

SENES Consultants Limited

MEMORANDUM



121 Granton Drive, Unit 12
Richmond Hill, Ontario
Canada L4B 3N4
Tel: (905) 764-9380
Fax: (905) 764-9386
E-mail: senes@senes.ca
Web Site: <http://www.senes.ca>

TO: Bruce Halbert 350047-020

FROM: Stacey Fernandes, Harriet Phillips and Jennifer Kirkaldy 30 July 2012

SUBJ: Exposure to Members of the Public Using Ingraham Trail from Air Emissions During GMRP

This memo provides an assessment of the potential risk to human health of a member of the public that could walk or run along the Ingraham Trail during Giant Mine Remediation Project (GMRP) activities. While there is talk of relocating Ingraham Trail to pass around the site, it currently goes through the site and people can hike or bike along the road.

1.0 BACKGROUND

SENES completed a screening level air dispersion modelling assessment, which was summarized in the Developer's Assessment Report (DAR) for the GMRP Environmental Assessment. The screening level assessment determined that, based on a reasonable level of mitigation during remediation activities, wind blown dust would be the primary emission source of TSP and arsenic. The assessment assumed that emissions from vehicle traffic on unpaved roads during non-freezing times of the year can be effectively controlled through watering and the application of calcium chloride to reduce evaporation rates. Dust associated with bulldozing activities can also be controlled through watering. The screening level model results predicted arsenic, TSP, PM₁₀ and PM_{2.5} concentrations during GMRP activities that did not result in exceedances of any criteria at the nearest identified sensitive receptor locations for all particulate based contaminants assessed.

Subsequent to the screening level assessment, SENES was retained to complete dispersion modelling for GMRP activities with the CALMET/CALPUFF air dispersion modelling package. GMRP activities for this assessment are considered to include total suspended particulate (TSP), PM₁₀, PM_{2.5}, arsenic and combustion emissions (NO_x and SO₂) from GMRP activities in addition to projected worst case operations of the Jackfish Power Plant.

The air dispersion modelling covered the property as well as seven discrete receptors locations including:

R1	Yellowknife River Park
R2	N'Dilo Residential Receptor
R3	Back Bay Residential Receptor
R4	Boat Launch Recreational Receptor
R5	Municipal Landfill Receptor
R6	Niven Lake Residential Receptor

