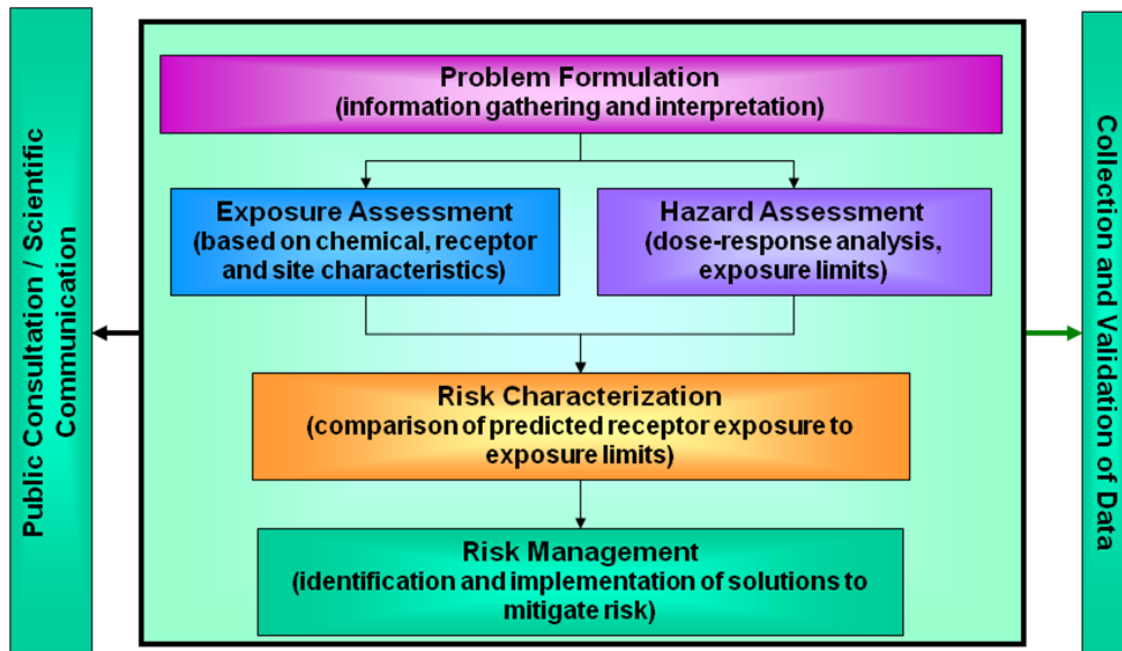
It should be noted that there is no specific regulatory guidance in Ontario for completing a SLHHRA for a sand and gravel pit. The Standards Development Branch (SDB) of the Ontario Ministry of the Environment (MOE) has provided recommendations on the use of SLHHRA related to emissions from transportation corridors (MOE, 2009), which can loosely be considered for these purposes. As noted in their memo, the MOE does provide some guidance for screening level risk assessments (for contaminated sites) as part of O. Reg. 153/04, and specifically the 2005 MOE Procedures document (refer to <http://www.ene.gov.on.ca/envision/gp/5404e.pdf>). As such, the current SLHHRA was conducted according to widely accepted risk assessment methodologies and guidance published and endorsed by regulatory agencies including the MOE (as noted above), Health Canada, the Canadian Council of the Ministers of the Environment (CCME), and the United States Environmental Protection Agency (US EPA).

Overall, the current SLHHRA follows the standard HHRA framework (see Figure 2-1) that is composed of the following steps:

- i) problem formulation;
- ii) exposure assessment;
- iii) hazard assessment; and,
- iv) risk characterization.

Typically, the assessment results provided in the risk characterization step can then be used by risk managers to develop appropriate mitigation plans to prevent adverse health risks in the surrounding community, should they be warranted. However, given this is a screening level assessment, it is likely that should the potential for health risks be identified, a more detailed human health risk assessment could be recommended to provide a more accurate evaluation of site-specific health risks in the community surrounding the proposed Project.





**Figure 2-1 Overview of Standard Risk Assessment Framework**

## 2.2 Problem Formulation

The first step in the SLHHRA process is an information gathering and interpretation stage that plans and focuses the study on critical areas of concern for the Project. Problem formulation defines the nature and scope of the work to be conducted, permits practical boundaries to be placed on the overall scope of work and ensures that the assessment is directed at the key areas and issues of concern. This step is critical to the success of the SLHHRA as sound planning during the problem formulation step reduces the need for significant modifications once the SLHHRA has begun. The data gathered and evaluated in this step provides information into the physical layout and characteristics of the assessment area, possible exposure pathways, potential human receptors, COC, and any other specific areas or issues of concern to be addressed.

The key tasks that comprise the problem formulation step of this SLHHRA include the following:

- **Chemical characterization**, which involves the identification of the COC;
- **Site characterization**, which consists of a review of available project-specific data to identify factors affecting the availability of chemicals to potential receptors;
- **Receptor characterization** to identify “receptors of concern”, which include those individuals with the greatest probability of exposure to chemicals from the proposed Project and those that have the greatest sensitivity to these chemicals; and,
- **Identification of exposure scenarios and pathways** takes into account chemical-specific parameters, such as solubility and volatility, characteristics of the site, such as physical geography, as well as the physiology and behaviour of the receptors.

The outcome of these tasks forms the basis of the approach taken in the current assessment. The following subsections describe the methodological details and outcomes of problem formulation, specific to identification of chemicals, receptors and pathways.

### 2.3 Exposure Assessment

The exposure assessment evaluates data related to all chemicals, receptors and exposure pathways and routes identified during the problem formulation phase. As noted previously, the assessment of potential occurrences of adverse effects from chemicals is based on the dose-response concept that is fundamental to the responses of biological systems to chemicals (Filov *et al.*, 1979; Amdur *et al.*, 1991). Since it is not usually practical to measure concentrations of chemicals at the actual site where the adverse response occurs within tissues and cells, these concentrations are estimated based on either the dose of the chemical that actually enters a receptor or, more commonly, by the concentrations in various environmental media that act as pathways for exposure. The degree of exposure of individuals to chemicals from the environment therefore depends on the interactions of a number of parameters, including:

- The concentrations of chemicals in various environmental media as determined by the magnitude of point sources as well as background or ambient concentrations;
- The characteristics of the chemicals of potential concern which affect environmental fate and persistence (*e.g.*, physical-chemical properties);
- The impact of site-specific characteristics, such as geology, geography and hydrogeology, on chemical behaviour;
- The physiological and behavioural characteristics of the receptors (*e.g.*, respiration rate, soils/dusts intake, time spent at various activities and in different environmental areas); and,
- The various physical, chemical and biological factors that determine the bioavailability of chemicals from various exposure pathways.

The primary objective of the current exposure assessment was to predict, using a series of conservative assumptions, the rate of exposure of individuals living or working in the area around the proposed Project to the COC through various exposure scenarios and pathways identified in the problem formulation step.

### 2.4 Hazard Assessment

The hazard assessment involves identifying and understanding potential health outcomes that can result from exposure to each COC and the conditions under which the outcomes might be observed. The hazard, or toxicity, assessment methodology is based on the fundamental dose response principle. That is, the response of biological systems to chemical exposures increases in proportion to the concentration of a chemical in critical target tissues where adverse health outcomes may occur.

Two basic and quite different chemical categories are commonly recognized by regulatory agencies, depending on the compound's mode of toxic action, and applied when estimating toxicological criteria for humans (FDA, 1982; US EPA, 1989). These are the threshold approach (or the no-observed-adverse-effect levels [NOAELs]/benchmark dose with extrapolation/uncertainty factor approach) typically used to evaluate non-carcinogens, and the non-threshold approach (or the mathematical model-unit risk estimation approach), typically used for carcinogenic compounds.

In the case of threshold chemicals, a benchmark or threshold level must be exceeded for toxicity to occur. A NOAEL can be identified for threshold chemicals, which is the dose or amount of the chemical that results in no observable response in the most sensitive test species and test endpoint. The application of uncertainty or safety factors to the NOAEL provides an

added level of protection, allowing for derivation of a *toxicity reference value* (TRV) that is expected to be safe to sensitive individuals following exposure for a prescribed period of time.

Non-threshold chemicals are capable of producing cancer by altering genetic material. Regulatory agencies such as Health Canada and the US EPA assume that any level of long term exposure to carcinogenic chemicals is associated with some “hypothetical cancer risk”. However, as there is no non-threshold chemicals selected as COCs for the current assessment, this particular approach will not be discussed further.

The terminology used to define threshold and non-threshold TRVs differs according to the source and type of exposure and often varies between regulatory jurisdictions. Generic nomenclature has been developed, with the following terms and descriptions commonly used.

**Reference concentration (RfC):** A reference concentration (or RfC) refers to the acceptable level of an airborne chemical for which the primary route of exposure is inhalation, and applies to either short term acute (e.g., 1-hour or 24-hour) or long term chronic exposure periods. It is expressed as a concentration of the chemical in air (*i.e.*, micrograms per cubic metre,  $\mu\text{g}/\text{m}^3$ ) and applies only to chemicals acting through a threshold mode of toxicological action. For chemicals such as irritants and some combustion gases, short term or acute non-systemic toxicity is frequently observed at the points of entry into the body (*i.e.*, the respiratory tract, eyes, and skin, for air-borne contaminants). In these cases, because the toxicity is enacted simply by direct contact between the receptor and the contaminated medium, the concentration in the air to which the receptor is exposed is the important measure of exposure, rather than the internal dose associated with multiple exposure pathways. For chemicals with these characteristics, short term RfCs are used to characterize health risk, and are intended to be protective of the general population.

The toxicity of a chemical has been observed to vary between acute (short term) and chronic (long term) exposure. Thus, it is important to differentiate TRVs based on duration of exposure. The two TRV durations used in the current HHRA can be described as follows:

- **Acute:** the amount or dose of a chemical that can be tolerated without evidence of adverse health effects on a short term basis. These benchmarks are routinely applied to conditions in which exposures extend from minutes through several hours or several days only (ATSDR, 2006). For the current HHRA, risks will be evaluated based upon 1 or 24-hour exposure periods, where a relevant acute TRV for that time period is available.
- **Chronic:** the amount of a chemical that is expected to be without effect, even when exposure occurs continuously or regularly over extended periods, possibly lasting for periods of at least a year, and possibly extending over an entire lifetime (ATSDR, 2006).

As it would be inappropriate to establish a generic hierarchy of source agencies by which to select TRVs given the breadth of COCs evaluated in a typical human health assessment, when TRVs for a particular COC were available from multiple regulatory agencies, all of the TRVs were reviewed and the professional judgment of experienced toxicologists was used to select the most appropriate TRV. The most critical considerations in selecting TRVs were the source (it must have been derived by a reputable agency), the data used to derive the benchmark, the date the TRV was derived (it must be as up to date as possible), and its relevance in terms of duration and route of exposure. Health Canada (2004) provides a list of acceptable jurisdictions that maybe be used to determine toxicity reference values. In some occasions, additional jurisdictions outside this list can be selected based on the professional judgement of an experienced toxicologist.

## 2.5 Risk Characterization

The final step of a risk assessment is risk characterization. This involves the estimation, description, and evaluation of risk associated with exposure to COC by comparing the estimated exposure to the appropriate TRV for a specific chemical or group of compounds. Risk characterization involves the comparison of estimated exposures (identified in the exposure assessment) with TRVs (identified during the hazard/toxicity assessment) to identify potential human health risks. This comparison is typically expressed as a Concentration Ratio (CR) for non-carcinogenic chemicals and is calculated by dividing the predicted exposure by the TRV.

Separate assessments were completed for short term (acute) and long term (chronic) durations because the health outcomes produced by some COC depend on the duration of exposure. It is important to distinguish between the health outcomes that might result from acute exposures *versus* effects that may occur following chronic exposures. In the chronic assessment, further distinction was made between inhalation and multiple pathway exposures (*i.e.*, oral and dermal) since the pathway of exposure could also influence the potential health outcomes associated with each of the COC.

In recognition of the influence of these exposure variables, risk estimates in inhalation assessments are typically segregated into two exposure categories:

- Acute inhalation (1-hour and 24-hour durations); and,
- Chronic inhalation (annual average durations).

If the COCs were deemed to have the potential to accumulate over time in soil, the assessment could also consider risks related to chronic multiple pathway exposures (*i.e.*, oral and dermal exposures). However, this step was not required for the current assessment given the nature of the assessed COCs and their relative ambient concentrations.

### 2.5.1 Estimating Potential Risk

#### 2.5.1.1 Threshold Chemicals (Non-carcinogens)

##### Concentration Ratios (CR)

CR values were used to evaluate the acute and chronic health risk from exposure to chemicals *via* inhalation. CR values were calculated by dividing the predicted ground-level air concentration (for 1-hour, 24-hour or annual average exposure durations) by the appropriate toxicity reference value (*i.e.*, RfC), according to the following example equation:

$$CR_{duration} = \frac{[Air]_{duration}}{RfC_{duration}}$$

Where:

- $CR_{duration}$  = the duration-specific CR (unitless), calculated for 1-hour, 24-hour and chronic durations, as appropriate
- $[Air]_{duration}$  = the predicted ground-level air concentration ( $\mu\text{g}/\text{m}^3$ ) for the specific time duration
- $RfC_{duration}$  = the RfC ( $\mu\text{g}/\text{m}^3$ ) for the specific time duration

### 2.5.1.2 Non-Threshold Chemicals (i.e., Genotoxic Carcinogens)

#### **Incremental Lifetime Cancer Risks (ILCR)**

ILCR estimates were used to evaluate the increased cancer risk resulting from a lifetime of exposure to non-threshold genotoxic carcinogenic chemicals. ILCR estimates provided the incremental lifetime cancer risk resulting from increased emissions from the Proposed Project, based upon the incremental change in air quality from the Project compared to the existing background air quality in the area.

