

**DEER CREEK ENERGY LIMITED
JOSLYN NORTH MINE PROJECT**

**JOSLYN NORTH MINE PROJECT
Responses to Third Round Alberta Environment Supplemental Information
Request**

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HEALTH1

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HEALTH**1. Secondary EUB SIRs, Response 9, Page 11**

DCEL states that the malfunction of the vapour recovery system could result in irregular and short-term emissions.

Confirm that these irregular and short-term emissions were accounted for in the Human Health Risk Assessment (HHRA). If not, update the HHRA to account for these irregular and short-term events.

Response:

The HHRA is based on the air emissions resulting from steady-state or continuous operations of the facility. During short term upsets of the vapour recovery system, emissions would be routed to the emergency flare. The air emissions related to the worst-case flaring scenario are presented in the supplemental information submission, Volume 1; Project Update, Section 4.13 of the Revised Air Quality Assessment (Appendix B).

Impacts to human health from irregular and short-term emissions were accounted for and provided as part of supplemental information submission, Volume 2, Alberta Environment SIRs # 91 and #138).

2. Secondary EUB SIRs, Response 9, Page 11

DCEL states fluctuations in production rates and the quality of bitumen product are typically experienced during start-up and ramp-up periods. Solvent losses might exceed the target of 4 m³/1000 m³ of bitumen production during these periods. DCEL expects the solvent losses to be equal to or less than an average 4 m³/1000 m³ of bitumen production within the first year that full production at the nominal production rate of 100,000 bpd is achieved.

Confirm that solvent losses were accounted for in the HHRA. If not, update the HHRA.

Response:

Solvent losses were accounted for in the HHRA. Air emissions related to solvent losses can be found in supplemental information submission, Volume 1; Project Update, Table 4.3 of the Revised Air Quality Assessment (Appendix B).

3. Secondary EUB SIRs, Response 33, Table 33-1, Page 32

The table summarizes the estimate of construction personnel transportation movements to and from the Joslyn Lease.

Confirm that the emissions from the estimated transportation to and from the Joslyn Lease have been accounted for in the HHRA. If not, update the HHRA.

Response:

Although emissions from personnel transport during the construction phase of the Project were not assessed directly in the HHRA, traffic emissions from traffic on Highways 63, 69 and 881, which would include personnel transport to and from the various oil sands projects in the region, were included in the assessment. Moreover, in the CEA Case, traffic emissions on these highways were assumed to increase 3% per year during the life of the project (25 years) as a result of increasing development in the region. As the CEA Case was assessed near the end of the life of the project, traffic emissions in the CEA scenario were assessed at a rate that was more than two-fold greater than those in the Baseline and Application cases. As such, traffic generated by personnel transport during the construction phase has been sufficiently accounted for in the HHRA.

4. Secondary EUB SIRs, Response 31, Page 30

DCEL states that it *plans to examine the results of this intended mercury monitoring to confirm that the results will not pose a risk to the aquatic ecosystem*. As stated in the Supplemental Information, Volume 2, AENV SIRs, Response 118, *DCEL will ensure that water released to local waterbodies and watercourses will be in compliance with Canadian Council of Ministers of the Environment (CCME) water quality guidelines for mercury*. It should be noted that monitoring is not a substitute for completion of a HHRA, especially in situations where data is not yet available (i.e., future scenarios when the Project is actually in operation, closure, etc.). The results of a HHRA are important in order to assess current but also future scenarios where potential impacts to human health might be identified and, as a consequence, risk management practices might be required by DCEL to address these potential impacts.

In response to AENV SIR 11 on page 44, DCEL makes qualitative comparisons to other mining projects in the region with respect to mercury levels and does not discuss or provide evidence related to their potential contribution to mercury levels.

In the Supplemental Information, Volume 1, Appendix C, Section 2.2.1 and Section B-2.7 on page B-22, DCEL notes that mercury was not evaluated in the HHRA, citing that *background mercury concentrations measured in fish from the oil sands region are largely (i.e., 90th percentile) below guidelines that are protective of human health*. Further, on page B-23, DCEL states *information in this figure indicates that approximately 90% of walleye and northern pike and practically 100% of lake white fish have concentrations below Canadian consumption guidelines (i.e., 0.5 ppm) for the protection of human health (CFIA 2002)*.

As there is a potential that the Project may release or liberate mercury to the environment, include mercury in the HHRA for the three development cases and for the assessment of reclamation waterbodies and watercourses.

The guideline of 0.5 ppm is for commercial fish only, and not for recreational or sport fish. Health Canada's Bureau of Chemical Safety of Health Products and Foods Branch uses two provisional tolerable daily intakes (pTDIs) in their HHRA to assess health impact of mercury on the population, i.e., the value is 0.2 ug/kg bw/day for "sensitive" subgroups (women of reproductive age and young children), and for the rest of the population, the pTDI is 0.47 ug/kg bw/day. Since the guideline concentration of 0.5 ppm is not protective of sensitive subgroups as well as the guideline is only for the commercial fish, provide an HHRA for the sensitive subgroups taking into account the lower TDI level.

Response to (a) and (b) combined:

An assessment of potential mercury (i.e., inorganic and methyl mercury) impacts to human health is provided below. Background environmental concentrations were obtained from previous environmental impact assessments (EIAs) in the Athabasca oil sands. The following EIAs and regional initiatives provided the necessary information to characterize background concentrations:

- Shell Jackpine Mine Phase 1 (Golder 2002)
- Imperial Oil Kearl Oil Sands Project - Mine Development 2005 (IOL 2005)
- Birch Mountain Resources (2006 & 2007)
- Fish and water quality data provided by regional aquatics monitoring program (RAMP 2001 to 2004)

Table 4-1 provides a summary of the measured concentrations for total mercury. Whenever possible, the 95% upper confidence limit on the mean (95UCLM) was used in the human health risk assessment (HHRA) as exposure point concentrations.

Table 4-1 Summary of Baseline Environment Mercury Concentrations Used for the HHRA

Media	Avg±StDev	Min-Max	95UCLM	Reference/Comment
Air (µg/m ³)	2.41E-06	na	na	Baseline annual average at MPOI from Birch Mountain Resources (2006&2007)
Deposition (kg/ha/year)	1.52E-05	na	Na	Baseline annual average at MPOI from Birch Mountain Resources (2006&2007)
Soil (mg/kg)	0.05±0.008	0.003-0.05	0.05	All 5 samples > detection limit; (Golder 2002 & IOL 2005)
Surface water – Ells R. (ng/L)	1.2±1.3	0.3-3.5	3.2 ^a	7/12 samples non-detect; (RAMP 2001 to 2004)
Game meat (mg/kg WW)	All < detection limits	na	Na	Predicted with exposure models
Moose (mg/kg WW)	0.0069	na	Na	Predicted based on exposure to baseline soil, plant, and Ells River surface water