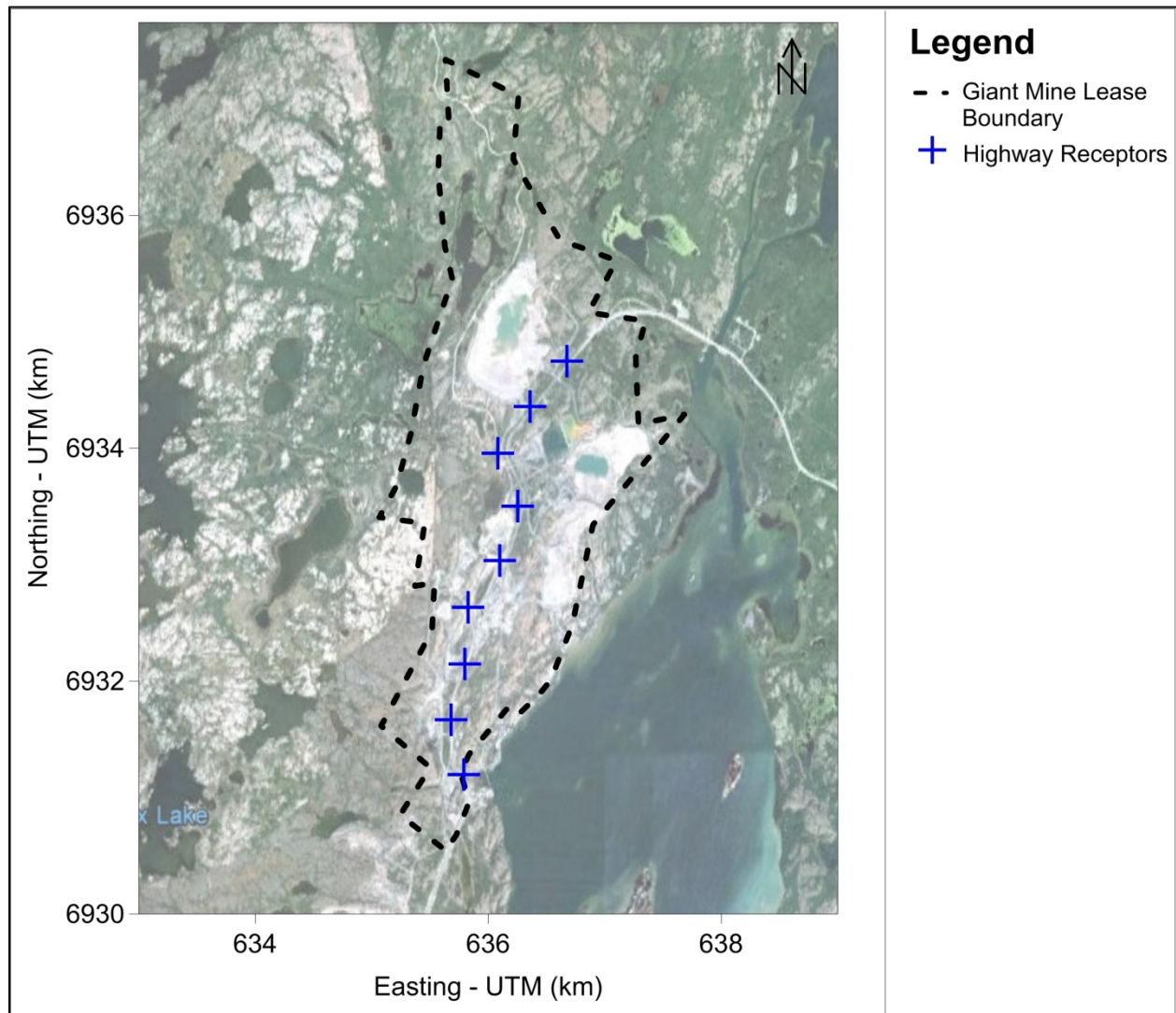
CALPUFF model results for GMRP activities were consistent with the screening level air dispersion modelling assessment. For the COPC that are influenced primarily from the GMRP activities (arsenic, TSP, PM₁₀ and PM_{2.5}), CALPUFF did not predict any exceedances of criteria at the nearest sensitive receptor locations.

As this assessment involved the evaluation of potential adverse effects to individuals who walk, run or bike along the Ingraham Trail, the CALPUFF model was rerun using the methodology described below. For the purposes of this evaluation it was assumed that someone could be present along the 4 km strip of road 2 hours a day. In other words someone walking 4 km/h would walk along the site and back in 2 hours.

2.0 METHODOLOGY FOR DEVELOPING AIR CONCENTRATIONS

Nine receptor locations along the Ingraham Trail within the Giant Mine site lease boundary were defined as illustrated in Figure 1. The maximum incremental one-hour average PM_{2.5} and arsenic concentrations from Giant Mine Remediation Project activities (including the Jackfish Power Plant producing 18 MW) were predicted at each of the nine receptors for each hour in the year. For each hour, the air concentrations were averaged across the nine receptors. The same emission rates, source configuration and model described in the CALPUFF Air Dispersion Modelling for the Giant Mine Remediation Project (SENES 2012), were used.

Figure 1
Receptor Locations along the Ingraham Trail



For $PM_{2.5}$, the health-based short-term benchmark is based on an incremental concentration on a 24-hour averaging period. As discussed previously, for the assessment of exposure of an individual on the Trail, it was assumed that the individual would be exposed up to a maximum of two-hours on any day. The maximum predicted consecutive two hour period in each day were used to derive an incremental 24 hour average exposure (assuming zero exposure for the remainder of the time) to an individual. Table 1 provides the maximum incremental $PM_{2.5}$ concentrations predicted by the model.