#### Direct Air Inhalation

ILCR estimates resulting from direct air inhalation were calculated as follows:

$$ILCR = [Air]_{\Delta Project} \times UR$$

Where:

<i>ILCR</i>	=	the incremental (or additional) lifetime cancer risk (unitless)
$[Air]_{\Delta Project}$	=	the predicted annual average ground-level air concentration ( $\mu\text{g}/\text{m}^3$ ) change between current conditions <i>versus</i> future conditions should emissions from the proposed Project be added
<i>UR</i>	=	the COC-specific unit risk ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup>

### **2.5.2 Interpretation of Risk Estimates**

The interpretation of the various risk evaluation metrics, as well as the appropriate benchmark by which to evaluate whether the predicted risk is acceptable or not, are discussed in the following section.

#### 2.5.2.1 Threshold Chemicals (Non-carcinogens)

#### **Concentration Ratio (CR)**

If the risk assessment evaluates risks associated with a single source (such as inhalation), the selection of a CR of 1.0 as an indication that predicted exposures do not exceed the toxicity reference values is appropriate. For example, as gaseous chemicals such as NO<sub>x</sub> only occur in air, and not in other media, the appropriate CR benchmark is 1.0 (i.e., 100% of the TRV is used as the evaluation benchmark).

When predicted risks are greater than the benchmark level (e.g., CR value greater than 1.0), this may indicate the potential for adverse health outcomes in sensitive individuals or in some of the exposure scenarios considered. Re-evaluation of such CRs is important since both the exposure estimates and the toxicological criteria are based on a series of conservative assumptions, particularly when considering the maximum “worst-case” exposure scenarios.

In general, interpretation of the CR values proceeded as follows:

#### CR ≤ 1

- Signifies that the estimated exposure is less than or equal to the TRV (i.e., the assumed safe level of exposure). This shows that negligible health risks are predicted. Added

assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV.

#### CR >1

- Signifies the exposure estimate exceeds the regulatory TRV. This suggests that the potential for an elevated level of risk may be present for some COC. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e.*, the margin of safety is reduced but not removed entirely).

#### 2.5.2.2 Non-Threshold Chemicals (*i.e.*, Genotoxic Carcinogens)

##### **Incremental Lifetime Cancer Risk (ILCR)**

Non-threshold chemicals that can alter genetic material (*i.e.*, genotoxic) are capable of producing cancer. Regulatory agencies such as Health Canada and the US EPA have therefore assumed that any level of long term exposure to a carcinogenic compound is associated with some “hypothetical cancer risk”. As a result, regulatory agencies have typically employed acceptable ILCR levels (*i.e.*, incremental cancer risks over and above background cancer incidence) between 1-in-100,000 and 1-in-1,000,000. ILCRs generally consider risks related to a particular facility (facility alone) in that the cancer risks are expressed on an incremental or additional basis as compared to cancer risks related to all sources.

As this HHRA is being conducted as part of the EIA process for the Province of Ontario, a benchmark ILCR of 1-in-1,000,000 ( $1 \times 10^{-6}$ ) was selected, based upon MOE policy for risk assessments in Ontario. The definition of a benchmark ILCR of 1-in-1,000,000 is a policy based decision, not a scientifically derived value. An ILCR of 1-in-1,000,000 increases a person’s lifetime cancer risk from 0.400000 (based on the 40% lifetime probability of developing cancer in Canada) to 0.400001. It is recognized that some amount of the “background” cancer risk of 40% is likely associated with exposures to environmental pollution. It must be noted, however, that an ILCR of 1-in-1,000,000 (a level below which the MOE considers acceptable) represents a 0.00025% increase over the background cancer incidence, an increase that cannot be detected using epidemiological data from the study area (Health Canada, 2004). It is noted that other regulatory agencies, including Health Canada, consider an ILCR of 1-in-100,000 as the *de minimus* risk level considered protective of public health.

In general, interpretation of the ILCR values proceeded as follows:

#### ILCR $\leq 1.0 \times 10^{-6}$ (1E-06)

- Signifies that the estimated exposure results in an incremental lifetime cancer risk less than or equal to 1-in-1,000,000 (*i.e.*, within the accepted level of risk set by MOE and 10 times lower (more conservative) than that set by the Health Canada). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV and exposure estimate.

#### ILCR $> 1.0 \times 10^{-6}$ (1E-06) $\leq 1.0 \times 10^{-5}$ (1E-05)

- Signifies the estimated exposure results in an incremental lifetime cancer risk greater than the acceptable regulatory-established cancer risk benchmark of 1-in-1,000,000, but less than the 1-in-100,000 benchmark accepted by Health Canada.

ILCR > 1.0 x 10<sup>-5</sup> (1E-05)

- Signifies the estimated exposure results in an incremental lifetime cancer risk greater than the acceptable regulatory-established cancer risk benchmark of 1-in-100,000. This suggests that the potential for an elevated level of risk may be present for some COC. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e.*, the uncertainty is reduced but not removed entirely).

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### **3.0 PROBLEM FORMULATION**

#### **3.1 Site Characterization**

Preston Sand & Gravel Company Limited (Preston) is proposing to develop an aggregate pit (Henning Pit) to be located in the Township of North Dumfries, Regional Municipality of Waterloo, Ontario. Preston is seeking a Class A licence (Category 3) under the Aggregate Resources Act, and a zoning by-law amendment under the Planning Act. Local residents (*i.e.*, Gerry Brown *et al.*) have filed an appeal, and the application has been referred to the Ontario Municipal Board (OMB) for consideration. The appellant cited issues with respect to whether dust and air emissions have been appropriately assessed so as to protect the health of nearby residents. RWDI was retained by Preston to undertake an assessment in support of the applications, to evaluate potential dust emissions and air quality associated with the proposed pit.

As part of the air quality assessment, Intrinsic Environmental Sciences Inc. (Intrinsic) was retained by Preston to assess the potential human health implications associated with modelled air emissions provided by RWDI in the local area of the proposed pit relating to the future facility emissions, as well as increased road traffic arising from the development. In addition, based on information provided by RWDI, Intrinsic evaluated the cumulative modelled air emissions in the area from both the proposed pit and existing background emissions. To evaluate the potential impacts of increased dust emissions arising from the proposed development, a screening level human health risk assessment (SLHHRA) was completed. The SLHHRA can be used to determine the health implications of these increased emissions to potentially sensitive individuals living, working, or playing in the surrounding communities.

The proposed Henning Pit consists of phased extraction and processing of sand and gravel, with up to 750,000 tonnes of material being removed from the site annually. To evaluate the worst-case emissions conditions, two specific scenarios were modelled by RWDI and carried through the current SLHHRA:

##### **Central Processing Plant in Phase 1 Area**

- Excavation and loading of raw material into the trucks at the active face, by front-end loader;
- Hauling of material to the central processing plant in the Phase 1 area;
- Dumping of material in the receiving hopper at the processing plant;
- Processing (crushing, screening, washing and stockpiling) of product in the Phase 1 area;
- Loading of trucks from stockpiles in the Phase 1 area for shipment off site, by front-end loader;
- Hauling of product by truck from stockpiles to the site exit; and,
- Material handling and processing rates based on 750,000 tonnes of annual production over 190 days/year and 11 hours/day.

##### **Auxiliary Processing Plant in Active Phase.**

- Excavation and loading of material directly to the processing plant by front-end loader;
- Processing (crushing, screening and stockpiling) of product in the active phase;
- Loading of trucks from stockpiles in the active phase for shipment off site; and,
- Material handling and processing based on a reduced production rate for the auxiliary plant.



As noted by RWDI (2013), these scenarios represent the worst-case combination of activities at the site likely to occur at the same time. However, Preston has noted that the operation of an auxiliary plant in the active phase and operation of the central plant in the Phase 1 area will not occur at the same time.

For the cumulative assessment, two additional existing adjacent aggregate operations were included in the cumulative effects assessment by RWDI to evaluate background conditions (see Figure 3-1). The first, the CBM-St. Marys site, is located west of the proposed Henning Pit, on the west side of Dumfries Road and consists of two adjacent extraction licences. The licence immediately adjacent to Dumfries Road (Brown Pit) is the more recent of the two and is currently undergoing extraction. The operations at the Brown Pit are similar to the proposed Henning Pit.

The second, the Lafarge site, is located immediately south of the proposed Henning Pit, on the east side of Dumfries Road. Extraction of aggregate is currently being conducted on the eastern half of the site, with a central processing plant located on the western half. Trucks are not used to transport aggregate between the extraction face and the central plant.

Operational plans for both the CBM and Lafarge sites were obtained from the Ministry of Natural Resources by RWDI. It was noted that the operations at these two sites were similar to those proposed for the Henning Pit (RWDI, 2013).

### 3.1.1 Selection of Nearby Receptor Locations

To assess potential risks related to the projected Project emissions, impacts to local air quality were evaluated at discrete locations in the surrounding community. As such, ground-level air concentrations were predicted at seven (7) off-site locations spaced around the footprint of the proposed Project: CRAND4, CRAND5, CRAND6, CRAND15, CHURCH12, RENTAL3, and PAULCAB14 (RWDI, 2013). These locations (see Table 3-1 below) are primary residences in the surrounding area, and one community church (*i.e.*, CHURCH12 which is the Cedar Creek Community Church). The geographical locations of each of these sensitive receptor locations are noted on Figures 3-1.

<b>Table 3-1 Location of Sensitive Receptors selected for Assessment</b>			
<b>Receptor Name</b>	<b>Address</b>	<b>UTM X (m)</b>	<b>UTM Y (m)</b>
CRAND4	1986 Cedar Creek Road, RR4 Cambridge	548506.79	4798914.82
CRAND5	1970 North Dumphries Road, RR4, Cambridge	548628.26	4798736.40
CRAND6	1697 Cedar Creek Road, RR4, Cambridge	549222.23	4798777.88
CRAND15	1809 Dumphries Road, RR4, Cambridge	548511.36	4797503.76
CHURCH2	Cedar Creek Church	548029.94	4798642.77
RENTAL3	Rental Farm House	548253.49	4798921.47
PAULCAB14	N/A	548525.63	4797551.29

It should be noted that since ambient concentrations decline with distance from the source, it would be expected that potential receptor locations further away from the proposed Project would observe even lower impacts than those of the selected locations.

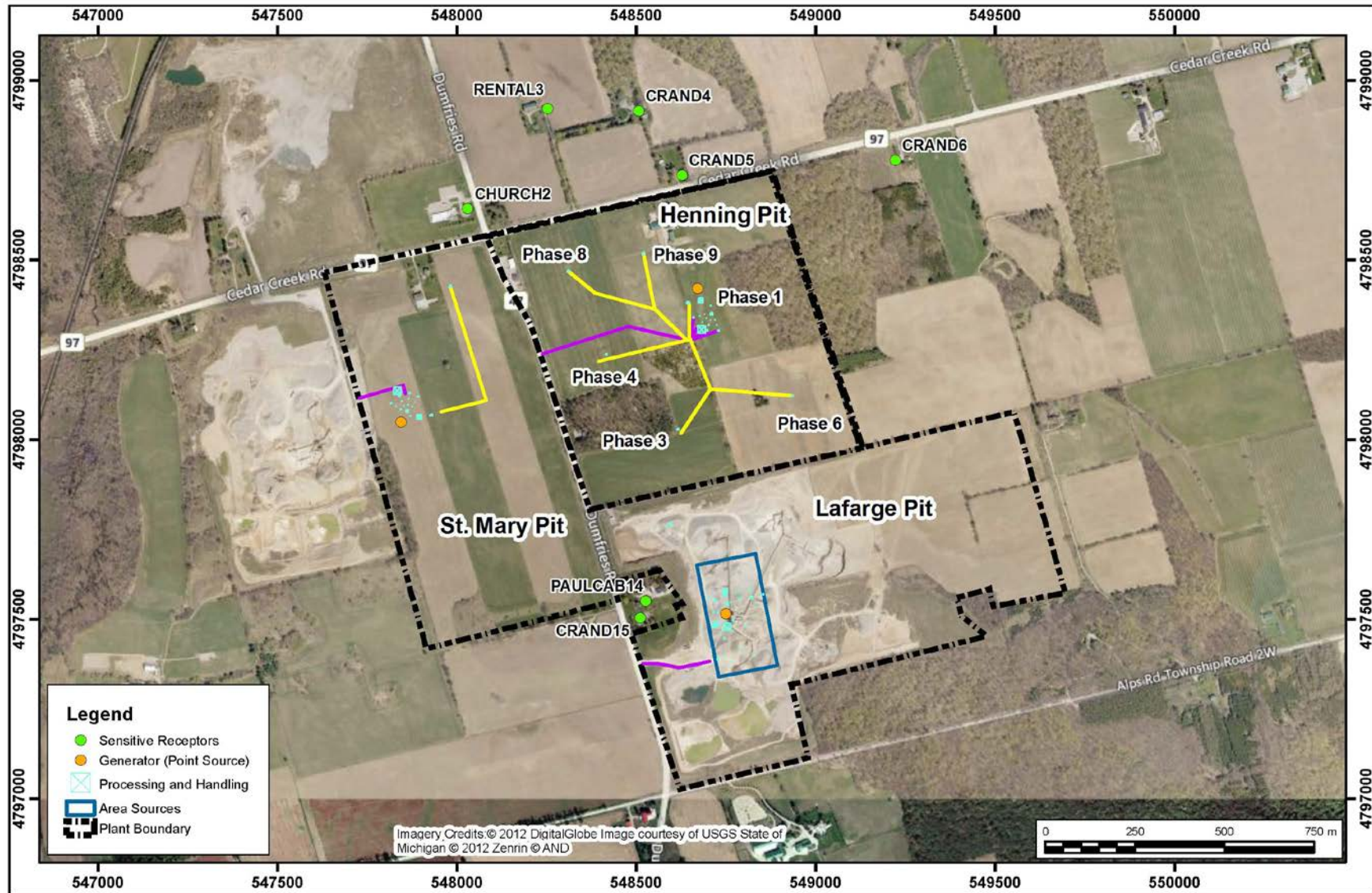


Figure 3-1 Map of Proposed Study Area (RWDI, 2013)

## **3.2 Chemical Characterization**

As noted by RWDI (2013), the primary health concerns for aggregate operations such as the proposed Project are related to their potential to produce dust emissions and products of diesel combustion.