Media	Avg±StDev	Min-Max	95UCLM	Reference/Comment
Snowshoe hare (mg/kg WW)	0.00011	na	Na	Predicted based on exposure to baseline soil, plant, and Ells River surface water
Grouse (mg/kg WW)	0.00024	na	Na	Predicted based on exposure to baseline soil, plant, invertebrate and Ells surface water
Walleye Athabasca R. (mg/kg WW)	0.36±0.20	0.07-0.84	0.41	All 53 samples > detection limit
Northern Pike Muskeg R. (mg/kg WW)	0.16±0.08	0.03-0.25	0.25	All 15 samples > detection limit
Lake whitefish Athabasca R. (mg/kg WW)	0.12±0.07	0.04-0.45	0.13	All 54 samples > detection limit
All fish (mg/kg WW)	na	na	0.3	Average of 95UCLM for Walleye, Northern Pike and Lake Whitefish
Labrador tea (mg/kg WW)	0.018±0.015	0.005-0.05	0.03	12/17 samples non-detect
Blueberries (mg/kg WW)	0.014±0.015	0.005-0.05	0.03	14/16 samples non-detect
Roots (mg/kg WW)	0.03±0.06	0.005-0.25	0.19	12/15 samples non-detect
Terrestrial invertebrate concentrations (mg/kg DW)	0.4	na	Na	Predicted based on exposure to baseline soil

^a Based on 95th percentile because the estimated 95UCLM is greater than the maximum measured concentration; Mercury results based on ultra-low mercury analysis only

MPOI = Maximum point of impingement

DW = Dry weight

WW = Wet weight

95UCLM = 95th upper confidence limit on mean

Project-related mercury health risks were based on a combination of information available from previously submitted EIAs and recent water quality modeling conducted for DCEL¹. Table 4-2 summarizes the information that was used to estimate incremental impacts of mercury in the terrestrial and aquatic environment. Changes in terrestrial concentrations (i.e., soil, plants and game) were based on the predicted air concentrations,

¹ E-mail correspondence from Peter McNamee (Hatfield Group) to Karl Bresee (Intrinsic) on pit lake mercury water quality modeling (Date: 19-Dec-07)

deposition and accumulation in the terrestrial environment, conservatively assuming that no losses would occur over time. Potential changes in the aquatic environment (i.e., water and fish concentrations) were based on the view that predicted surface water concentrations in the Ells River are not expected to change significantly when compared to baseline conditions. The database of mercury monitoring of process-affected water, modified fine tailings, dewatering of polishing ponds and water courses from other oil sands mines that are currently operating have consistently reported non-detectable concentrations of mercury or concentrations of mercury that fall within the range of natural (or background) levels. Therefore, water and fish tissue concentrations in the Ells River were not predicted to change from baseline. However, water quality in pit lakes is expected to change over time. As a result, contaminant loading could occur in pit lake fish over time. However, access to the pit lake for fishing or other recreational activities will be restricted and public access will only be available when fish and water concentrations meet appropriate regulatory guidelines.

Table 4-2 Mercury Concentrations Used to Estimate Incremental Risks in the HHRA

Media	Value	Reference / Comment
CEA air concentration ($\mu\text{g}/\text{m}^3$)	2.75E-06	CEA annual average at MPOI from Birch Mountain Hammerstone supplemental submission. CEA case includes DCEL emissions (Birch Mountain resources 2006&2007)
CEA deposition (kg/ha/year)	2.14E-05	CEA annual average at MPOI from Birch Mountain Hammerstone supplemental submission. CEA case includes DCEL emissions (Birch Mountain resources 2006&2007).
CEA Ells River water concentration ($\mu\text{g}/\text{L}$)	0.003	Same as baseline; no change predicted
Pit lake starting water concentration ($\mu\text{g}/\text{L}$)	0.03	Represents "baseline" water quality in pit lake or water concentration in aquatic system before impacts are predicted
Long-term pit lake concentration on DCEL lease in year 5 ($\mu\text{g}/\text{L}$)	0.04	Predicted based on water quality modeling
Long-term pit lake concentration on DCEL lease in year 10 ($\mu\text{g}/\text{L}$)	0.05	Predicted based on water quality modeling
Long-term pit lake concentration on DCEL lease in year 15 ($\mu\text{g}/\text{L}$)	0.06	Predicted based on water quality modeling. After 15 years mercury concentrations "stabilize"
CEA fish tissue concentration (mg/kg WW)	0.6	Water quality in pit lake observed to double (i.e., 0.03 to 0.06 $\mu\text{g}/\text{L}$); therefore, fish tissue concentrations in pit lake assumed to double (i.e., 0.3 to 0.6 mg/kg WW)

MPOI = Maximum point of impingement

DW = Dry weight

WW = Wet weight

Table 4-3 provides a summary of predicted CEA exposure concentrations that were used in the HHRA.

Table 4-3 Summary of Predicted CEA Exposure Concentrations Used in the HHRA

Parameter	Mercury (Inorganic)	Methyl Mercury	Comment
Soil (mg/kg)	0.053	0	Slight increase due to atmospheric deposition
Drinking water (µg/L)	0.0032	0	No change
Pit lake (µg/L)	0.06	0	
Fish (mg/kg WW)	0.03 ^a	0.6	HHRA modeling based on assumption that 100% of fish are taken from pit lake
Terrestrial invertebrate (mg/kg DW)	0.4	0	Predicted based on exposure to CEA soil
Berries (mg/kg WW)	0.0302	0	Predicted based on exposure to CEA soil and air and site-specific BCF of 4
Plant (mg/kg WW)	0.0302	0	Predicted based on exposure to CEA soil and air and site-specific BCF of 4
Root (mg/kg WW)	0.19	0	Predicted based on exposure to CEA soil and air and site-specific BCF of 25
Moose (mg/kg WW)	0.007	0	Predicted based on exposure to CEA soil, plant, and pit lake concentration
Snowshoe hare (mg/kg WW)	0.00012	0	Predicted based on exposure to CEA soil, plant, and pit lake concentration
Grouse (mg/kg WW)	0.00024	0	Predicted based on exposure to CEA soil, plant, invertebrate, and pit lake concentration

^a Assumed that 5% of total mercury is in inorganic form (Hg) while the remaining 95% is in methylated form (MeHg) (Health Canada 2007).

DW = Dry weight

WW = Wet weight

BCF = Bio-concentration factor

The methods use to estimate exposures (e.g., consumption rates and body weights) and risks are identical to those presented in the project update. With the exception of the mercury and methyl mercury chronic exposure limit which was assumed to be the following:

- Mercury exposure limit (i.e., 0.3 µg/kg/day) based on US EPA (1995); and
- Methyl mercury limit (i.e., 0.1 µg/kg/day) based on US EPA (2001), which is lower than Health Canada’s provisional TDI of 0.2 µg/kg/day for sensitive subgroups.

Table 4-4 provides a summary of the exposure ratios (ER) for the Aboriginal receptor. Only the Aboriginal receptor is presented, because it was assumed that the consumption patterns of Aboriginal peoples (i.e., residents of Fort McKay) would represent the high end of the range of exposures. Methyl mercury exposure estimates are predicted to exceed the exposure limit, while inorganic mercury exposure estimates are less than the exposure limit. The exceedance of methyl mercury must be considered in the context of the conservative assumptions used in the assessment, which are:

- Baseline risks
- Risk estimates are based on a conservative exposure limit that uses a 10 fold uncertainty factor.
- Health Canada (2007) recommends a tolerable daily intake value of 0.47 µg/kg/day for adults and 0.2 µg/kg/day for women of child-bearing age and young children. These exposure limits are 2 and 5 times higher than the US EPA exposure limit. Using the Health Canada limits would reduce child ER values 2-fold and reduce adult ER values below the threshold value of 1.0.
- The HHRA team assumed that fish were harvested 100% of the time from the pit lake. Typically, fish would be harvested from a variety of locations (i.e., Athabasca River, Ells River, Muskeg River) with varying concentrations of mercury in fish.
- Changes in soil, game, and traditional plant concentrations were based on exposure to the maximum point of impingement air concentration.