Table 1
Maximum Incremental 24 Hour Average PM_{2.5} Concentration due to Giant Mine Remediation Project Activities to an Individual Traversing the Site for 2 Hours

Frequency Distribution		Incremental Concentration ($\mu\text{g}/\text{m}^3$)
Maximum		5.00
Percentile	99.9	4.98
	99.5	4.66
	99	4.02
	98	2.87
	95	2.16
	90	1.56

Background PM_{2.5} concentrations in Yellowknife on average range from approximately 2 to 4 $\mu\text{g}/\text{m}^3$ depending on the year. However PM_{2.5} concentrations in the area can be substantially higher, in excess of 35 $\mu\text{g}/\text{m}^3$ (24-hour average), if there are forest fires in the region. This is particularly the case in dry summer months.

For arsenic, the maximum predicted incremental 1-hour average concentrations were calculated for the average of the same 9 receptor locations along the Highway traversing the site. The maximum incremental 1-hour average arsenic concentrations predicted by the model are provided in Table 2. The background arsenic concentrations are in the order of 0.004 $\mu\text{g}/\text{m}^3$ and thus do not add significantly to the predicted incremental concentrations.

Table 2
Maximum Incremental 1 Hour Average Arsenic Concentration due to Giant Mine Remediation Project Activities to an Individual Traversing the Site

Frequency Distribution		Incremental Concentration ($\mu\text{g}/\text{m}^3$)
Maximum		0.37
Percentile	99.9	0.33
	99.5	0.25
	99	0.18
	98	0.14
	95	0.099
	90	0.07

3.0 ASSESSMENT FOR ARSENIC

3.1 Selection of a Health-Based Benchmark

Inorganic arsenic dust can cause respiratory irritation and mucous membrane damage leading to rhinitis, pharyngitis or laryngitis (RAIS 1997). A review of the available information was conducted in order to determine an appropriate benchmark for assessing health impacts on a short-term (or acute) basis.

The Agency for Toxic Substances and Disease Registry (ATSDR) conducted a recent review of the overall database of literature for arsenic (ATSDR 2007). They report that: *Increased risk of lung cancer, respiratory irritation, nausea, skin effects, and neurological effects have been reported following inhalation exposure. There are only a few quantitative data on noncancer effects in humans exposed to inorganic arsenic by the inhalation route. Animal data similarly identify effects on the respiratory system as the primary noncancer effect of inhaled inorganic arsenic compounds, although only a few studies are available. Only limited data on the effects of inhaled organic arsenic compounds in humans or animals are available; these studies are generally limited to high-dose, short-term exposures, which result in frank effects.* (ATSDR 2007). They determined that the database of information was insufficient to develop an exposure limit.

Environment Canada (1999) indicates that the lowest reported no-observed-effect level (NOEL) following inhalation in limited available short-term and subchronic studies is $1.3 \mu\text{g}/\text{m}^3$ arsenic trioxide, based on the observation of slower conditioned reflexes and histological changes in the brain, liver, and lungs of rats at higher concentrations ($0.005 \text{ mg}/\text{m}^3$ or $5 \mu\text{g}/\text{m}^3$) (Rozenshtein 1970).

The California Environmental Protection Agency (CalEPA) has developed a 1-hour limit of $0.2 \mu\text{g}/\text{m}^3$ (CalEPA 2008) based on a short-term inhalation study with mice (Nagymajtenyi *et al.*, 1985). The ATSDR reviewed the study used by CalEPA and found that there were significant limitations in how the study was conducted (e.g. failure to quantify malformations on a litter basis, discuss the nature and severity of the observed malformations, or report on the occurrence of maternal effects). Therefore, this value should be used with caution.

The United States Environmental Protection Agency does develop Acute Exposure Guideline Levels (AEGLs) that are intended to describe the risk to humans resulting from once-in-a-lifetime, or rare, exposure to airborne chemicals. AEGL-2 is the airborne concentration of a substance above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape. There are interim AEGL-2 values for arsenic trioxide for several averaging times including $3,000 \mu\text{g}/\text{m}^3$ for a 1-h averaging period and $1,200 \mu\text{g}/\text{m}^3$ for an 8-h averaging period (U.S. EPA 2012). As the basis for these values are not available and the order

of magnitude is not consistent with other information these values are not used in the assessment.