### **3.2.1 Overview of Characteristics of Dusts**

The chemical composition of the dust is based on the composition of the aggregates at the site. No site-specific data on the composition of dust from the proposed Project is available, but RWDI (2013) was able to obtain general data on the composition of limestone and unconsolidated sand in Southern Ontario.

As noted in RWDI (2013), published literature from quarries throughout southern Ontario have indicated that at sites associated with the limestone formation that includes the North Dumfries area (the Guelph-Lockport Dolomite formation), more than 96% (99% in most cases) of the composition consisted of the following:

- Calcium carbonate;
- Oxides of iron, magnesium, and aluminum; and,
- Silica (SiO<sub>2</sub>).

Dust generated at the proposed Henning Pit will originate from a combination of unconsolidated sand, and limestone gravel at the site, with the largest potential source of dust arising from truck movements on unpaved haul roads. Based on this, RWDI (2013) did additional analysis on the available information on the percentage of silica present within the dust emitted from the site, and assumed a middle value of 20%. Refer to the Air Quality Assessment report (RWDI, 2013) for the detailed methodology for this calculation.

Subsequent to the completion of the RWDI (2013) Air Quality Assessment report, Golder Associates (Golder, 2013) conducted an evaluation of silica content in the Henning Pit feed materials. In this analysis, Golder took a sample of sand and gravel from the vicinity of the proposed Project, and determined that the silica content was approximately 16% by mass of the raw material (Golder, 2013). Based on this information, the continued use of an assumed silica content of 20% in airborne particulate is an additional degree of conservatism built into both the Air Quality Assessment and SLHHRA reports.

Operational plans also permit recycled materials (*e.g.*, concrete and asphalt) to be stockpiled and processed in the Phase 1 area of the site, and will be composed of the following materials based on information researched by RWDI (2013). The recycled concrete will consist of aggregates mixed with Portland cement. The Portland cement is made up predominantly of calcium silicates and calcium aluminates, with trace amounts of free calcium oxide (up to about 5%), crystalline silica (up to 7%), slag, fly ash and various free metal oxides. The slag typically consists mainly of calcium oxide and magnesium oxide, and fly ash consists mainly of amorphous silica and metal oxides.

Recycled asphalt consists of aggregates mixed with asphalt cement (bitumen). The asphalt cement consists mainly of a range of organic compounds, of which the primary chemicals of concern would be the family of polycyclic aromatic hydrocarbons (PAHs). As this was based on recycled road asphalt, RWDI (2013) estimated the PAH content of asphalt dust using published data for paved road dust.

### 3.2.2 Overview of Characteristics of Diesel Emissions

In brief, diesel engines operate through the injection of fuel into air within a combustion chamber at high pressure and temperature (US EPA, 2002). The ignition of injected fuel results in the release of chemical energy, and the expanding combustion gases pushing a piston prior to being released into the atmosphere. The amount of injected fuel controls the power output of the engine. Diesel exhaust emissions are constituted of a complex mixture of chemical and physical components, including gases, vapours, and fine particles (known as “soot”), that are formed through the complete and incomplete combustion of fuel (US EPA, 2002; Health Canada, 2006a).

Major gaseous products emitted include oxygen, nitrogen, water vapour, carbon dioxide (CO<sub>2</sub>), carbon monoxide (CO), oxides of nitrogen (NO<sub>x</sub>), sulphur dioxide (SO<sub>2</sub>), and volatile organic compounds (VOC) (*i.e.*, low-molecular-weight hydrocarbons) (WHO, 1996; US EPA, 2002). Such gaseous hydrocarbons include benzene, 1,3-butadiene, aldehydes (*e.g.*, formaldehyde, acetaldehyde, acrolein), polycyclic aromatic hydrocarbons (PAHs), and nitro-PAHs (US EPA, 2002). Generally, diesel engines operating without emission controls emit high concentrations of particles, NO<sub>x</sub>, and aldehydes and low concentrations of CO and hydrocarbons. The NO<sub>x</sub> in diesel exhausts contain a higher fraction of NO<sub>2</sub> than exhaust from gasoline engines because the excess air intake of diesel engines allows greater conversion of NO to NO<sub>2</sub>.

Diesel particles are complex, covering a range of sizes and morphologies, and having a myriad of chemical components that vary with engine characteristics, operating conditions, and fuels (WHO, 1996; US EPA, 2002). Diesel particulate is released directly from diesel-powered engines, but can also be formed from the gaseous compounds emitted by diesel-powered engines (*i.e.*, secondary formation (US EPA, 2002)). Diesel particulate emissions are composed of elemental carbon (EC) as the central core and chemical species that condense onto these nuclei when exhaust gases cool (US EPA, 2002). Typically, adsorbed organic compounds contribute 10 to 30% of the total particulate matter (PM) and originate from fuel and lubricating oil (WHO, 1996). Particles are also composed of much smaller amounts of inorganic compounds including sulphate, nitrates, metals, and other trace elements which originate from diesel oil and engine material (Health Canada, 2006a). PAHs, nitro-PAHs, and oxidized PAH derivatives can also be present on diesel exhaust particles (US EPA, 2002). Diesel engines characteristically release significant amounts of PM, typically producing particles at a rate about 20-times greater than from gasoline engines; however, over the past decade, modifications of diesel engine components have significantly reduced particle emissions (WHO, 1996; US EPA, 2002).

Diesel exhaust emissions are significantly reduced by the use of engine designs that exploit clean-diesel technologies and use such features as particle traps/filters to remove particles and catalytic converters to reduce levels of carbon monoxide and gaseous hydrocarbons emitted into the environment (WHO, 1996). The use of low-sulphur diesel fuel also effectively reduces emission of sulphur particulates, and ensures that sulphur emissions do not impair the effectiveness of catalytic converters and particles filter devices.

For the current screening level risk assessment, NO<sub>x</sub> and PAHs were selected as the worst-case surrogates for exposures to diesel emissions from the proposed Project, with PM<sub>10</sub> and PM<sub>2.5</sub> also carried forward in combination with the dust emissions from the Project and surrounding area.

### 3.2.3 Selection of Chemicals of Concern (COC)

Based upon the primary components present in aggregate dust and diesel exhaust from the vehicles involved with the operations, and the contaminants addressed in the Air Quality Assessment Report (RWDI, 2013), the following contaminants were selected as COC for the current screening level assessment:

- Crystalline silica;
- Respirable particulate matter (PM<sub>10</sub>);
- Fine particulate matter (PM<sub>2.5</sub>);
- Oxides of nitrogen (NO<sub>x</sub>); and,
- Benzo[a]pyrene (surrogate for the PAH group).

The following sections provide a brief description of each selected COC, and their typical sources within the environment (both natural and anthropogenic).

#### 3.2.3.1 Crystalline Silica

Crystalline silica is a basic component of soil, sand, granite, and many other minerals. Quartz is the most common form of crystalline silica, followed by cristobalite and tridymite being the other two forms. All three forms may become respirable size particles when industrial activities chip, cut, drill, or grind objects that contain crystalline silica.

Silica is one of the most documented workplace contaminants. Occupational exposure to crystalline silica is regulated based on evidence that it has the potential to cause adverse health effects such as silicosis (a type of pneumoconiosis marked by inflammation and scarring in forms of nodular lesions in the upper lobes of the lungs) and lung cancer. It is well established that silicosis development in worker populations is exposure and dose-related, and that there is strong evidence for crystalline silica being the causal agent.

The toxicity of silica appears to be largely mediated by the alveolar macrophage response to the presence of foreign particles, and the subsequent inflammatory, oxidative stress and fibrotic responses that are associated with the macrophage response. These processes can continue after external inhalation exposure has ceased. The deposition of silica particles in the alveolar region can lead to a positive feedback cycle of oxidative stress, inflammation and fibrosis.

Thus, crystalline silica-induced pulmonary effects are irreversible; a fact that has been demonstrated in a number of human and animal studies, where limited or no recovery to a pre-exposure state was observed. The mode of toxic action for crystalline silica is also believed to be influenced by the form of silica and surface features, such as the presence of trace elements that are sorbed to the silica mineral and surface-active radicals associated with freshly fractured crystalline silica.

Four subtypes of silicosis have been characterized:

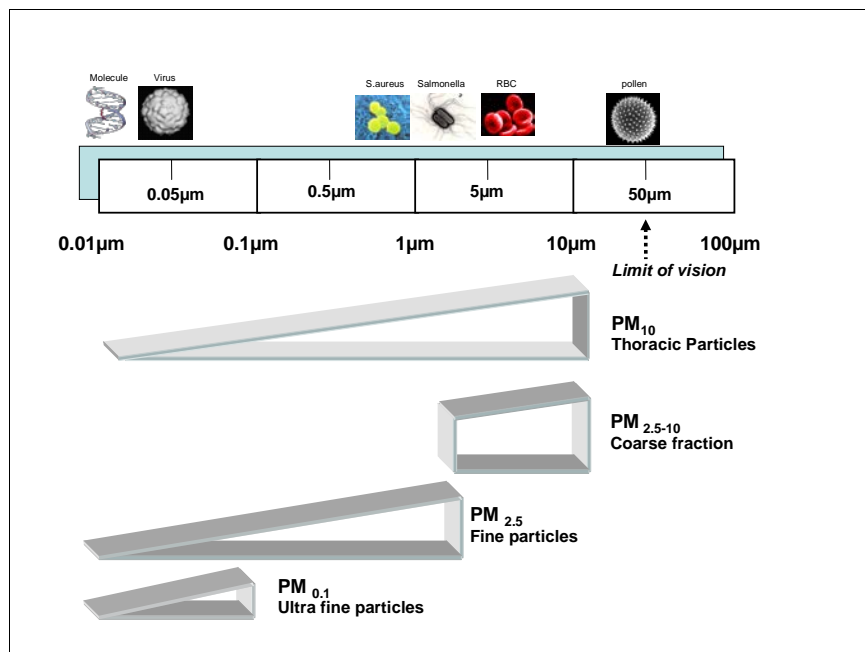
- **Chronic silicosis** usually develops after 10 or more years of prolonged inhalation exposure to crystalline silica, often at relatively low concentrations. It is the most common form of silicosis. This condition can continue to progress even after a subject is removed from the source of silica exposure.

- **Complicated (or conglomerate) silicosis** can occur when the silicotic lesions in subjects with chronic silicosis coalesce into larger lesions. This condition is sometimes referred to as progressive massive fibrosis.
- **Accelerated silicosis** usually develops after 5 to 10 years exposure to elevated crystalline silica air concentrations, or 5 to 10 years after a single high intensity exposure. If the initial exposures are sufficiently high, this condition can progress quickly such that death from respiratory failure could occur in as little as 10 years, even if the worker is removed from the source of silica exposure.
- **Acute silicosis** develops after massive over-exposure to very high air concentrations of respirable crystalline silica and results in symptoms within a few weeks to 4 or 5 years after the initial exposure. This form of silicosis is very rare, often severe, and frequently fatal. This condition is largely attributed to high intensity exposure to freshly fractured crystalline silica.

From a non-occupational point-of-view (such as for exposures in the residential areas around the proposed Project), typically the only type of silicosis which one could experience would be chronic silicosis where one is exposed to prolonged elevated concentrations of crystalline silica for an extended period of time (e.g., 10 or more years). Ambient concentrations outside of occupational settings rarely reach consistent exposure levels so as to result in acute or accelerated silicosis. Furthermore, chronic silicosis is a condition rarely observed outside of occupational settings due to the need for prolonged inhalation exposures to silica.

### 3.2.3.2 Particulate Matter (PM)

PM consists of airborne particles in solid or liquid form, the size of ambient PM ranging from approximately 0.005 to 100 microns ( $\mu\text{m}$ ) in aerodynamic diameter (WHO, 2000).