Table 4-4 Summary of Baseline and CEA Scenario ER Values for the Fort McKay Receptor

Life stage	Mercury (Inorganic)	Methyl Mercury
<i>Baseline</i>		
Child	0.2	3.8
Adult	0.1	2.2
<i>CEA</i>		
Child	0.3	7.7
Adult	0.2	4.3

Based on the results of the mercury human health risk assessment, DCEL recommends monitoring of water quality and fish tissue quality in the Ells River and pit lake to ensure that concentrations meet regulatory guidelines for the protection of human health.

References

- Birch Mountain Resources Ltd. 2006. Hammerstone Project Application and Environment Impact Assessment. Prepared by AMEC Earth and Environmental.
- Birch Mountain Resources Ltd. 2007. Responses to Supplemental Information Request - Hammerstone Project Application. Prepared by AMEC Earth and Environmental.
- Golder. 2002. Appendix VI Baseline chemistry data for plants, fish, animal and soil for Jackpine Mine - Phase 1. Prepared for Shell Canada Limited. Draft. January 2002.
- Imperial Oil Ltd (IOL). 2005. Kearl Oil Sands Project - Mine Development Submitted to the Energy and Utilities Board and Alberta Environment.
- Health Canada 2007. Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption. Bureau of Chemical Safety Food Directorate Health Products and Food Branch. March 2007.
- Regional Aquatic Monitoring Program (RAMP). 2007. RAMP Reports can be accessed at the following internet site. <http://www.ramp-alberta.org/>
- US EPA 1995. United States Integrated Risk Information System (IRIS) Internet Site accessed 20-Dec-07 <http://cfpub.epa.gov/ncea/iris/index.cfm>
- US EPA 2001. United States Integrated Risk Information System (IRIS) Internet Site accessed 20-Dec-07 <http://cfpub.epa.gov/ncea/iris/index.cfm>

5. Secondary AENV SIRs, Response 10, Page 43

DCEL was asked to explain why and provide references to support the use of 10% CrVI in all media. In response, DCEL states *the assumption that 10% of chromium in all media is in its hexavalent form is conservative* and provides rationale for assuming that chromium released to the environment from the Project would not be 100% hexavalent chromium, however, a reference for selection of the benchmark of 10% was not provided.

- a) Provide evidence that 10% is truly conservative and explain why the value of 10% was selected.

Response

The 10% was originally selected based on the California Air Resources Board (CARB 1985) claim that hexavalent chromium made up between 3 and 8% of total chromium in ambient air. The HHRA team rounded 8% up to 10% and assumed that the ratio of total chromium to hexavalent chromium in air would remain consistent for other media as well (i.e., soils, plants and game).

Although limited information is available on the fraction of hexavalent chromium in soils and biota, recent review of peer reviewed literature has identified the following with respect to hexavalent chromium in the environment:

- Hexavalent chromium rarely occurs naturally, but is usually produced from anthropogenic sources (ATSDR 2005).
- Trivalent chromium is the most stable and abundant form in both native and contaminated soil. Hexavalent chromium is transformed to trivalent chromium) during the growing season by microorganisms and reduction by iron(II), organic matter and sulphide. About 2% of chromium in native soils was found as hexavalent chromium (Fengxiang et al. 2004).
- Chromium is a toxic, nonessential element to plants; hence specific mechanisms for uptake are unavailable. Therefore, uptake of this metal is through carriers used for the uptake of essential metals. Unlike trivalent chromium, hexavalent chromium uptake depends on the expenditure of metabolic energy by the plant. Uptake of hexavalent chromium is probably readily reduced to trivalent chromium in the root, which is the primary (~98%) area where chromium is stored in the plant (ATSDR 2000, Shanker et al. 2005).
- Trivalent chromium uptake by plants occurs more rapidly than hexavalent chromium (US EPA 2005).
- In the Priority Substances Assessment List for chromium, Environment Canada and Health Canada (1994) states that nearly all of the chromium in soils, sediments and biological tissues is likely present as trivalent chromium.
- Hexavalent chromium that is ingested by mammals is reduced to trivalent chromium before reaching sites of absorption or hexavalent chromium is rapidly reduced to trivalent chromium after penetration of biological membranes (US EPA 2005, ATSDR 2005).

Given that the study by Fengxiang et al. (2004) indicated that hexavalent chromium is 2% of total chromium in soils and that plants and mammals rapidly reduce hexavalent chromium to trivalent chromium, the assumption that hexavalent chromium is 10% of total chromium in all media is reasonable worst case.

References

- ATSDR 2000. Toxicological Profile for Chromium. U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES. Public Health Service. Agency for Toxic Substances and Disease Registry. September 2000.
- CARB (California Air Resources Board). 1985. Public Hearing to Consider Adoption of a Regulatory Amendment identifying Hexavalent Chromium as a Toxic Air Contaminant. Staff Report: Initial Statement of Reasons for Proposing Rulemaking. December 1985 Sacramento, California. Cited in CEPA 1994.
- CEPA (Canadian Environmental Protection Act). 1994. Chromium and its Compounds. Priority Substance List Assessment Report. Government of Canada. Environment Canada. Health Canada. Pp.35.
- Environment Canada and Health Canada, 1994. Priority Substances List Assessment Report: Chromium and Its Compounds.
- Fengxiang H., Yi S., Sridhar B.B., and Monts D.L. 2004. Distribution, transformation and bioavailability of trivalent and hexavalent chromium in contaminated soil. *Plant and Soil* 265, pp 243-252.
- Shanker A.K., Cervantes C., Loza-Tavera H., and Avudainayagam S. 2005. Chromium toxicity in plants *Environ. Inter.* 31, pp. 739-753.
- US EPA 2005. Ecological Soil Screening Levels for Chromium. Interim Final. March 2005.

6. Secondary AENV SIRs, Response 12, Page 47

DCEL states *metal emissions from diesel combustion were based on a research report provided by the Health Effects Institute (HEI 2006)...Molybdenum and nickel were not included because...molybdenum emissions from diesel were reported to be zero...nickel was not listed in the report's emission profile.*

Are the metal emissions from the Health Effects Institute (2006) consistent with the US EPA AP-42 diesel emissions estimates for point sources? Provide an explanation, if there are any discrepancies.

Response:

The U.S. EPA AP-42 document does not provide any metal emission factors for diesel combustion in internal combustion engines. The U.S. EPA has published emission factors for various metals species in a 1979 paper (U.S. EPA 1979) but a comparison between that paper with the Health Effects Institute (HEI) report indicates that HEI emission factors are typically higher by three to four orders of magnitude.

Reference

United States Environmental Protection Agency (US EPA). 1979. Emission Assessment of Conventional Stationary Combustion Systems; Volume II: Internal Combustion Sources. EPA-600/7-79-029c.