ATSDR (2007) did state that: *Arsenic is a known human carcinogen by both the inhalation and oral exposure routes. By the inhalation route, the primary tumor types are respiratory system cancers, although a few reports have noted increased incidence of tumors at other sites, including the liver, skin, and digestive tract.*

Due to the limited database on non-carcinogenic effects of arsenic following the inhalation route of exposure, this assessment derived a value was based on carcinogenic effects. Using the unit risk provided by Health Canada of 6.4 per mg/m^3 (Health Canada 2009) a risk-based air concentration was calculated for exposure lasting 2-hour a day, 7 days a week, 10 weeks a year for 20 years based on a 1×10^{-5} risk level. A derived concentration of $0.4 \mu\text{g}/\text{m}^3$ can be used to assess potential carcinogenic effects.

In summary, this evaluation used a 1-hour value of $0.2 \mu\text{g}/\text{m}^3$ to assess non-carcinogenic effects due to arsenic exposure and a value of $0.4 \mu\text{g}/\text{m}^3$ was used to assess carcinogenic effects.

3.2 Assessment of Effects

As seen from Table 2, the maximum predicted 1-hour concentration of arsenic was $0.37 \mu\text{g}/\text{m}^3$, which would occur once in the year. For 99% of the time, the arsenic concentrations are predicted to be equal to or less than $0.18 \mu\text{g}/\text{m}^3$. This concentration is below both the non-carcinogenic and carcinogenic health based concentrations of $0.2 \mu\text{g}/\text{m}^3$ and $0.4 \mu\text{g}/\text{m}^3$ discussed above. Thus adverse effects from exposure to arsenic while walking along the Ingraham Trail 2-hours a day, every day for 10 weeks are not expected. In reality, it is not likely that someone would spend that length of time along the Highway that passes through the Giant Mine site. As previously noted, the background arsenic level is approximately $0.004 \mu\text{g}/\text{m}^3$, which is well below both criteria.

4.0 ASSESSMENT FOR PARTICULATE MATTER

There is a growing body of scientific studies linking air pollutants to health effects. Recent assessments of the available health data are implying a stronger link between particulate matter (PM) and health effects resulting from short- and long-term exposures.

The U.S. EPA (2009) conducted detailed reviews of the literature associated with exposure to air particulate matter. Including available evidence from atmospheric chemistry and exposure assessment studies enabled the agency to also develop causal determinations for a variety of health outcome categories. The U.S. EPA (2009) indicated that epidemiological, controlled human exposure and animal toxicological studies provide only suggestive evidence for relationships between short-term exposure to PM_{10} and cardiovascular effects, respiratory effects, and increased mortality. However, they observed that short-term epidemiologic studies

do consistently report positive associations between short-term exposure to PM_{2.5}. Thus this evaluation focused on exposure to PM_{2.5}.

Brook *et al* (2010) reported world-wide averages of 0.4 to 1% increase in daily mortality for a 10 µg/m³ increase in PM_{2.5} within 5 days of exposure. Similarly, Ostro *et al.* (2006) and Zanobetti and Schwartz (2009) reported a 0.1% and 0.98% increase in mortality (respectively) for a 10 µg/m³ increase in fine particulates.

As seen from Table 2, the maximum incremental increase of PM_{2.5} over background was predicted to be 5 µg/m³ with 98% of the time the incremental increase in the PM_{2.5} concentration is less than 2.9 µg/m³. Thus, the theoretical increase in mortality would be in the order of 0.1% to 0.2% which would not be discernible from baseline levels.