PM is operationally separated into three groups: total suspended particulate (TSP), inhalable coarse particles ( $PM_{10}$  and  $PM_{2.5-10}$ ) and fine or respirable particles ( $PM_{2.5}$  and  $PM_{0.1}$ ) (Environment Canada, 2000). It is important to recognize that TSP contains all particles smaller than 44 microns;  $PM_{10}$  contains all particles with a mean aerodynamic diameter of less than 10 microns; and  $PM_{2.5}$  contains particles smaller than 2.5 microns as well as ultrafine PM of less than 0.1 micron (US EPA, 2004). The largest particles (coarse particles in particular) form the highest proportion of the mass of ambient particles; the smallest, ultrafine particles, comprise only 1 to 8% of this mass.

PM is ubiquitous in the environment as it is emitted from both natural and anthropogenic sources. Suspended particulate may be emitted directly into the atmosphere (*i.e.*, primary PM), or can be formed in the atmosphere from precursor gases *via* physical and chemical transformations (*i.e.*, secondary PM), such as nitrogen oxides ( $NO_x$ ) reacting to form nitrate PM (Environment Canada, 2006). When characterizing possible health effects associated with exposure to PM it is important to consider the source and the associated chemical composition of particulate mixtures.

#### *Inhalable Coarse Particles ( $PM_{10}$ )*

Characteristics of coarse particles include composition and sources. Coarse  $PM_{10}$  is composed of suspended soil or street dust, fly ash from uncontrolled combustion of coal, oil, and wood, nitrates/chlorides/sulfates from  $HNO_3/HCl/SO_2$  reactions with coarse particles. It also includes oxides of crustal elements (Si, Al, Ti, Fe),  $CaCO_3$ ,  $CaSO_4$ , NaCl, sea salt, bacteria, pollen, mold, fungal spores, plant and animal debris, tire, brake pad, and road wear debris. Sources of  $PM_{10}$  include resuspension of particles deposited onto roads; suspension from disturbed soil (*e.g.*, farming, mining, unpaved roads); construction and demolition, and uncontrolled coal and oil combustion.

$PM_{10}$  has become the indicator for purposes of regulating the coarse fraction (referred to as thoracic coarse particles or coarse-fraction particles; generally including particles with a nominal mean aerodynamic diameter greater than 2.5  $\mu m$  and less than or equal to 10  $\mu m$ , or  $PM_{10-2.5}$ ) (US EPA, 2008a)

According to 2005 estimates, open sources (*e.g.*, paved and unpaved roads, construction operations, agricultural tilling and wind erosion, *etc.*) account for approximately 90% of total  $PM_{10}$  emissions in Canada (Environment Canada, 2006). Fixed site monitoring of 24-hour concentrations showed that the long term mean  $PM_{10}$  concentrations in Canada during the mid-1980s to mid-1990s ranged from 11 to 42  $\mu g/m^3$  at urban sites, while rural sites in the mid-1990s experienced  $PM_{10}$  concentrations ranging between 11 and 17  $\mu g/m^3$  (Health Canada, 1999).

#### *Fine Particles ( $PM_{2.5}$ )*

Fine PM ( $PM_{2.5}$ ) and its precursor gases largely originate from combustion processes (Health Canada, 1999). PM is typically composed of sulphate, nitrate, ammonium, inorganic and organic carbon compounds and heavy metals such as lead and cadmium (Health Canada, 1999). Sulphate has repeatedly been shown to be the most abundant single component of fine particles ( $PM_{2.5}$ ). The sulphate component of PM tends to be acidic in nature. Primary precursor pollutants in the formation of secondary  $PM_{2.5}$  include  $SO_2$ ,  $NO_x$ , VOCs, and  $NH_3$  (Environment Canada, 2006).

According to 2005 estimates, open sources (e.g., paved and unpaved roads, forest fires and prescribed burning, construction operations, etc.) account for approximately 64% of total PM<sub>2.5</sub> emissions in Canada (Environment Canada, 2006), while industrial processes (e.g., wood and pulp and paper industries), non-industrial fuel combustion (e.g., residential heating), and transportation (e.g., diesel vehicles) account for the majority of other emissions (Environment Canada, 2000). Measured concentrations of PM<sub>2.5</sub> at urban and rural sites ranged from 6.9 to 20.2 µg/m<sup>3</sup> and 7.0 to 10.5 µg/m<sup>3</sup>, respectively (Health Canada, 1999).

Refer to Section 4.3 for a further discussion of ultrafine particulate matter (PM<sub>0.1</sub>).

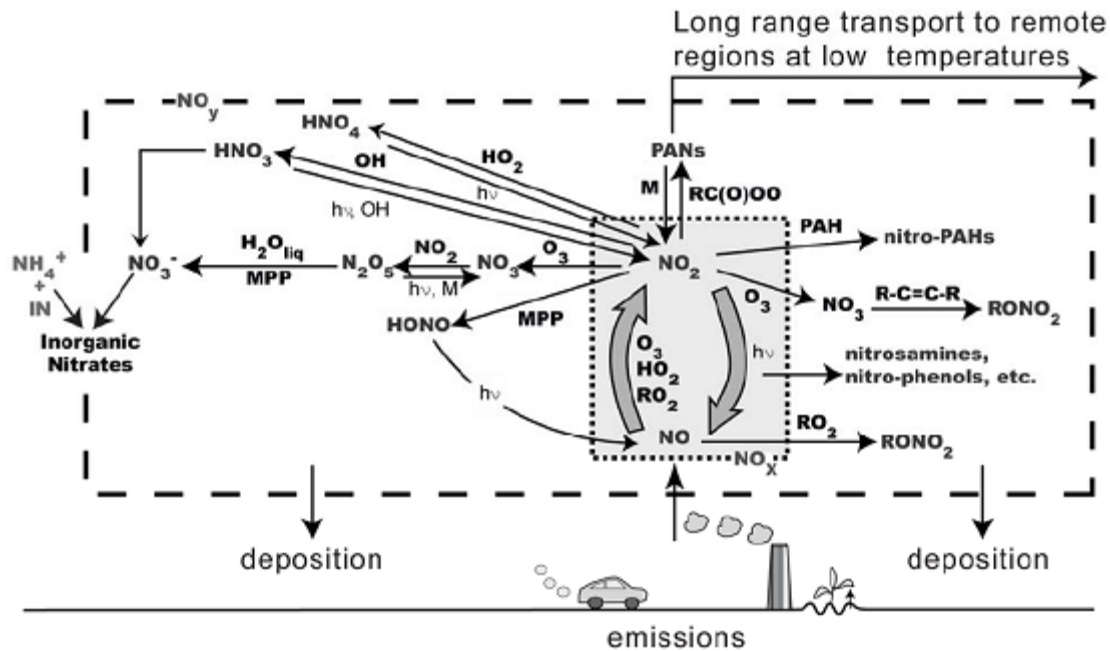
### 3.2.3.3 Oxides of Nitrogen (NOx)

Nitrogen is the most abundant element in ambient air, existing primarily as N<sub>2</sub> (Cal EPA, 2007). Oxides of nitrogen (NOx), including nitric oxide (NO) and nitrogen dioxide (NO<sub>2</sub>) gases, are produced by both natural and human activities, primarily formed by the reaction of atmospheric oxygen and nitrogen during high temperature combustion processes (Environment Canada, 2006). Specific anthropogenic sources of NOx include transportation, stationary source fuel combustion, industrial processes, solid waste disposal, and other sources (e.g., forest fires) (US EPA, 1993). Approximately 2.4 million tonnes of NOx were released in Canada in 2005, not accounting for the emissions from open sources (e.g., forest fires, prescribed burning) (Environment Canada, 2006). The primary emitting sources were on-road and off-road transportation (53 % of emissions), the upstream oil and gas industry (19% of emissions), and fossil-fuelled electric power plants (10% of emissions) (Environment Canada, 2006). Even in the absence of human activity, NOx is ubiquitous in the environment, produced naturally by biological and abiological processes in soils, biomass burning, lightning, and to a lesser extent by oxidation of ammonia, stratospheric intrusion, and oceans (US EPA, 1993; Cal EPA, 2007).

NOx released *via* combustion processes (e.g., motor vehicles, fossil fuel power stations) is mostly in the form of NO and to a lesser extent NO<sub>2</sub> (usually less than 10%) (WHO, 1997). In the presence of ozone (O<sub>3</sub>) or in a photochemically active reactive atmosphere, NO is quickly oxidized to NO<sub>2</sub> (US EPA, 1993; WHO, 1997). NO<sub>2</sub> is subject to further extensive atmospheric transformations, ultimately leading to the formation of strong oxidants (*i.e.*, ozone) and the conversion of NO<sub>2</sub> to nitric acid (NO<sub>3</sub>) (Forastiere *et al.*, 2006). NO<sub>3</sub> may be found as a vapour, or deposited on particular matter or other surfaces; NO<sub>3</sub> levels can affect visibility (Cal EPA, 2007). Ammonium nitrate (NH<sub>4</sub>NO<sub>3</sub>) particles may be subsequently produced from NO<sub>3</sub> *via* reaction with ammonia (NH<sub>3</sub>) in the atmosphere (Cal EPA, 2007). Thus, through the photochemical reaction sequence initiated by solar-radiation-induced activation of NO<sub>2</sub>, the newly generated pollutants are an important source of nitrate particles currently measured as PM<sub>2.5</sub>. For these reasons, NO<sub>2</sub> is a key precursor of a range of secondary pollutants whose effects on human health are well-documented (Forastiere *et al.*, 2006). NO and NO<sub>2</sub> can also undergo reactions to form other oxides of nitrogen (both in indoor and outdoor air) including HNO<sub>2</sub>, HNO<sub>3</sub>, nitrogen trioxide (NO<sub>3</sub>), dinitrogen pentoxide (N<sub>2</sub>O<sub>5</sub>), peroxyacetyl nitrate (PAN) and other organic nitrates (WHO, 1997).

Figure 3-3 (from US EPA, 2008b) provides a schematic diagram of the cycle of reactive oxidized N species in the atmosphere, and demonstrates the potential for inter-conversion of various NOx species in the environment. It should be noted that the “NOx” group of chemicals are generally considered to consist of all nitrogen-containing compounds shown inside the large dashed-line box.





**Figure 3-3 Schematic Diagram of the Cycle of Reactive Oxidized N species in the Atmosphere (US EPA, 2008b)**

Significant concentrations of NO<sub>x</sub> can be found in both ambient and indoor air (WHO, 1997); heaters and gas stoves may produce substantial amounts of NO<sub>2</sub> in indoor settings (Cal EPA, 2007). Human exposures to NO<sub>x</sub> vary greatly from indoors to outdoors and from urban to nonurban areas. NO<sub>x</sub> due to the proximity to combustion sources (traffic or industry) and exposure is also dependant on the time of day and season (WHO, 1997). During the summer months, photochemical reactions tend to increase the ratio of NO<sub>x</sub> to NO. The primary route of exposure to NO<sub>x</sub> is through inhalation. Ambient concentrations of NO and NO<sub>x</sub> tend to be greatest in the cities (WHO, 1997). Higher concentrations of NO are commonly found in street canyons due to vehicular emissions (WHO, 1997). In rural areas, where there is less vehicular traffic, NO<sub>x</sub> may have spent considerable time in the atmosphere and thus undergone reactions to produce significant concentrations of other species, such as HNO<sub>3</sub> and PAN (*i.e.*, 1-(2-pyridylazo)-2-naphthol) (WHO, 1997). While the NO<sub>x</sub> emitted and produced in the atmosphere are removed *via* wet precipitation and dry deposition, NO<sub>x</sub> in indoor air dissipates *via* infiltration into household materials (WHO, 1997).

It is important to note that while NO<sub>x</sub> is emitted from a diesel vehicle or generator, nitrogen dioxide (NO<sub>2</sub>) is the most toxicological significant of the oxides of nitrogen. As NO is considerably less toxic than NO<sub>2</sub>, one is over-estimating potential health risk if one conservatively assumes all NO<sub>x</sub> is in the form of NO<sub>2</sub>, as we done in the current assessment.

#### 3.2.3.4 Polycyclic Aromatic Hydrocarbons (PAHs)

PAH are a large class of organic compounds (>100 compounds) made up of carbon and hydrogen grouped into two or more fused aromatic rings (ATSDR, 1995; WHO, 1998). PAHs almost always occur in the environment as complex mixtures which are difficult to characterize; the chemical constituents of the mixtures generally vary with the source (WHO, 1998). PAHs are primarily formed during the incomplete combustion of organic matter, including the burning of gas, oil, coal, complex petroleum products, products of coal liquefaction processes, plant and

animal matter, garbage, wood (biomass), and tobacco (Cal EPA, 1994). To a far lesser extent, PAHs may be produced by diagenesis or biosynthesis, and are found naturally in coal derivatives and petroleum (Environment Canada, 1994).