7. Secondary AENV SIRs, Response 14, Pages 49-51

DCEL states chemicals were not screened out of the assessment, but all chemicals were evaluated via the inhalation pathway on an acute and chronic basis. The fate and persistence screening was used to determine which chemicals need to be considered for oral exposures in addition to inhalation...no chemicals other than methane, butane and propane were screened out of the assessment. These chemicals do not represent a health hazard and were thus not included in the risk assessment because maximum concentrations would be well below those that would result in hypoxia or an explosive hazard. To date, the information related to the screening process is still insufficient, especially related to the inherent toxicity of chemicals which screened out using the fate and persistence criteria.

Provide the toxic potency screening to supplement the bioaccumulation/persistence screening. Update the HHRA.

According to DCEL's response, only methane, butane and propane were screened out in the HHRA. However, the environmental fate and persistence screening as shown in the response indicates that more chemicals were screened out than just methane, butane and propane for oral exposures and multi-media pathway. When the list of screened in chemicals on page 51 is compared to the list on page 50, there are 25 chemicals that have been screened out, including some polycyclic aromatic hydrocarbons (PAHs) and some carcinogens.

Response:

The results of the toxic potency screening to supplement the bioaccumulation/persistence screening are presented in Tables 7-1 and 7-2 below. A compound's toxic potency was determined by dividing its total emission estimate (for the mine and plant site) by its oral exposure limit (i.e., toxicological reference value or TRV). A compound's relative toxic potency was calculated by dividing the summed toxic potency of the emissions profile by the compound's individual toxic potency. Note that the compounds presented in Tables 7-1 and 7-2 match those that were originally "screened" using bioaccumulation/persistence criteria in the human health risk assessment (HHRA) (please refer to supplemental information submission, Volume 1; Project Update, Table 2-3 in Section 2.5 of the Appendix B).

Table 7-1 presents the results of the toxic potency screening for all individual chemicals of potential concern (COPCs), while Table 7-2 presents the results

for the COPCs when the carcinogenic PAHs are screened together as part of the benzo(a)pyrene group. In both tables, benzene, C9-C16 aliphatics and C5-C8 aliphatics comprise >99% of the emissions profile cumulative oral toxic potency. Of these three, only benzene was screened off using the bioaccumulation/persistence criteria in the Joslyn North Mine Project Application (Appendix B).

Use of the toxic potency screening exercise to supplement the bioaccumulation/persistence screening would result in benzene being added to the multiple exposure pathway assessment. However, level I fugacity modeling for benzene suggests that greater than 99% of benzene will preferentially partition to air (Mackay et al. 1992). The Agency for Toxic Substances and Disease Registry also claims that approximately 99% of human exposure to benzene is through inhalation (ATSDR 2005a). In its Priority Substances List Assessment Report for benzene, Environment Canada and Health Canada state that “accumulation of benzene is not expected to be important in any terrestrial or aquatic organism and [that] there are no reports indicating any significant bioconcentration in organisms or biomagnification in the food chain. The main route of exposure for terrestrial biota is, therefore, inhalation rather than exposure via the food chain” (EC and HC 1993). For the reasons stated and the fact that benzene will only be released into the ambient air, benzene should not be assessed through multiple routes of exposure, despite screening on with the toxic potency exercise.

The only PAHs that were not assessed through multiple routes of exposure in the HHRA were naphthalene, 2-methylnaphthalene and acenaphthene. All other PAHs were assessed through multiple routes of exposure either as part of the benzo(a)pyrene group or the aromatic C17-C34 group. The results of the oral toxic potency screen appeared to corroborate the exclusion of the same PAHs from the multiple exposure assessment.

ATSDR recognizes that the most likely pathway by which the general public is exposed to naphthalene and substituted naphthalenes is via inhalation, due to the release of the substances from combustion fuels, moth repellants and cigarette smoke (ATSDR 2005b). Further, CCME’s draft Canadian Soil Quality Guidelines for Carcinogenic and other Polynuclear Aromatic Hydrocarbons states that “the produce, meat and milk check was not calculated because PAHs are poorly bioaccumulated into plants and there is limited potential for food web mediated transfer” (Potter et al. 2007). Finally, ongoing environmental monitoring in the Athabasca oil sands region consistently fails to detect measurable concentrations of “lighter” PAHs (e.g., naphthalene) in soils or vegetation. For the reasons stated and in light of the results of both the oral toxic potency screening and bioaccumulation/persistence screening, the exclusion of naphthalenes, acenaphthylene and 1-methylnaphthalene from the multiple exposure assessment seems appropriate.

References

- ATSDR (Agency for Toxic Substances and Disease Registry), 2005a. Toxicological Profile for Benzene. Atlanta, GA. U.S. Department of Health and Human Services, Public Health Service. September 2005.
- ATSDR (Agency for Toxic Substances and Disease Registry), 2005b. Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene. Atlanta, GA. U.S. Department of Health and Human Services, Public Health Service. August 2005.
- EC (Environment Canada) and HC (Health Canada), 1993, Priority Substances List Assessment Report for Benzene: Canadian Environmental Protection Act. ISBN 0-662-20434-4.
- Mackay, D. , W.C. Shiu and K.C. Ma, 1992. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volumes I-IV*. Lewis Publishers. Chelsea, Michigan.
- Potter, K., Bright, D., Coad, S., Rowat, C. and D. Spry, 2007. Draft Canadian Soil Quality Guidelines for Carcinogenic and other Polynuclear Aromatic Hydrocarbons. Presented at the 2007 SETAC Conference.

Table 7-1 Oral toxic potency screen to determine COPC assessed through multiple routes of exposure (all chemicals)