In addition, Pope and Dockery's (2006) summary comments on the results of short-term risks of air pollutants are very useful for putting these results into perspective:

"It seems unlikely that relatively small elevations in exposure to particulate air pollution over short periods of only 1 or a few days could be responsible for very large increases in death. In fact, these studies of mortality and short-term daily changes in PM are observing small effects. For example, assume that a short-term elevation of PM_{2.5} of 10 µg/m³ results in an ~1% increase in mortality. Based on the year 2000 average death rate for the United States (8.54 deaths/1000 per year), a 50-µg/m³ short-term increase in PM_{2.5} would result in an average of only 1.2 deaths per day in a population of 1 million (compared with an expected rate of ~23.5/day). That is, on any given day, the number of people dying because of PM exposure in a population is small.

It is remarkable that these studies of mortality and short-term changes in PM are capable of observing such small effects. Uncertainties in estimating such small effects legitimately create some doubts or concerns regarding the validity or accuracy of these estimates. Nevertheless, associations between daily changes in PM concentrations and daily mortality counts continue to be observed in many different cities and, more importantly, in large multi-city studies, which have much less opportunity for selection or publication bias. The estimated size of these associations is influenced by the methods used to control for potential confounding by long-term time trends, seasonality, weather, and other time-dependent covariates. However, numerous researchers using various methods, including alternative time series analytic approaches and case-crossover designs, continue to fairly consistently observe adverse mortality associations with short-term elevations in ambient PM."

It summary, it is unlikely that an individual using the Ingraham Trail during remediation activities at the Giant Mine site will experience any adverse health effects from exposure to particulate.

REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR) 2007. *Toxicological Profile for Arsenic*. U.S. Department of Health and Human Services. Public Health Service. August.
- Brook, R. D., Rajagopalan, S., Pope, C. A., III, Brook, J. R., Bhatnagar, A., Diez-Roux, A. V., *et al.* 2010. *Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement From the American Heart Association*. *Circulation*, 121(21), 2331-2378.
- California Environmental Protection Agency (CalEPA) 2008. *TSD for Noncancer RELs Appendix D. Individual Acute, 8-Hour, and Chronic Reference Exposure Level Summaries* December.
- Environment Canada 1999. *Canadian Soil Quality Guidelines Arsenic (Environmental and Human Health Effects) Scientific Supporting Document*. Prepared by National Guidelines and Standards Office.
- Health Canada 2009. *Federal Contaminated Site Risk Assessment in Canada Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors*. Draft.
- Nagymajtenyi L, Selyes A and Berencsi G (1985). *Chromosomal aberrations and fetotoxic effects of atmospheric arsenic exposure in mice*. *J Appl Toxicol* 5(2): 61-3. (Cited in CalEPA 2008)
- Ostro, B., Broadwin, R., Green, S., Feng, W. Y., & Lipsett, M. (2006). Fine particulate air pollution and mortality in nine California counties: results from CALFINE. *Environ Health Perspect*, 114(1), 29-33.
- Pope, C.A. and D.W. Dockery 2006. *Human Effects of Fine Particulate Air Pollution: Lines that Connect*. *Journal of the Air & Waste Management Association*, 56: 709-742.
- Risk Assessment Information System (RAIS) 1997. *Toxicological Profile for Arsenic*. Available at: <http://rais.ornl.gov/tox/profiles/arsenic.html>
- Rozenshtein, I.S. 1970. *Sanitary toxicological assessment of low concentrations of arsenic trioxide in the atmosphere*. *Hyg. Sanit.* 34:16-22. (Cited in Environment Canada 1999).
- United States Environmental Protection Agency (U.S. EPA) 2012. AEGL Program. Accessed 9 July 2012 at: <http://www.epa.gov/oppt/aegl/index.htm>

350047-020

30 July 2012

Memo to B. Halbert (Continued)

Page 9

United States Environmental Protection Agency (U.S. EPA) 2009. *Integrated Science Assessment for Particulate Matter*. EPA/600/R-08/139F. December 2009

Zanobetti, A., and Schwartz, J. 2009. *The Effect Of Fine And Coarse Particulate Air Pollution On Mortality: A National Analysis*. Environ Health Perspect, 117(6), 898-903.