Sources in the environment include natural (volcanic eruptions, peat fires, forest fires, and burning crude oil or shale) and anthropogenic sources (burning of fossil fuels, coke oven emissions, aluminum smelters, coal combustion, conversion industries, vehicle exhaust, tobacco smoke, incinerators, and biomass burning) (Cal EPA, 1994). PAHs may be present in relatively high concentrations in some manufactured products despite the fact that most compounds are not generally manufactured commercially (ATSDR, 1995). PAHs are largely emitted to the atmosphere adsorbed onto PM, but may also be found in the gaseous phase (ATSDR, 1995). PAHs are found in water and soil largely from deposition of air emissions (Environment Canada, 1994). According to 1990 estimates, forest fires were the largest source of atmospheric PAHs in Canada (representing 47% of total emission inventoried) (Environment Canada, 1994). The aluminum smelting industry, residential wood heating, open-air fires/agricultural burning, incineration by saw mills, and transportation represent other significant sources of PAHs in Canada.

As indicated in Health Canada (2006b), as well as most other regulatory guidance, the assessment of risks related to exposures to carcinogenic PAHs is primarily conducted through the use of potency or toxicity equivalence factors (PEF or TEF). TEFs allow large groups of compounds with a common mechanism of action such as PAHs to be assessed when limited data is available for all but one of the compounds (*i.e.*, benzo(a)pyrene or b[a]p). Through this approach, exposures to each of the carcinogenic PAHs are adjusted by their carcinogenic potency relative to benzo[a]pyrene. These potency-adjusted exposures can then be summed to provide an overall exposure to the group of carcinogenic PAHs, based on benzo[a]pyrene as the primary surrogate.

For the current assessment, emission data for the entire family of PAHs were available as a toxicological equivalence of benzo[a]pyrene (*i.e.*, b[a]p-TEQ). Using this approach, the quantity of each PAH emitted by the diesel vehicles was adjusted for its toxicological potency *versus* benzo[a]pyrene (*i.e.*, its TEF), and summed to produce an overall b[a]p-TEQ emission rate for PAHs emitted from the Project vehicles, as well as present in recycled asphalt.

Benzo[a]pyrene, itself, is a five-ring PAH that accounts for less than five percent of the total amount of atmospheric PAHs (Cal EPA, 1994), but is a persistent compound, poorly volatile, slightly soluble in water, readily sorbed to air and dust particles and one that tends to partition predominantly in soil and sediments (Cal EPA, 1994). Benzo[a]pyrene has an equally distributed electronic density leading it to be metabolized as a carcinogen (ATSDR, 1995). Humans are exposed to PAHs through the inhalation of ambient air, tobacco smoke, wood smoke, and consumption of PAHs in foods (ATSDR, 1995). While most direct releases of benzo[a]pyrene are to the atmosphere, there is some suggestions that the dominant pathway of human exposure is the food chain (Hattermer-Frey and Travis, 1991). Inhalation of benzo[a]pyrene and ingestion of contaminant water are much more minor pathways of exposure.

### 3.3 Receptor Characterization

A human receptor is a hypothetical person (*e.g.*, infant, toddler, child, adolescent, or adult) who resides and/or works in the area being investigated and is, or could potentially be, exposed to the chemicals identified as being of potential concern. The assessment must be sufficiently comprehensive to ensure inclusion of those receptors with the greatest potential for exposure to

COC, and those who have the greatest sensitivity, or potential for developing adverse health outcomes from these exposures.

For the assessment of inhalation risks, as a straight comparison between predicted short term, acute (*i.e.*, for 1-hour and 24-hour exposure durations) and long term, chronic (*i.e.*, annual average exposures) air concentrations and the corresponding regulatory benchmark (RfC) is made, the resulting CR value is receptor-independent (*i.e.*, the same value is calculated for all receptor types).

### **3.4 Identifying Exposure Scenarios and Pathways**

#### **3.4.1 Exposure Scenarios**

To properly evaluate potential exposures to residents living and working in the area directly surrounding the proposed Project, the following exposure scenarios were assessed:

- Central Processing Plant in Phase 1 Area
  - Project Only (Henning Pit)
  - Cumulative Exposure (*i.e.*, Project plus existing background conditions)
- Auxiliary Processing Plant in Active Phase
  - Project Only (Henning Pit)
  - Cumulative Exposure (*i.e.*, Project plus existing background conditions)

#### **3.4.2 Exposure Pathways**

The primary exposure pathway under evaluation in the current assessment is the inhalation of the COC by individuals living, working or playing in the residencies surrounding the proposed Project. Deposition onto soils is not a significant risk for the COCs selected for the current assessment, and as such, oral and dermal exposures were not evaluated.

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## 4.0 EXPOSURE ASSESSMENT

The exposure assessment evaluates data related to all chemicals, receptors and exposure pathways and routes identified during the problem formulation phase.

For the inhalation exposure assessment, specific rates of exposure were not calculated. Rather, ambient air concentrations (measured or modelled) of each COC (expressed as  $\mu\text{g}/\text{m}^3$ ) were compared to acceptable air concentrations (also expressed as  $\mu\text{g}/\text{m}^3$ ). The inhalation exposure assessment identified potential health risks from acute and chronic exposures (*via* direct air inhalation only) for all of the COC at each of the assessed human health receptor locations.

### 4.1 Estimation of Ambient Ground Level Air Concentrations

Estimates of the potential impacts on air quality related to activities associated with the proposed Project were based on predicted ground level air concentrations for each of the COC. Air dispersion modelling considered not only particulate-based emissions from the Project site, but also re-entrainment of particulate from access roads surrounding the Project location. Ground level air concentrations were estimated by the air quality assessment team at each of the seven (7) sensitive receptor locations, for each of the COC (RWDI, 2013).

Ground level air concentrations for each COC were predicted for 1-hour, 24-hour, and annual average exposure durations, based upon the results of air dispersion modeling for the Central Processing Plant and Auxiliary Processing plant scenarios. Concentrations were predicted for both Project Only and Cumulative exposure conditions.

When evaluating the impact of a project on local air quality, it is important for the evaluation to include a general estimate of baseline (*i.e.*, "background") ambient air quality in the region. By combining both existing local background air quality with the estimated future contribution from the Project site, a conservative estimation of cumulative exposures can be estimated.

An appropriate reasonable worst-case regional background concentration for dust and diesel-related COC for the study area was estimated using ambient air quality data collected from a number of distinct federal and provincial monitoring stations near the study area. These monitors are part of the National Air Pollution Surveillance (NAPS) network operated by the MOE and Environment Canada. Refer to RWDI (2013) for detailed information on the various monitoring stations and the methodology used to estimate local background conditions.

Note that the concentrations presented in Tables 4-1 and 4-2, and carried through the current assessment, are based upon the full dataset modelled by RWDI. The values presented in the RWDI (2013) report may be slightly less in some cases, as the O. Reg. 419 protocol used by RWDI resulted in the removal of some data points arising from meteorological outliers in the reporting of their analysis. For the current assessment, all modelled results were conservatively assumed for the prediction of worst-case risk at each sensitive receptor location.

**Table 4-1 Maximum Predicted Henning Pit and Cumulative Ground-Level Air Concentrations (in  $\mu\text{g}/\text{m}^3$ ) at each Sensitive Receptor Locations for the Central Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>1-hour Exposure Period</b>															
NO <sub>x</sub>	35	263	337	352	409	<200	<200	219	259	202	599	222	296	235	280
<b>24-hour Exposure Period</b>															
NO <sub>x</sub>	35	26.8	74.3	36	76.9	19.7	56.3	22.7	67.9	25.7	87.9	25.7	63.7	24.8	71.6
PM10	39	14.5	73	18.9	76	14.3	64	16.1	123	14.7	65	8.8	57	17.2	133
PM2.5	21	2.7	26	3.4	26	2.4	25	3.1	30	2.5	25	1.5	24	3.4	31
Silica	2.34	2.9	9	3.8	10	2.9	7	3.2	19	3.0	7	1.8	6	3.4	21
<b>Annual Exposure Period</b>															
PM2.5	8	0.08	8.1	0.18	8.2	0.060	8.1	0.042	8.5	0.080	8.3	0.053	8.1	0.048	8.6
PAHs (B[a]P surrogate)	0.000015	0.00000082	0.000017	0.0000016	0.000018	0.00000076	0.000017	0.00000060	0.000022	0.00000081	0.000020	0.00000070	0.000017	0.00000068	0.000022
Silica	0.96	0.074	1.1	0.15	1.2	0.058	1.1	0.045	1.8	0.074	1.3	0.053	1.1	0.051	1.8

Note: All units are in  $\mu\text{g}/\text{m}^3$ .

1-hour NO<sub>x</sub> values shown as "<200" indicate air concentrations modelled by RWDI were below the 200  $\mu\text{g}/\text{m}^3$  threshold and were not retained individually in the subsequent model output. As such, better precision as to the predicted concentration is unavailable at this time.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

**Table 4-2 Maximum Predicted Henning Pit and Cumulative Ground-Level Air Concentrations (in  $\mu\text{g}/\text{m}^3$ ) at each Sensitive Receptor Locations for the Auxiliary Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>1-hour Exposure Period</b>															
NO <sub>x</sub>	35	<200	214	<200	219	<200	154	<200	236	<200	596	<200	293	<200	275
<b>24-hour Exposure Period</b>															
NO <sub>x</sub>	35	15.9	59.4	27.8	65.6	8.2	47.7	5.8	68.0	21.8	88.1	14.8	51.5	6.3	71.6
PM10	39	9.6	67.5	19.9	72.6	8.5	58.0	12.4	123	13.7	65.4	7.9	55.4	13.5	133
PM2.5	21	1.1	24.2	2.2	25.7	0.97	23.2	1.4	30	1.6	25.0	0.89	23.3	1.6	31.4
Silica	2.3	1.9	8.0	4.0	9.1	1.7	6.1	2.5	19.1	2.7	7.6	1.6	5.6	2.7	21.1
<b>Annual Exposure Period</b>															
PM2.5	8	0.055	8.1	0.12	8.2	0.030	8.1	0.025	8.5	0.057	8.3	0.042	8.1	0.030	8.6
PAHs (B[a]P surrogate)	0.000015	0.0000020	0.000019	0.0000039	0.000021	0.0000013	0.000017	0.00000061	0.000022	0.0000018	0.000021	0.0000015	0.000018	0.00000072	0.000022
Silica	0.96	0.050	1.1	0.15	1.2	0.035	1.1	0.038	1.8	0.083	1.3	0.052	1.1	0.044	1.8

Note: All units are in  $\mu\text{g}/\text{m}^3$ .

1-hour NO<sub>x</sub> values shown as "<200" indicate air concentrations modelled by RWDI were below the 200  $\mu\text{g}/\text{m}^3$  threshold and were not retained individually in the subsequent model output. As such, better precision as to the predicted concentration is unavailable at this time.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

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## 4.2 Exposure Analysis of Particulate Matter

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 about 10 microns ( $\mu m$ ) in aerodynamic diameter ( $>PM_{10}$ ) are deposited almost exclusively in the nose, throat, and upper respiratory tract, and tend to be coughed out or swallowed 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. Health effects associated with particles greater than  $PM_{10}$  are considered less critical compared to fractions less than 10 microns in size since they are less likely to be absorbed into the body *via* inhalation. Fine and ultrafine particles ( $<2.5 \mu m$ ), on the other hand, are small enough to reach the alveoli (air spaces) deep in the lungs. In general, it may be assumed that the smaller the particle, the greater the potential to reach respiratory structures such as alveoli where blood-gas exchange occurs. Inhaled fine and ultrafine particles can also carry adsorbed chemical pollutants to the deeper lung structures. Smaller particles tend to be present in greater numbers, and they possess a greater total surface area than larger particles of the same mass.

The potential impacts of human exposure to the respirable fraction of PM (*i.e.*,  $PM_{2.5}$ ) is emphasized in the current HHRA, rather than the broader size fraction represented by total suspended particulate (*i.e.*, TSP, comprising particles ranging up to 44  $\mu m$  in size). The inhalable fraction (*i.e.*,  $PM_{10}$ ) is also widely used to evaluate potential health issues, since this size of particle primarily affects tissues in the upper airways, but can also travel deep into the lung. When both sets of data are available ( $PM_{10}$  and  $PM_{2.5}$ ), the  $PM_{2.5}$  data tends to carry more weight in determining the potential for health risks because of the large body of scientific literature characterizing both the epidemiological and toxicological properties of the finer size fraction. Refer to the US EPA *Quantitative Health Risk Assessment for Particulate Matter* (US EPA, 2010) for further discussion.

## 5.0 HAZARD ASSESSMENT

The following section provides the acute and chronic inhalation TRV for each COC evaluated in the current assessment. For the purposes of the current screening level risk assessment, the most conservative TRV from a variety of credible regulatory agencies was selected for each COC and averaging period.

It should be noted that there are National Ambient Air Quality Objectives (NAAQOs) established by Health Canada and provincial Ambient Air Quality Criteria (AAQCs) established by the Ontario Ministry of the Environment under O. Reg. 419/05 for a number of the COCs. While still in regulatory force both provincially and federally, these benchmarks are often dated (*i.e.*, NO<sub>2</sub>, SO<sub>2</sub>) and may not represent the most recent scientific or regulatory knowledge on health effects related to exposures to these COCs.

### 5.1 Acute Inhalation Toxicity Reference Values

The acute (*i.e.*, for 1-hour and 24-hour exposure durations) non-carcinogenic inhalation TRVs for each COC, as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 4-1.