COPC	(A) Total Emission (g/sec)	Screened "on" in Section 2.5 of Project Update HHRA	(B) Oral TRV (mg/kg/day)	Source	(A)/(B) Toxic Potency	Relative Potency	Cumulative Potency
Benzene	3.33E-01	No	0.0000322	HC	1.04E+04	9.68E-01	96.8%
Aliphatic C9-C16 group	1.72E+01	Yes	0.1	MADEP	1.72E+02	1.61E-02	98.4%
Aliphatic C5-C8 group	4.85E+02	Yes	5	CCME	9.70E+01	9.07E-03	99.3%
Dibenzo(a,h)anthracene	2.15E-05	Yes	0.000001273	EPA	1.69E+01	1.58E-03	99.5%
Aromatic C9-C16 group	5.47E-01	Yes	0.04	CCME	1.37E+01	1.28E-03	99.6%
Fluoranthene	2.49E-04	Yes	0.000028	EPA	8.88E+00	8.31E-04	99.7%
Toluene	5.85E-01	No	0.08	EPA	7.31E+00	6.84E-04	99.8%
Benzo(a)pyrene	9.90E-06	Yes	0.0000014	EPA	7.07E+00	6.62E-04	99.9%
Acrolein	2.46E-03	No	0.0005	EPA	4.93E+00	4.61E-04	99.9%
Benzo(b)fluoranthene	3.93E-05	Yes	0.000014	EPA	2.81E+00	2.63E-04	99.9%
Xylenes	4.91E-01	No	0.2	EPA	2.46E+00	2.30E-04	99.9%
Indeno(1,2,3-cd)pyrene	1.70E-05	Yes	0.000014	EPA	1.22E+00	1.14E-04	100.0%
Ethylbenzene	1.02E-01	No	0.1	EPA	1.02E+00	9.57E-05	100.0%
Chrysene	4.75E-05	Yes	0.00004667	EPA	1.02E+00	9.52E-05	100.0%
Phenanthrene	1.80E-03	Yes	0.0028	EPA	6.44E-01	6.02E-05	100.0%
Fluorene	9.09E-04	No	0.0028	EPA	3.25E-01	3.04E-05	100.0%
Benzo(g,h,i)perylene	2.24E-05	Yes	0.00007	EPA	3.19E-01	2.99E-05	100.0%
Naphthalene	6.33E-03	No	0.02	EPA	3.17E-01	2.96E-05	100.0%
Benzo(k)fluoranthene	8.78E-06	Yes	0.000028	EPA	3.14E-01	2.93E-05	100.0%
Formaldehyde	5.25E-02	No	0.2	EPA	2.63E-01	2.46E-05	100.0%
Benzo(a)anthracene	4.40E-05	Yes	0.00028	EPA	1.57E-01	1.47E-05	100.0%
Pyrene	1.87E-04	Yes	0.0014	EPA	1.34E-01	1.25E-05	100.0%
Anthracene	7.54E-05	Yes	0.0028	EPA	2.69E-02	2.52E-06	100.0%
Aliphatic C17-C34 group	5.34E-02	Yes	2	CCME	2.67E-02	2.50E-06	100.0%
Acenaphthene group	5.70E-04	Yes	0.06	EPA	9.50E-03	8.88E-07	100.0%
Dichlorobenzene	3.46E-04	No	0.09	EPA	3.85E-03	3.60E-07	100.0%
2-Methylnaphthalene	6.93E-06	No	0.004	EPA	1.73E-03	1.62E-07	100.0%
Aromatic C17-C34 group	5.14E-06	Yes	0.03	CCME	1.71E-04	1.60E-08	100.0%
1,3-Butadiene	1.92E-03	No	No data		0.00E+00	0.00E+00	100.0%
Acetaldehyde	1.94E-02	No	No data		0.00E+00	0.00E+00	100.0%

THIRD ROUND ALBERTA ENVIRONMENT SIR RESPONSES

COPC	(A) Total Emission (g/sec)	Screened "on" in Section 2.5 of Project Update HHRA	(B) Oral TRV (mg/kg/day)	Source	(A)/(B) Toxic Potency	Relative Potency	Cumulative Potency
Hexane group	1.27E+00	No	No data		0.00E+00	0.00E+00	100.0%
					1.07E+04	1.00E+00	

Notes: Shaded cells identify those COPCs that make up >99% of the emissions profile cumulative toxic potency

HC = Health Canada

EPA = Environmental Protection Agency

CCME = Canadian Environmental Protection Agency

TRV = Toxicological Reference Value

Table 7-2 Oral toxic potency screen to determine COPC assessed through multiple routes of exposure (carcinogenic PAHs screened as part of benzo(a)pyrene group)

COPC	Emission (g/sec)	Screened "on" in Section 2.5 of the Project Update HHRA	Oral TRV (mg/kg/day)	Toxic Potency	Relative Potency	Cumulative Potency
Benzene	0.333407679	No	0.0000322	1.04E+04	9.68E-01	96.8%
Aliphatic C9-C16 group	17.23343908	Yes	0.1	1.72E+02	1.61E-02	98.4%
Aliphatic C5-C8 group	485.2077311	Yes	5	9.70E+01	9.07E-03	99.3%
Benzo(a)pyrene group	5.5753E-05	Yes	0.0000014	3.98E+01	3.72E-03	99.7%
Aromatic C9-C16 group	0.547202624	Yes	0.04	1.37E+01	1.28E-03	99.8%
Toluene	0.584721263	No	0.08	7.31E+00	6.84E-04	99.9%
Acrolein	0.002463347	No	0.0005	4.93E+00	4.61E-04	100.0%
Xylenes	0.491000257	No	0.2	2.46E+00	2.30E-04	100.0%
Ethylbenzene	0.102325239	No	0.1	1.02E+00	9.57E-05	100.0%
Naphthalene	0.006330126	No	0.02	3.17E-01	2.96E-05	100.0%
Formaldehyde	0.052534464	No	0.2	2.63E-01	2.46E-05	100.0%
Aliphatic C17-C34 group	0.053391868	Yes	2	2.67E-02	2.50E-06	100.0%
Acenaphthene group	0.000570037	No	0.06	9.50E-03	8.88E-07	100.0%
Dichlorobenzene	0.000346494	No	0.09	3.85E-03	3.60E-07	100.0%
2-Methylnaphthalene	6.92975E-06	No	0.004	1.73E-03	1.62E-07	100.0%
Aromatic C17-C34 group	5.13938E-06	Yes	0.03	1.71E-04	1.60E-08	100.0%
1,3-Butadiene	0.001917689	No	NA	0.00E+00	0.00E+00	100.0%
Acetaldehyde	0.019429217	No	NA	0.00E+00	0.00E+00	100.0%
Hexane group	1.270478656	No	NA	0.00E+00	0.00E+00	100.0%
				1.07E+04	1.00E+00	

Notes: Shaded cells identify those COPCs that make up >99% of the emissions profile cumulative toxic potency

HC = Health Canada

EPA = Environmental Protection Agency

CCME = Canadian Environmental Protection Agency

TRV = Toxicological Reference Value

As mentioned in part a), confirm that the chemicals screened out using the fate and persistence criteria in the HHRA do not have a high potential for human exposure or do not have inherent human toxicity, and provide a rationale for each chemical that has been screened out.

In AENV SIRs, Responses 21a) and b) on pages 60-61, DCEL discusses how the range of natural variability was determined for incremental effects on water quality.

In AENV SIRs, Response 33b) on page 81, DCEL states that it *expects the end pit lake to develop into a naturally functioning lake similar to other lakes in the region and thus provide the same food resource supply to waterfowl as those lakes...Depending upon the connectivity of the end pit lake with fishbearing waterbodies, various fish species might eventually occupy the lake, offering a forage base for various diving species of ducks.*

It is still unclear how the water screening was employed in the revised HHRA.

Response:

All chemicals shown on page 50 were assessed in the HHRA. The list on page 51 of the responses to the Secondary Alberta Environment SIRs, shows only those PAHs and petroleum hydrocarbon groups that screened onto the multiple exposure assessment using the bioaccumulation/persistence screening. As noted, all metals were included in the multiple exposure assessment. Those compounds not shown on page 51 (excluding the metals) were assessed through the inhalation pathway only. Please refer to the response to question 7(a) above for further clarification on the screening approach used in the Project Update HHRA.

Provide a chemical screening for the waterbodies and reclamation waterbodies and watercourses. Provide this information and update the HHRA.

Response

c) With respect to surface water quality, the compounds assessed in the Project Update HHRA were listed in Table B1-3 in Appendix B1 (Appendix C of the Project Update). Note for the Project Update, no screening was done of any of the compounds in the area waterbodies or watercourses. All metals and PAHs for which predicted water concentrations were available were assessed in the Project Update HHRA.

8. Secondary AENV SIRs, Response 15, Pages 52-53

DCEL was asked to reassess the impacts to human health to include acrolein for the acute respiratory irritant group. In response, DCEL states the acute exposure limit used for acrolein predicted from the Joslyn North Mine Project was based on eye irritation...and not respiratory irritation...Therefore, these two toxicologically different endpoints should not be added to evaluate potential additive interactions, instead, potential additive interactions were identified for chemicals of potential concern (COPCs) known to cause eye irritation.