<b>Table 5-1 Summary of Selected Acute Non-carcinogenic Inhalation TRVs</b>						
COC	<i>Acute Non-Carcinogenic Inhalation TRVs (µg/m<sup>3</sup>)</i>					
	1-hour			24-hour		
	Value	Critical Effect	Source	Value	Critical Effect	Source
NO <sub>2</sub>	200	Effects in the pulmonary function of asthmatics	WHO, 2005	200	Respiratory irritant	Health Canada, 2006b
PM <sub>10</sub>	NV	-	-	50	Total, cardiopulmonary and lung cancer mortality increased	WHO, 2005
PM <sub>2.5</sub>	NV	-	-	25	Lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase	WHO, 2005
Silica	NV	-	-	5	Silicosis	MOE, 2012

NV No acute regulatory toxicity reference values are available for that specific averaging time.

### 5.2 Chronic Inhalation Toxicity Reference Values

The chronic non-carcinogenic and carcinogenic inhalation TRVs for each COC (where they were available), as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 4-2.

<b>Table 5-2 Summary of Chronic Non-carcinogenic and Carcinogenic Inhalation TRVs</b>						
COC	<i>Chronic Toxicity Reference Values</i>					
	<i>Non-Carcinogenic Inhalation TRVs (µg/m<sup>3</sup>)</i>			<i>Carcinogenic Inhalation Unit Risk Values (µg/m<sup>3</sup>)<sup>-1</sup></i>		
	Value	Critical Outcome	Source	Value	Critical Outcome	Source
PM <sub>2.5</sub> <sup>a</sup>	8.8	Not provided	CCME, 2012	NC	-	-
Silica	3	Silicosis	OEHHA, 2005	NC	-	-
Benzo[a]pyrene (PAH-surrogate)	NV	-	-	1.1 x 10 <sup>-3</sup>	Respiratory tract tumor	Cal EPA, 2009

NC This chemical is not considered to be a carcinogen, or no inhalation unit risk value is available.

NV No value. No chronic TRVs are available for this COC.

<sup>a</sup> Proposed annual CAAQS for PM<sub>2.5</sub>, based on a 3-year average of the annual average concentrations, intended to be enforceable in 2020.

It should be noted that some regulatory jurisdictions (such as the California EPA) consider diesel particulate to be carcinogenic. However, a more recent scientific review by the US EPA (in 2002) disagreed with Cal EPA (1998), and concluded that while diesel exhaust is “likely to be carcinogenic in humans by inhalation” at environmental or higher exposure conditions, due to the uncertainty in available exposure-response data, a cancer unit risk/cancer potency for diesel exhaust could not be derived (US EPA, 2002).

For purpose of the current assessment, PM (*i.e.*, PM<sub>10</sub> and PM<sub>2.5</sub>), in itself, is not considered to be carcinogenic. Rather, the potential carcinogenicity of the diesel particulate mixture is a result of the carcinogenic nature of various chemicals adsorbed to the surface of the particulate, with the PM (such as PM<sub>2.5</sub>) being the delivery vehicle by which these carcinogenic chemicals are carried deep into an individual’s lungs. For the current assessment, the PAH group were considered to be carcinogenic, and the implications of exposure to these diesel particulate contaminants to the surrounding community have been evaluated.

### 5.3 Ultrafine Particulate Matter

One area of ongoing research is into the health implications of ultrafine particulate matter (UFP). UFP constitute particulate matter smaller than 0.1 microns (or 100 nanometres) in size (*i.e.*, PM<sub>0.1</sub>). Due to their small size, UFPs are considered to be respirable particles and are able to travel deep within the lung with the potential to penetrate tissue and undergo interstitialization and therefore are not easily removed from the body. These smaller particles also have proportionally greater surface area per mass than larger particles, which can interact more readily with cell surfaces and can adsorb more of other chemicals, which in turn can have toxicological impacts on cells.

Like PM<sub>2.5</sub>, UFP can also lead to cardiopulmonary and respiratory disease, as well as stimulate immune and non-immune pathogenic process, oxidative stress, inflammatory mediator release, and other systemic effects (Oberdörster, 2000).

Currently there are no established regulatory benchmarks or standardized approaches to evaluation of the health impact related to exposures to this particulate matter fraction. However, it is important to note that the UFP fraction is collected in monitoring equipment as a subset of the PM<sub>2.5</sub> fraction, and as such the health effects arising from exposure to UFPs are inherently accounted for in the epidemiological studies used to establish health-based regulatory benchmarks for PM<sub>2.5</sub>. Both *in vivo* and *in vitro* studies of various UFP species are currently ongoing in a variety of animal models to better establish the toxicological profiles necessary for the establishment of regulatory benchmarks used in risk assessment (refer to the nanotechnology and ultrafine particle research provided by the US EPA National Center for Environmental Research at [http://www.epa.gov/ncercqa/nano/research/particle\\_index.html](http://www.epa.gov/ncercqa/nano/research/particle_index.html) for further information).

For the current assessment, the UFP fraction was considered as part of the evaluation of health impacts related to the PM<sub>2.5</sub>.



## **6.0 RISK CHARACTERIZATION**

The following section provides the results of the acute and chronic assessment of risks related to emissions from the proposed Project for all evaluated exposure scenarios, at each receptor location.

As noted previously, potential acute human health inhalation risks were evaluated for both 1-hour and 24-hour exposure periods for individuals living, working or playing in the area directly around the Project site, under both the Central Processing Plant and Auxiliary Processing Plant scenarios.

### **6.1 Acute Inhalation Assessment Results**

#### **6.1.1 Acute (1- and 24-Hour) Exposures**

Tables 6-1 and 6-2 provide summaries of the maximum predicted acute 1- and 24-hour inhalation risks for each COC at the sensitive receptor locations evaluated around the proposed Project for the Central Processing Plant and Auxiliary Processing Plant scenarios, respectively.

##### **6.1.1.1 Central Processing Plant Scenarios**

Results of the inhalation assessment of the Central Processing Plant scenarios for the 1-hour exposure period indicated that worst-case 1-hour NO<sub>x</sub> emissions related to the Project itself exceeded the regulatory benchmark at all receptor locations except CRAND6 and CHURCH12. In most cases, the contribution very marginally exceeded the benchmark. Accordingly, the addition of the contribution from the other nearby operating facilities and regional background resulted in exceedances of the 1-hour NO<sub>x</sub> regulatory benchmark for all receptor locations except CRAND6.

Results of the inhalation assessment of the Central Processing Plant scenarios for the 24-hour exposure period indicated that none of worst-case 24-hour NO<sub>x</sub>, PM<sub>10</sub>, PM<sub>2.5</sub>, or silica emissions related to the Project itself exceeded the regulatory benchmark at any of the sensitive receptor. In the case of NO<sub>x</sub>, none of the cumulative exposures exceeded the 24-hour regulatory benchmark as well.

For PM<sub>10</sub> and PM<sub>2.5</sub>, existing regional background concentrations resulted in exceedances (or near exceedances) of the 24-hour regulatory PM benchmarks for all of the sensitive receptor locations. In all cases, the contribution from the proposed Henning Pit was minor to negligible depending on the location of the particular receptor under assessment.

Finally, in the case of silica, existing regional background concentrations and worst-case 24-hour contributions from the other existing operating facilities resulted in exceedances of the 24-hour regulatory benchmark for silica at all of the sensitive receptor locations.

##### **6.1.1.2 Auxiliary Processing Plant Scenarios**

Results of the inhalation assessment of the Auxiliary Processing Plant scenarios for the 1-hour exposure period indicated that none of the worst-case 1-hour NO<sub>x</sub> emissions related to the Project itself exceeded the regulatory benchmark at any of the sensitive receptor locations. However, the addition of the contribution from the other nearby operating facilities and regional

background did result in predicted exceedances of the 1-hour NO<sub>x</sub> regulatory benchmark for all receptor locations except CRAND6.

Results of the inhalation assessment of the Auxiliary Processing Plant scenarios for the 24-hour exposure period indicated that none of worst-case 24-hour NO<sub>x</sub>, PM<sub>10</sub>, PM<sub>2.5</sub>, or silica emissions related to the Project itself exceeded the regulatory benchmark at any of the sensitive receptor. In the case of NO<sub>x</sub>, none of the cumulative exposures exceeded the 24-hour regulatory benchmark as well.

For PM<sub>10</sub> and PM<sub>2.5</sub>, existing regional background concentrations resulted in exceedances (or near exceedances) of the 24-hour regulatory PM benchmarks for all of the sensitive receptor locations. In all cases, the contribution from the proposed Henning Pit was minor to negligible depending on the location of the particular receptor under assessment.

Finally, in the case of silica, existing regional background concentrations and worst-case 24-hour contributions from the other existing operating facilities resulted in exceedances of the 24-hour regulatory benchmark for silica at all of the sensitive receptor locations.

**Table 6-1 Worst-Case Predicted Acute Concentration Ratios (CR) at each Sensitive Receptor Locations for the Central Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>1-hour Exposure Period</b>															
NO <sub>x</sub>	0.18	<b>1.3</b>	<b>1.7</b>	<b>1.8</b>	<b>2.0</b>	<1	<1	<b>1.1</b>	<b>1.3</b>	1.0	<b>3.0</b>	<b>1.1</b>	<b>1.5</b>	<b>1.2</b>	<b>1.4</b>
<b>24-hour Exposure Period</b>															
NO <sub>x</sub>	0.18	0.13	0.37	0.18	0.38	0.099	0.28	0.11	0.34	0.13	0.44	0.13	0.32	0.12	0.36
PM10	0.78	0.29	<b>1.5</b>	0.38	<b>1.5</b>	0.29	<b>1.3</b>	0.32	<b>2.5</b>	0.29	<b>1.3</b>	0.18	<b>1.1</b>	0.34	<b>2.7</b>
PM2.5	0.84	0.11	1.0	0.14	<b>1.1</b>	0.096	0.99	0.12	<b>1.2</b>	0.10	<b>1.0</b>	0.060	0.96	0.14	<b>1.3</b>
Silica	0.47	0.58	<b>1.8</b>	0.76	<b>1.9</b>	0.58	<b>1.5</b>	0.64	<b>3.8</b>	0.60	<b>1.5</b>	0.36	<b>1.2</b>	0.68	<b>4.2</b>

Note: All values are presented as Concentration Ratios (CRs) where the predicted ground-level air concentrations have been divided by the regulatory benchmark for that COC relevant to the appropriate exposure period. **Bolded** and highlighted cells represent predicted ground-level air concentrations which exceed the relevant regulatory benchmark. 1-hour NO<sub>x</sub> CR values shown as "<1" indicate air concentrations modelled by RWDI were below the 200 µg/m<sup>3</sup> threshold and were not retained individually in the subsequent model output. As such, better precision as to the predicted concentration and related CR value is unavailable at this time.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

**Table 6-2 Worst-Case Predicted Acute Concentration Ratios (CR) at each Sensitive Receptor Locations for the Auxiliary Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>1-hour Exposure Period</b>															
NO <sub>x</sub>	0.18	<1	<b>1.1</b>	<1	<b>1.1</b>	<1	0.77	<1	<b>1.2</b>	<1	<b>3.0</b>	<1	<b>1.5</b>	<1	<b>1.4</b>
<b>24-hour Exposure Period</b>															
NO <sub>x</sub>	0.18	0.080	0.30	0.14	0.33	0.041	0.24	0.029	0.34	0.11	0.44	0.074	0.26	0.032	0.36
PM10	0.78	0.19	<b>1.3</b>	0.40	<b>1.5</b>	0.17	<b>1.2</b>	0.25	<b>2.5</b>	0.27	<b>1.3</b>	0.16	<b>1.1</b>	0.27	<b>2.7</b>
PM2.5	0.84	0.044	0.97	0.088	1.0	0.039	0.93	0.056	<b>1.2</b>	0.064	1.0	0.036	0.93	0.064	<b>1.3</b>
Silica	0.47	0.38	<b>1.6</b>	0.80	<b>1.8</b>	0.34	<b>1.2</b>	0.50	<b>3.8</b>	0.54	<b>1.5</b>	0.32	<b>1.1</b>	0.54	<b>4.2</b>

Note: All values are presented as Concentration Ratios (CRs) where the predicted ground-level air concentrations have been divided by the regulatory benchmark for that COC relevant to the appropriate exposure period. **Bolded** and highlighted cells represent predicted ground-level air concentrations which exceed the relevant regulatory benchmark.

1-hour NO<sub>x</sub> CR values shown as "<1" indicate air concentrations modelled by RWDI were below the 200 µg/m<sup>3</sup> threshold and were not retained individually in the subsequent model output. As such, better precision as to the predicted concentration and related CR value is unavailable at this time.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

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## **6.2 Chronic Inhalation Assessment Results**

### **6.2.1 Non-Carcinogenic Risks**

Table 6-3 and 6-4 provide summaries of the maximum predicted chronic inhalation risks for PM<sub>2.5</sub> and silica at the sensitive receptor locations evaluated around the proposed Project for the Central Processing Plant and Auxiliary Processing Plant scenarios, respectively.

Results of the inhalation assessment indicate that all predicted Project Only (*i.e.*, Henning Pit) and cumulative exposures were below the annual chronic regulatory benchmark for PM<sub>2.5</sub> and silica at all sensitive receptor locations. It should be noted that the PM<sub>2.5</sub> benchmark is intended to be compared to a 3-year average of the predicted annual average concentrations (CCME, 2012). For the current assessment, the worst-case 1-year annual average of PM<sub>2.5</sub> concentrations was conservatively used.

**Table 6-3 Worst-Case Predicted Chronic Concentration Ratios (CR) at each Sensitive Receptor Locations for the Central Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>Annual Average Exposure Period</b>															
PM2.5	0.91	0.0091	0.92	0.020	0.94	0.0068	0.92	0.0048	0.97	0.0091	0.94	0.0060	0.92	0.0055	0.98
Silica	0.32	0.025	0.37	0.051	0.40	0.019	0.36	0.015	0.59	0.025	0.43	0.018	0.37	0.017	0.61

Note: All values are presented as Concentration Ratios (CRs) where the predicted ground-level air concentrations have been divided by the regulatory benchmark for that COC relevant to the appropriate exposure period.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

**Table 6-4 Worst-Case Predicted Chronic Concentration Ratios (CR) at each Sensitive Receptor Locations for the Auxiliary Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>Annual Average Exposure Period</b>															
PM2.5	0.91	0.0063	0.92	0.014	0.94	0.0034	0.92	0.0028	0.97	0.0065	0.94	0.0048	0.92	0.0034	0.98
Silica	0.32	0.017	0.36	0.052	0.40	0.012	0.36	0.013	0.59	0.028	0.42	0.017	0.37	0.015	0.61

Note: All values are presented as Concentration Ratios (CRs) where the predicted ground-level air concentrations have been divided by the regulatory benchmark for that COC relevant to the appropriate exposure period.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

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### 6.2.2 Carcinogenic Risks

Table 6-5 and 6-6 provide summaries of the maximum predicted chronic inhalation incremental lifetime cancer risks (ILCR) for the PAH group (represented by benzo[a]pyrene as a surrogate) at the sensitive receptor locations evaluated around the proposed Project for the Central Processing Plant and Auxiliary Processing Plant scenarios, respectively.

Results of the assessment of ILCR risks for PAH inhalation exposures indicate that all predicted Project Only (*i.e.*, Henning Pit) were significantly below the one-in-one-million ( $1.0 \times 10^{-6}$ ) ILCR regulatory benchmark at all sensitive receptor locations.

For the purpose of evaluating carcinogenic risks related to an airborne emission source as part of an Environmental Assessment (EA) or related process, the evaluation of incremental lifetime cancer risks *versus* the Ontario regulatory benchmark of acceptable incremental cancer risk (*i.e.*, one-in-one-million or  $1 \times 10^{-6}$ ) is done by calculating the incremental increase in lifetime cancer risk related to emissions predicted for the proposed Project **above** those predicted for existing background conditions (*i.e.*, the incremental lifetime cancer risks related to addition of the proposed Henning Pit *versus* status quo with existing background conditions including pre-existing operating pits in the area).

However, results of the assessment of ILCR risks for PAH inhalation exposures indicate that all predicted cumulative risks (*i.e.*, Henning Pit contribution plus existing pits and regional background) were still significantly below the one-in-one-million ( $1.0 \times 10^{-6}$ ) ILCR regulatory benchmark at all sensitive receptor locations.

**Table 6-5 Worst-Case Predicted Chronic Incremental Lifetime Cancer Risk (ILCR) at each Sensitive Receptor Locations for the Central Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>Annual Average Exposure Period</b>															
PAHs (B[a]P surrogate)	1.7E-08	9.1E-10	1.9E-08	1.7E-09	2.0E-08	8.4E-10	1.9E-08	6.6E-10	2.4E-08	8.9E-10	2.2E-08	7.7E-10	1.9E-08	7.5E-10	2.4E-08

Note: All values are presented as incremental lifetime cancer risk (ILCR) predictions where the predicted ground-level air concentrations have been multiplied by the regulatory inhalation cancer unit risk value for benzo[a]pyrene. Predicted ILCR values exceeding 1.0E-6 (i.e., one-in-one-million) have the potential for unacceptable cancer risk over a lifetime of exposures.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

**Table 6-6 Worst-Case Predicted Chronic Incremental Lifetime Cancer Risk (ILCR) at each Sensitive Receptor Locations for the Auxiliary Processing Plant Scenarios**

COCs	Regional Background	Sensitive Receptor Locations													
		CRAND4		CRAND5		CRAND6		CRAND15		CHURCH2		RENTAL3		PAULCAB14	
		Henning Pit Only	Cumulative <sup>a</sup>	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative	Henning Pit Only	Cumulative
<b>Annual Average Exposure Period</b>															
PAHs (B[a]P surrogate)	1.7E-08	2.2E-09	2.0E-08	4.3E-09	2.3E-08	1.4E-09	1.9E-08	6.8E-10	2.4E-08	2.0E-09	2.3E-08	1.6E-09	2.0E-08	8.0E-10	2.4E-08

Note: All values are presented as incremental lifetime cancer risk (ILCR) predictions where the predicted ground-level air concentrations have been multiplied by the regulatory inhalation cancer unit risk value for benzo[a]pyrene. Predicted ILCR values exceeding 1.0E-6 (i.e., one-in-one-million) have the potential for unacceptable cancer risk over a lifetime of exposures.

<sup>a</sup> Cumulative concentrations include modelled Henning Pit contributions, as well as the estimated contributions from both the St. Mary and Lafarge active pits, truck traffic on the surrounding arterial roads, as well as regional background.

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## 7.0 UNCERTAINTY ANALYSIS

In any HHRA, screening level or detailed, the intention is to obtain the most accurate evaluation of risk based upon the available data and state of knowledge, without underestimating the potential health risks. With any such assessment, there are always a number of administrative and technical boundaries that limit the ability of the assessment to quantify risk with absolute certainty. The following section provides an overview of the key administrative and technical boundaries inherent within the current assessment.

Quantitative human health assessments involve assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing chemical concentrations in environmental media, chemical fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk. The US EPA (2000) advocates that the risk characterization process maintain transparency, clarity, consistency, and reasonableness. The goal of risk characterization is to clearly communicate the key findings of the assessment and to provide a clear and balanced assessment of the strengths and limitations of the process. Risk characterization involves both scientific and policy based decision making, thereby resulting in a decision making process that blends both elements.

When assumptions are made during the risk assessment process, either because of data gaps or knowledge gaps, each can result in some degree of uncertainty in the overall conclusions. In order to understand the uncertainties within the assessment and to ensure that the implications of these uncertainties are understood and addressed, it is important to document and characterize them. To ensure that the risk assessment does not underestimate the potential for the occurrence of adverse effects, it is necessary to make assumptions that are conservative (protective). In other words, assumptions should be made that tend to overestimate exposure, toxicity and risk, rather than underestimate these parameters.

The following sections describe uncertainty within the current assessment, and discuss the potential impacts of these limitations on the conclusions drawn from the assessment. Given the tendency for the assumptions described below to overestimate both exposure and toxicity, it is likely that the risk characterization errs on the side of caution and over predicts risk. A summary of the conservative assumptions that were incorporated into the assessment can be found in Table 7-1, arranged according to the steps of the risk assessment paradigm. Examination of the table shows that conservatism was introduced at virtually every step of the assessment, and extended to both the exposure and toxicity assessment of the overall human health assessment.

Furthermore, refer to the Air Quality Assessment report (RWDI, 2013) for a detailed discussion of the uncertainty and conservatism built into the assumptions used to predict ground-level air concentrations for each of the COCs.



**Table 7-1 Major Assumptions Used in the Current Assessment**

<b>Risk Assessment Paradigm</b>	<b>Assumption</b>	<b>Discussion of Conservatism</b>
Problem Formulation	Seven (7) discrete receptor locations were selected in and around the proposed Project site.	Care was taken to select residential locations around the Project site that would likely demonstrate the highest potential impacts from project site and related traffic emissions.
	Chemical characterization and selection of COC were based upon the primary contaminants of importance in dust and diesel emissions.	The key chemicals with respect to diesel emissions from the perspective of either regulatory or public health were selected as COC for the current assessment. Diesel particulate emissions include a large number of different chemicals. While the selected COC typically represent the most toxic of the chemicals in this mixture, the current assessment does not attempt to evaluate all of the contaminants emitted from diesel vehicles. In addition to the particulates (PM <sub>2.5</sub> and PM <sub>10</sub> ), NOx and the PAHs were used as worst-case surrogates for all the relevant diesel emissions.
	Measured background air quality is intended to provide an indication of air quality in areas similar to the Project Area, and as such may not be exactly representative of baseline air quality surrounding the site.	An appropriate background concentration for dust particulate and diesel-related COC for the study area was prepared using ambient air quality data from a number of federal and provincial monitoring stations near the study area. A number of these monitoring stations lie some distance from the site, itself.
Exposure Assessment	Te assessment relied on hypothetical estimates of ground level air quality concentrations that describe effects of project activities on-site and off-site emissions from roadway use, and are predicted by air dispersion models.	The SLHHRA relied on the results of air dispersion modelling to evaluate the health risks from direct inhalation exposure as well as to predict inhalation health risks. The MOE has discussed matters of confidence and uncertainty in the predictions of dispersion models with regard to ground level concentrations and deposition rates. This remains the best mechanism to forecast future distributions of emissions in built environments. The air dispersion models used to provide data for the current assessment are approved by the MOE and the US EPA for use on these types of emission studies.  Refer to the Air Quality Impact Assessment study for further discussion of this uncertainty (RWDI, 2013).
	Maximum predicted short term ( <i>i.e.</i> , for 1-hour and 24-hour exposure durations) ground level air concentrations at each receptor location were used to evaluate all acute inhalation risk estimates.	In reality, the frequency with which the maximum would occur at any one receptor location varies with respect to the COC and the receptor location. Individual exposure to a 1-hour or 24-hour maximum ground-level air concentration requires that a receptor (person) be present at the same time and duration of the maximum predicted air concentration anywhere in the area around the site.
	Silica concentrations were conservatively estimated based on predicted worst-case particulate concentrations and a blend of expected crystalline silica concentrations present in both the ore and from ground road materials.	Predicted silica concentrations are likely over-estimated given the conservative assumptions built into the modelling conducted by the Air Quality team.

**Table 7-1 Major Assumptions Used in the Current Assessment**

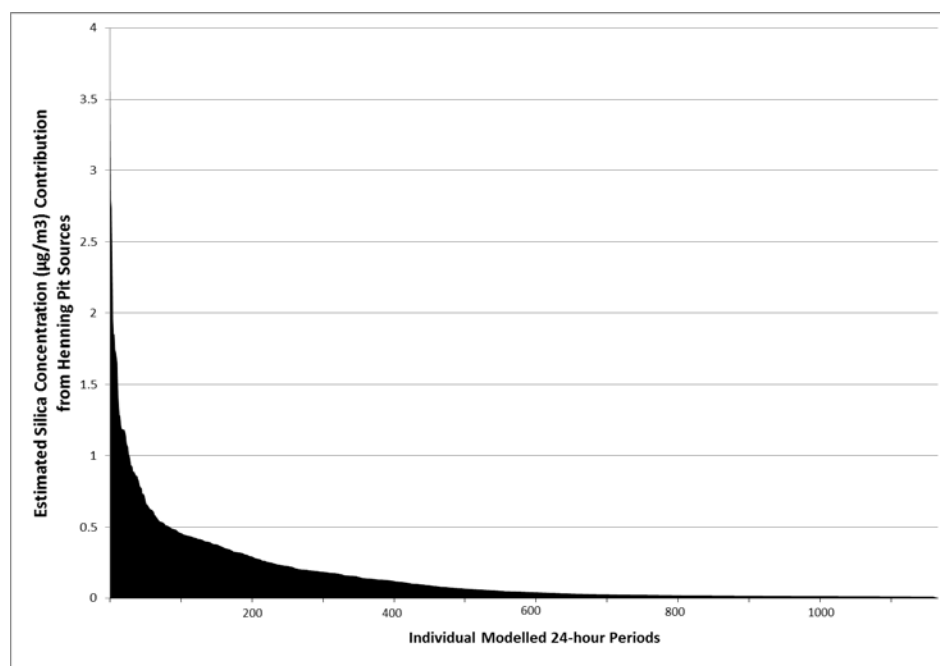
<b>Risk Assessment Paradigm</b>	<b>Assumption</b>	<b>Discussion of Conservatism</b>
Exposure Assessment (continued)	Air quality monitoring was conducted assuming all processing plants (e.g., either central or auxiliary) were continuously working at full capacity.	This assumption likely greatly over-estimates potential exposures, as throughput of the processing plants – in particular the Auxiliary processing plant as it moves around the property – is expected fluctuate depending on daily activities and business requirements. In fact, it is expected that the Auxiliary processing plant will only operate at half the capacity modelled in the Air Quality assessment.
	Predicted ground-level air concentrations assume the presence of ongoing dust mitigation measures.	Ground-level air concentrations of particulate-bound COCs will be highly dependent on the success of proscribed dust mitigation measures outlined in the Air Quality Assessment report.
Toxicity Assessment	Toxicity reference values (TRVs) have been developed by regulatory agencies with sufficient conservatism assure protection of the sensitive and more susceptible individuals within the general population (e.g., infants and young children, the elderly, individuals with compromised health).	A considerable amount of conservatism is incorporated in the TRVs. These benchmarks are deliberately set by regulatory agencies with the protection of sensitive individuals in mind. Typically, the benchmarks used in the current assessment were derived from the most sensitive health-related endpoints, and then adjusted to account for differences in sensitivity to chemicals among individuals. The use of uncertainty factors is directed, in part, toward the protection of sensitive individuals.  The most sensitive toxicological endpoint (for example, decreased growth, body weight loss/gain, reproductive effects) was selected for each chemical from the available scientific literature to represent the exposure limit (TRV).
	All emitted oxides of nitrogen was considered to be in the form of the most toxicologically potent NO <sub>2</sub> .	It is important to note that while NO <sub>x</sub> is emitted from a diesel vehicle or generator, nitrogen dioxide (NO <sub>2</sub> ) is the most toxicological significant of the oxides of nitrogen. As NO (and other lesser oxides of nitrogen) is considerably less toxic than NO <sub>2</sub> , one is over-estimating potential health risk if one conservatively assumes all NO <sub>x</sub> is in the form of NO <sub>2</sub> , as we done in the current assessment.
	For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions.	The existence of enzymes and biological pathways that routinely repair damage to genetic material (DNA) is well documented in the scientific literature. The potential adverse health outcomes arising from damage to DNA is usually observed only when the ability of these repair enzymes to "fix" the damage is blocked or exceeded.
	Uncertainty factors were applied at exposure levels reported in animal or human studies where no adverse effects were observed (i.e., NOAEL). Thus, exceeding the toxicological criterion should not mean that adverse health outcomes would occur. Rather, it means that the uncertainty factor beyond the no-effect exposure is somewhat reduced.	Large uncertainty factors (i.e., 100-fold or greater) were used in the estimation of the TRVs for threshold type chemicals.
	Humans were assumed to be the most sensitive species with respect to toxic effects of the COC.	For obvious reasons, toxicity assays are not generally conducted on humans, so toxicological data from the most sensitive laboratory species were used in the estimation of toxicological criteria for humans.