- a) **Include acrolein using a respiratory endpoint and reassess the respiratory mixtures.**

Response

In order to include the risks associated with acrolein in the nasal and respiratory tract irritant mixtures, a second acute inhalation exposure limit based on nasal and respiratory tract irritation was identified. The Agency for Toxic Substances and Disease Registry (ATSDR 2006) provides an acute minimal risk level (MRL) of 0.003 ppm (0.0069 mg/m³) based on decreased respiratory rate and nose and throat irritation. Forty-six (46) volunteers were exposed to a gradually increasing concentration of acrolein for 40 minutes. Participants subjectively scored irritancy at 5-minute intervals as the concentrations increased from 0 to 0.6 ppm (0 to 1.3 mg/m³). For the final 5 minutes of exposure, participants were exposed to 0.6 ppm. A LOAEL for nose irritation of 0.26 ppm (0.60 mg/m³) was identified. The ATSDR applied an uncertainty factor of 100 to the LOAEL to account for the use of a LOAEL (10-fold) and intra-species variability (10-fold). The resultant acute MRL of 6.9 µg/m³ was used as a 1-hour inhalation limit in the assessment of nasal and respiratory tract irritant mixtures.

Table 8-1 provides a summary of the revised CR values, which included acrolein as part of the respiratory irritant mixture using the limit described above. With the exception of the Mannix location, CR values for all assessment scenarios and locations are below 1.0, indicating that adverse effects from exposures to acute respiratory mixtures are not expected.

CR values were calculated to be slightly above 1.0 in the Baseline (CR value = 1.1), Application (CR value = 1.1) and CEA (CR value = 1.3) scenarios at Mannix. The CR values are the same for the Application and Baseline scenarios, suggesting that the respiratory irritant risks at Mannix are not expected to increase as a result of the Project. The exceedances of the respiratory irritants at Mannix are primarily (93%) due to NO₂ (22%) and SO₂ (71%).

Nitrogen dioxide can be inhaled deeply into the lungs, acting as a deep-lung irritant, whereas SO₂ is more soluble in water and is readily absorbed through the upper respiratory tract, inducing increases in airway resistance higher up in the respiratory tract (Calabrese 1991). The dose-response relationships for these chemicals are somewhat independent in that the primary responses occur in different regions of the respiratory tract. As well, there is conservatism incorporated into the acute inhalation

exposure limits for some of the respiratory tract irritants (e.g., sulphur dioxide and acrolein).

There is a margin of safety between the predicted acute SO₂ concentrations at Mannix (i.e., 1-hr & 24-hr value of 179 & 118 µg/m³, respectively) and the threshold of effects for severe asthmatics (530 – 1060 µg/m³).

At sufficiently high concentrations, SO₂ acts as an upper respiratory tract irritant. At lower concentrations, it is effectively “scrubbed” by the nose. This scrubbing action prevents the movement of SO₂ further down the respiratory tract, thereby reducing the likelihood of bronchoconstriction and other signs of respiratory distress.

The response to the irritant action of SO₂ is immediate. Short-term peak exposures are the primary determinants of the response.

- Asthmatic individuals are known to be very sensitive to the irritant effects of SO₂. Exercising asthmatics are especially sensitive. Sensitivity will vary depending on the severity of the asthmatic condition, the level of physical activity, and the pattern of breathing (i.e., oral vs. nasal).
- The majority of clinical evidence suggests that most individuals will not notice any response to short-term exposures to concentrations of SO₂ at or below 1,000 µg/m³. This finding applies even to exercising individuals with mild asthma.
- The threshold of response among severe asthmatics appears to rest between 530 and 1,060 µg/m³ based on responses observed among freely-breathing asthmatics engaged in moderate exercise.
- For the majority of healthy individuals, responses to SO₂ are unlikely to be noticed by the individual unless concentrations exceed 2,000 µg/m³, with some evidence to suggest that concentrations as high as 2,600 µg/m³ can be tolerated without significant discomfort. Above this “threshold” concentration, effects can range from mild respiratory effects among normal individuals during exercise (2,600 to 5,200 µg/m³) to more severe effects, including irritation and inflammation of the lungs and breathing passages that can progress to pneumonia (more than 26,000 µg/m³). The effects of these higher concentrations on the health of asthmatics can be serious and life-threatening.

The maximum predicted acute NO₂ air concentrations (i.e., 1-hr & 24-hr value of 99 & 49 µg/m³, respectively) is below effect levels reported for decreased airway responsiveness, inflammation, and pulmonary function among asthmatics (375 to 565 µg/m³), and far below levels known to induce lung function decrements in healthy individuals (greater than 1,900 µg/m³). Some studies have reported mild respiratory effects in asthmatics below 375 µg/m³ (Cal EPA 2007); however, due to the lack of a clear dose-response relationship and statistical uncertainty in these studies, the findings of these studies are not considered to be indicative of acute effects associated with NO₂ exposure (WHO 2000; Forastiere et al. 2006; Cal EPA 2007).

Based on the degree of conservatism incorporated into the acute inhalation exposure limits for some of the respiratory irritants (e.g., NO₂ and SO₂), adverse effects from the respiratory irritant mixture at Mannix are not expected.

Table 8-1 Summary of Acute Respiratory Irritant CR Values, Including Acrolein

Receptor	Baseline	Application	CEA
Fort McKay	0.5	0.5	0.7
Patricia McInnes Station, Fort McMurray	0.5	0.5	0.8
Athabasca Valley, Fort McMurray	0.5	0.5	0.7
Grandjambe Camp	0.5	0.5	0.6
Mine Construction Camp	0.5	0.5	0.5
SAGD Operators Camp	0.5	0.6	0.7
Albian Mine Site	0.9	0.9	0.9
Lower Camp	0.6	0.6	0.6
Mannix	1.1	1.1	1.3
Millenium	0.5	0.5	0.5

References

- ATSDR 2006. Minimal Risk Levels (MRLs) for Hazardous Substances. US Department of Health and Human Services, Public Health Service. Atlanta, GA.
- Calabrese, E.J. 1991. Multiple Chemical Interactions. Lewis Publishers Inc., Chelsea, Michigan. Toxicology and Environmental Health Series.
- Cal EPA. 2007. Review of the California Ambient Air Quality Standard For Nitrogen Dioxide: Technical Support Document. California Environmental Protection Agency Air Resources Board and Office of Environmental Health and Hazard Assessment. Available at: <http://www.arb.ca.gov/research/aaqs/no2-rs/no2tech.pdf>
- Forastiere, F., Peters, A., Kelly, F.J., and Holgate, S.T. 2006. Nitrogen dioxide. In: Air Quality Guidelines Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. ISBN 92 890 2192 6. WHO Regional Office for Europe, Scherfigsvej 8, DK-2100 Copenhagen O, Denmark, World Health Organization. pp 331-394
- WHO. 2000. Air Quality Guidelines for Europe, Second Edition. Copenhagen, Denmark: World Health Organization, Regional Office for Europe. WHO Regional Publications, European Series, No. 91. Available at: <http://www.euro.who.int/document/e71922.pdf>.

9 Secondary AENV SIRs, Response 19, Page 57

DCEL provides the list of age categories for the Wein (1989) study, but does not provide the frequency of intake for each category and how they were used to adjust the ingestion rates for each age class used for this assessment.

Provide this information and any supporting calculations.

Response:

Consumption rates used in the HHRA for Fort McKay residents were based in part on the Wein (1989) study. Table 9-1 provides a summary of the values that were used in the HHRA of the Project Update. Wein (1989) describes the number of occasions when country foods were used in 120 native households (Table 9-2). The frequency of consumption was applied to the total game meat consumption rate of 207 g/day. This value is found on page 66 of Wein (1989) and is derived from a Nutrition Canada survey (Health and Welfare Canada 1977) that determined Aboriginal people consume 207 g/day of wild meat, poultry, fish and eggs, on average. The HHRA team assumed that this consumption rate represented an adult consumption rate.

The adult large game or moose consumption rate was calculated as follows: 207 g/day x 51% = 106 g/day. Similar calculations were used to calculate adult consumption rates for the remaining country foods. Finally the consumption rate for the remaining life stages (i.e., toddler, child and adolescent) were derived by multiplying the adult consumption rate for each food group by the ratio of the life stage body weight to the adult body weight. For example, the toddler moose meat consumption rate was calculated as follows: 106 g/day x 31 kg-BW/ 70.9 kg-BW = 50 g/day. Infant consumption rates were assumed to be zero based on the recommendation by Health Canada (2004).

Table 9-1 Summary of Consumption Rates for Fort McKay Receptors Derived from Wein

Food	Adolescent	Adult	Child	Infant	Toddler
Moose	90	106	49	0	25
Ruffed Grouse	9	11	5	0	3
Snowshoe Hare	19	22	10	0	5

Table 9-2 Number of Occasions When Country Foods were Used in 120 Native Households

Country Food	Frequency of consumption (Wein 1989 pg 60)	Percentage of total frequency
Large mammals (moose)	128	51%
Small mammals (hare)	27	11%
Upland birds (grouse)	13	5%
Fish	62	25%
Waterfowl	19	8%
Total frequency	249	100%

References

Health Canada. 2004. Contaminated Site Program. Federal Contaminated Site Risk Assessment in Canada. Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment. September 2004.

Health and Welfare Canada 1977. Nutrition Canada Food Consumption Patterns Report. Ottawa: Bureau of Nutritional Sciences, health Protection Branch.

Wein, E.E. 1989. Nutrient intakes and use of country foods by native Canadians near Wood Buffalo National Park. A thesis presented to the Faculty of Graduate Studies of the University of Guelph. February, 1989.

10. Secondary AENV SIRs, Response 20, Pages 58 to 59

DCEL states *the assessment for the operations phase is conservatively representative of the construction phase*. DCEL does not provide a list of emission rates for particular compounds emitted from the Project.

Provide a comparison using emission rates (t/d) for construction and operational phases of the Project for SO₂, NO_x, CO, VOCs and PM_{2.5}.

Response:

Emissions for SO₂, NO_x, CO, VOCs and PM_{2.5} during the construction phase are approximated using fuel consumption estimates provided in the response for Secondary Alberta Environment SIR #20 (i.e. diesel consumption is approximately 56% of the amount used during peak operations, gasoline consumption is approximately one-third of diesel consumption during the construction phase, and natural gas emissions is approximately 1% of natural gas emissions during peak operations). Table 10-1 below compares the emissions of the construction and operational phases.

Table 10-1 Comparison of Construction versus Operational Emissions

Phase	Emission Rates (t/d)				
	SO ₂	NO _x	CO	PM _{2.5}	VOCs
Project Only -- Construction	0.01	4.40	6.73	0.16	0.88
Project Only - Operations	0.05	12.66	9.95	0.39	46.58 ¹

¹ Values represent peak summer emission rates

Table 10-1 demonstrates that emission rates for all chemicals are lower in the construction phase than in the operations phases

11 Secondary AENV SIRs, Response 24, Pages 63 and 64

DCEL states *aliphatic VOC C5-C8 group and C9-C18 group at Mannix for the Baseline, Application and CEA Cases would present an exceedance of acute health-based criteria. In all circumstances, the risk value is exactly the same for each assessment case, indicating that the Joslyn North Mine Project will not affect air quality at this location.*

- a. If doubling of the VOCs loading to the tailings pond and the resultant approximate doubling of fugitive VOC emissions from the tailings pond might occur provide an updated HHRA to address this issue. Explain why the risk value does not double as VOCs from the froth treatment tailings may be approximately 2x their original estimate?

Response:

- a) Updated acute and chronic health risks are provided below. The doubling of VOC emissions would not necessarily result in a doubling of concentrations at all receptor locations because those locations that are located further away from the tailings pond (i.e., primary VOC emission source) would be influenced to a lesser degree. In addition to the tailings pond, VOCs will also be emitted from the processing plant and mine fleet. Despite this, the updated health risks are based on the conservative assumption that the doubling of the VOC emissions will translate to a doubling of ground level air concentrations at all receptor locations.

The updated acute and chronic health risks are presented as concentration ratios in Tables 11-1 and 11-6, respectively. Comparison of the health risks at the Mannix location (i.e., the only location at which exceedances are predicted to occur) indicates that the risks are identical for the three development cases. At the other locations, risks for the CEA scenario are always higher than the risks for the Baseline and Application scenarios. If risks are acceptable in the CEA scenario, then by default the risks will be acceptable in the Baseline and Application scenario.

As indicated in Secondary Alberta Environment SIR Response #24, the aliphatic C5-C8 group and C9-C18 group exceeded health based criteria at Mannix. The exceedance of health based criteria at the Mannix location is hypothetical. It is unlikely that the doubling of VOC emissions from the tailings pond would double the short-term air concentrations at Mannix, which is 40km away. Meteorological conditions and air dispersion would influence emissions and dramatically reduce the impact of VOC emissions at the Mannix station.

Health risks associated with the exceedance of the health based exposure limit for the aliphatic C5-C8 group at the Mannix location are not expected for the following reasons:

- The hourly air concentrations are just slightly above the health-based exposure limit;
- The health based acute exposure limit is based on a no observable adverse effect level (NOAEL);

- The health based acute exposure limit is based on a sub-chronic study not an acute study; and
- The health based acute exposure limit incorporates an uncertainty factor of 1000.

Health risks associated with the exceedance of the health based exposure limit for the aliphatic C9-C18 group at the Mannix location are not expected for the following reasons:

- The hourly air concentrations are just slightly above the health-based exposure limit;
- The acute exposure limit is based on a 6-months sub-chronic study instead of a true acute study; and
- The health based acute exposure limit incorporates an uncertainty factor of 1000.

In all other cases, the acute CR values are below the threshold value of 1.0.

Even with doubled ground-level air concentrations, chronic CR values are less than 1.0 at all locations (Table 11-6).

Acute and chronic mixture toxicity CEA CR values at receptor locations are below the threshold value of 1.0 in all circumstances, except acute risks at the Mannix location (CR = 1.1). Based on the conservative assumptions presented above (i.e., conservative nature of toxicity reference values), the distance of the Mannix location from the tailings pond and the marginal exceedance of the health based criteria, adverse effects are not expected.