## 8.0 DISCUSSION OF RESULTS

Based on the results of the assessment, worst-case contributions from the proposed Henning Pit facility do not exceed the relevant regulatory benchmarks in any scenario, with the exception of minor 1-hour NO<sub>x</sub> exceedances in the Central Processing Plant scenario at most of the receptor locations.

However, it should be noted that the current assessment conservatively assumes that 100% emitted oxides of nitrogen are inter-converted to NO<sub>2</sub> (the most toxic of the oxides of nitrogen). This assumption greatly over-estimates predicted concentrations of NO<sub>2</sub> as a result of emissions from diesel engines. Under typical conditions in the presence of ozone, only approximately 30% of NO (and other lesser oxides of nitrogen) are inter-converted to NO<sub>2</sub> (see discussion in Section 3.2.3.3). Even if one assumed 50% of the oxides of nitrogen are converted to NO<sub>2</sub>, ambient concentrations at the receptor locations arising from Project emissions would not exceed the relevant acute benchmark.

It is also important to remember that the assessed concentrations represent the worst-case maximum concentration predicted by the Air Quality assessment. This worst-case concentration prediction is a result of the aligning of worst-case operations, meteorological, and situational conditions. For example, the maximum predicted concentration of 24-hour silica exposures at the worst-case sensitive receptor location (CRAND5) arising from Project emissions was 3.8 µg/m<sup>3</sup>. However, if one reviews the predicted daily concentrations of silica at the CRAND5 sensitive receptor location over the five years modelled by the Air Quality assessment arising from the proposed Project (see Figure 8-1), only 2.3% of the days have 24-hour silica concentrations that exceed even 1 µg/m<sup>3</sup> and less than 0.35% of the days exceed the 2 µg/m<sup>3</sup> threshold for silica. Given the 24-hour health-based AAQC for silica is 5 µg/m<sup>3</sup>, contributions to ambient concentrations of silica from the proposed Project at the sensitive receptor locations are well below levels which would be of concern to human health (*i.e.*, silicosis).



**Figure 8-1 Estimated 24-Hour Silica Concentrations at CRAND5 from Henning Pit Sources over Five Years**

The same pattern is observed for all of the other COCs evaluated in the current assessment. For 24-hour  $PM_{2.5}$  at the worst-case sensitive receptor location, the maximum predicted concentration arising from Project sources is  $3.4 \mu\text{g}/\text{m}^3$ . However, if one reviews the predicted daily concentrations of  $PM_{2.5}$  at this worst-case sensitive receptor location over the five years modelled, only 1.5% of the days have 24-hour  $PM_{2.5}$  concentrations that exceed even  $1 \mu\text{g}/\text{m}^3$  and less than 0.35% of the days exceed the  $2 \mu\text{g}/\text{m}^3$  threshold. Again, with a regulatory benchmark of  $25 \mu\text{g}/\text{m}^3$ , the contributions of fine particulate from the proposed Project are typically orders of magnitude below levels that could potential cause health concerns in the surrounding population.

Given the hourly and daily variability in COC concentrations, and the multitude of conservative assumptions used within the Air Quality Assessment, one would not expect that the predicted emissions from the proposed Project would result in any elevated unacceptable health concerns.

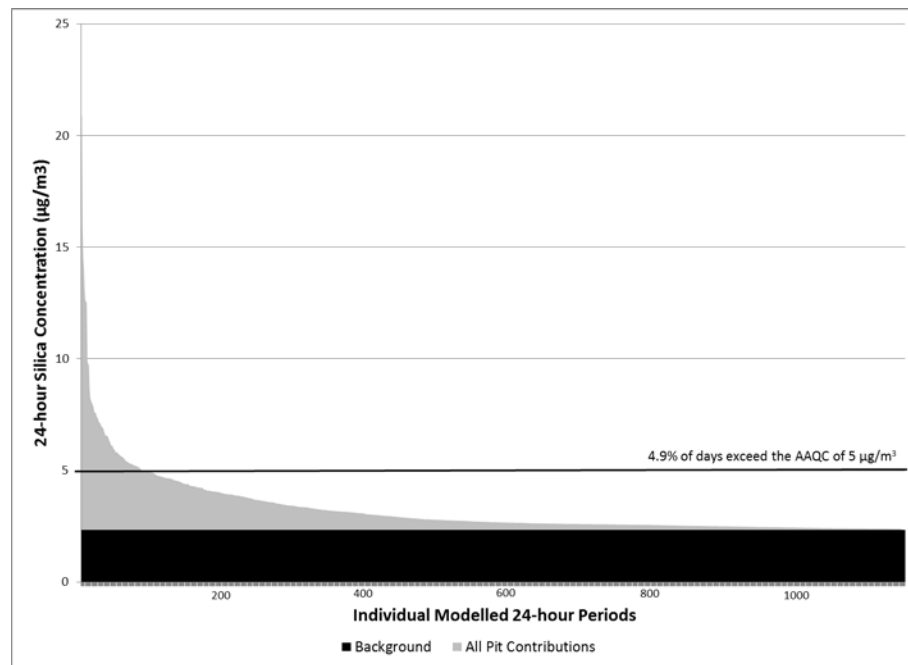
The Air Quality Assessment also predicted cumulative concentrations for each of the COCs assuming the contributions for the proposed Project are added to already existing contributions from the other existing pit facilities, as well as regional background concentrations. The cumulative assessment did indicate that the worst-case exposure conditions could result in ambient concentrations that exceeded the respective regulatory benchmark for some of the COCs. However, in each of these cases, the contribution from the proposed Project was minimal to negligible compared to already existing background sources.

Furthermore, like the pattern observed for emissions from the proposed Project, cumulative concentrations also varied greatly on an hourly and daily basis, depending on a variety of operational, meteorological, and situational conditions.

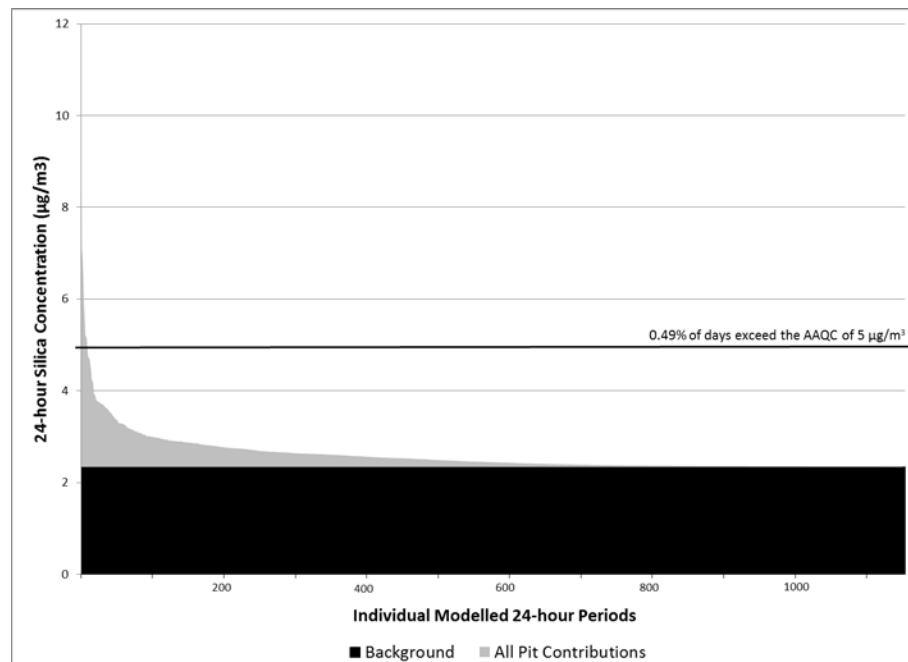
Using 24-hour silica concentrations as an example again, the maximum predicted **cumulative** concentration of 24-hour silica exposures at the worst-case sensitive receptor location for cumulative exposures (PAULCAB4) was  $21.2 \mu\text{g}/\text{m}^3$ . Interestingly, the contribution of silica exposure from the proposed Project on that particular modelled day was  $0.079 \mu\text{g}/\text{m}^3$ , with the other existing pits (*i.e.*, St. Mary's and Lafarge pits) and related road traffic providing nearly all of the estimated silica concentrations on this worst-case day. This discrepancy is due to the location of the PAULCAB4 receptor in relation to both the proposed Project site and the adjacent pit operations, specifically the Lafarge facility.

If one reviews the predicted daily **cumulative** concentrations of silica at the PAULCAB4 sensitive receptor location over the five years modelled by the Air Quality assessment (see Figure 8-2), one again sees a similar pattern as observed in the Project Only emission scenarios. In this case, less than 5% of the days have 24-hour silica concentrations from cumulative sources that exceed the  $5 \mu\text{g}/\text{m}^3$  regulatory benchmark.

A similar review of the predicted daily **cumulative** concentrations of silica at the CRAND5 receptor (*i.e.*, the worst-case location for emissions from the proposed Project) shows that the maximum cumulative exposure was  $9.7 \mu\text{g}/\text{m}^3$  of which only the  $3.8 \mu\text{g}/\text{m}^3$  of silica noted previously was related to emissions from the proposed Project. Figure 8-3 shows the rapid decline in ambient daily silica concentrations around CRAND5, with less than 0.5% of the days have 24-hour silica concentrations from cumulative sources that exceeded the  $5 \mu\text{g}/\text{m}^3$  regulatory benchmark.



**Figure 8-2 Estimated 24-Hour Silica Concentrations at PAULCAB4 from Cumulative Sources over Five Years**



**Figure 8-3 Estimated 24-Hour Silica Concentrations at CRAND5 from Cumulative Sources over Five Years**

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As noted previously, chronic silicosis usually develops after 10 or more years of prolonged and consistent inhalation exposure to crystalline silica. The 24-hour regulatory benchmark of  $5 \mu\text{g}/\text{m}^3$  is intended to provide a threshold with a built-in margin of safety to prevent the potential for developing chronic silicosis over an extended period of continuous exposure. Given exceedances of the  $5 \mu\text{g}/\text{m}^3$  threshold are predicted to be a very infrequent event even under worst-case conditions, and the degree of conservatism built into the assumptions used to predict silica concentrations in the Air Quality Assessment, it is not expected that current emissions would result in the potential to develop silicosis in individuals living around the proposed Project site.

This conclusion is further confirmed when one compares predicted annual exposures of silica at each of the sensitive receptor locations with the chronic regulatory benchmark for silica. In this case, none of the predicted Project Only or cumulative concentrations of silica over a long-term basis exceeds the health-based regulatory benchmark protective of silicosis, at any of the sensitive receptor locations. As such, though occasional short-term elevated concentrations of silica could be observed around the proposed Project (largely due to existing background and extraction activities), ambient concentrations over the longer term period are unlikely to reach levels that would result in potential health concerns related to silica exposures.

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## 9.0 CONCLUSIONS AND RECOMMENDATIONS

Based on the results of the assessment, and given the considerable conservatism built into the Air Quality Assessment, no unacceptable health risks related to emissions from the proposed Project would be expected. In fact, estimated emissions from the proposed Project typically represent a minimal to negligible component of the overall cumulative exposures for each of the COCs predicted for the sensitive receptor locations around the proposed Project site. Furthermore, results of the assessment also indicate that cumulative exposures to the evaluated chemicals are not expected to result in unacceptable health risks in the surrounding community given the degree of conservatism built into the assessment assumptions and scenarios.

However, to ensure ambient dust concentrations are kept at a minimal level, it is recommended that the dust suppression and related mitigation measures outlined in the Air Quality Assessment report be considered and implemented for the proposed Project where feasible.

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