Table 11-1 Summary of Baseline Acute CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort Mckay	0.00001	0.05	0.06	0.000001	0.002
Patricia McInnes Station, Fort McMurray	0.00001	0.05	0.06	0.00001	0.001
Athabasca Valley, Fort McMurray	0.00000	0.03	0.04	0.00002	0.001
Grandjambe Camp	0.00002	0.05	0.06	0.000001	0.001
Mine Construction Camp	0.00001	0.05	0.06	0.000001	0.001
SAGD Operators Camp	0.00001	0.1	0.1	0.000001	0.001
Albian Mine Site	0.00008	0.2	0.3	0.000003	0.01
Lower Camp	0.00002	0.3	0.4	0.000001	0.002
Mannix	0.00003	1.1	1.4	0.000001	0.004
Millenium	0.00002	0.2	0.2	0.000001	0.001

Table 11-2 Summary of Baseline Chronic CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort McKay	0.00001	0.01	0.03	0.00000005	0.003
Patricia McInnes Station, Fort McMurray	0.000002	0.004	0.01	0.0000002	0.001
Athabasca Valley, Fort McMurray	0.000001	0.003	0.01	0.0000002	0.001
Grandjambe Camp	0.00001	0.01	0.02	0.0000000	0.003
Mine Construction Camp	0.000003	0.01	0.02	0.0000001	0.002
SAGD Operators Camp	0.000002	0.01	0.03	0.0000001	0.001
Albian Mine Site	0.00003	0.02	0.1	0.0000003	0.01
Lower Camp	0.00001	0.03	0.1	0.0000001	0.005
Mannix	0.00001	0.1	0.3	0.0000001	0.006
Millenium	0.00001	0.01	0.04	0.00000002	0.001

Table 11-3 Summary of Application Acute CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort Mckay	0.00001	0.05	0.06	0.000001	0.002
Patricia McInnes Station, Fort McMurray	0.00001	0.05	0.06	0.00001	0.001
Athabasca Valley, Fort McMurray	0.00000	0.03	0.04	0.00002	0.001
Grandjambe Camp	0.00002	0.05	0.06	0.000002	0.001
Mine Construction Camp	0.00002	0.08	0.10	0.000001	0.002
SAGD Operators Camp	0.00001	0.1	0.1	0.000001	0.001
Albian Mine Site	0.00008	0.2	0.3	0.000004	0.01
Lower Camp	0.00002	0.3	0.4	0.000001	0.002
Mannix	0.00003	1.1	1.4	0.000001	0.004
Millenium	0.00002	0.2	0.2	0.000001	0.001

Table 11-4 Summary of Application Chronic CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort Mckay	0.00001	0.01	0.03	0.00000005	0.003
Patricia McInnes Station, Fort McMurray	0.000002	0.004	0.01	0.0000002	0.001
Athabasca Valley, Fort McMurray	0.000001	0.003	0.01	0.0000002	0.001
Grandjambe Camp	0.00001	0.01	0.03	0.0000000	0.003
Mine Construction Camp	0.000004	0.01	0.03	0.0000001	0.003
SAGD Operators Camp	0.000003	0.01	0.04	0.000001	0.002
Albian Mine Site	0.00003	0.02	0.1	0.0000003	0.01
Lower Camp	0.00001	0.03	0.1	0.0000001	0.005
Mannix	0.00001	0.1	0.3	0.0000001	0.006
Millenium	0.00001	0.01	0.04	0.00000002	0.001

Table 11-5 Summary of CEA Acute CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort Mckay	0.00002	0.05	0.07	0.00001	0.002
Patricia McInnes Station, Fort McMurray	0.00001	0.05	0.06	0.00002	0.001
Athabasca Valley, Fort McMurray	0.00001	0.03	0.04	0.00003	0.001
Grandjambe Camp	0.00002	0.08	0.10	0.000002	0.001
Mine Construction Camp	0.00002	0.09	0.12	0.000001	0.002
SAGD Operators Camp	0.00002	0.1	0.1	0.000002	0.001
Albian Mine Site	0.00009	0.3	0.4	0.000004	0.01
Lower Camp	0.00002	0.3	0.3	0.000001	0.002
Mannix	0.00003	1.1	1.4	0.000002	0.004
Millenium	0.00002	0.2	0.2	0.000001	0.001

Table 11-6 Summary of CEA Chronic CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Aliphatic C19-C34	Aliphatic C5-C8 group	Aliphatic C9-C18	Aromatic C17-C34 group	Aromatic C9-C18 group
Fort Mckay	0.00001	0.01	0.03	0.0000001	0.003
Patricia McInnes Station, Fort McMurray	0.000002	0.005	0.01	0.0000003	0.001
Athabasca Valley, Fort McMurray	0.000001	0.004	0.01	0.0000005	0.001
Grandjambe Camp	0.00001	0.01	0.03	0.0000001	0.003
Mine Construction Camp	0.000005	0.01	0.03	0.0000001	0.003
SAGD Operators Camp	0.000003	0.01	0.04	0.0000001	0.002
Albian Mine Site	0.00003	0.02	0.1	0.0000004	0.02
Lower Camp	0.00001	0.03	0.1	0.0000001	0.005
Mannix	0.00001	0.1	0.3	0.0000004	0.006
Millenium	0.00001	0.01	0.03	0.00000004	0.001

b. Would the same conclusions be applicable for the mixture groups as well

Response:

b) As shown in Tables 11-7 and 11-8, the acute and chronic CRs for the mixtures are less than 1.0 in most cases, with the lone exception occurring at the Mannix location (CR = 1.1). However, based on the conservative assumptions presented above (i.e., conservative nature of toxicity reference values), the distance of the Mannix location from the tailings pond and the marginal exceedance of the health based criteria, adverse effects are not expected.

Table 11-7 Summary of CEA Acute Mixture CR Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Hepato-toxicants	Nephro-toxicants	Neurotoxicants
Fort Mckay	0.05	0.05	0.03
Patricia McInnes Station, Fort McMurray	0.05	0.05	0.05
Athabasca Valley, Fort McMurray	0.03	0.03	0.04
Grandjambe Camp	0.08	0.08	0.01
Mine Construction Camp	0.09	0.09	0.01
SAGD Operators Camp	0.1	0.1	0.03
Albian Mine Site	0.3	0.3	0.04
Lower Camp	0.3	0.3	0.02
Mannix	1.1	1.1	0.06
Millenium	0.2	0.2	0.01

Table 11-8 Summary of CEA Chronic CR Mixture Values Based on Doubling of VOC Concentrations at Receptor Locations

Receptor	Hepato-toxicants	Nephro-toxicants	Neurotoxicants
Fort Mckay	0.003	0.004	0.3
Patricia McInnes Station, Fort McMurray	0.001	0.001	0.3
Athabasca Valley, Fort McMurray	0.001	0.001	0.3
Grandjambe Camp	0.003	0.003	0.3
Mine Construction Camp	0.003	0.003	0.3
SAGD Operators Camp	0.002	0.002	0.3
Albian Mine Site	0.02	0.02	0.4
Lower Camp	0.005	0.005	0.4
Mannix	0.006	0.006	0.8
Millenium	0.001	0.001	0.3

c) Natural Resources Canada (NRCAN) has indicated that DCEL has taken statements from the Kasperski and Munoz report regarding the distribution of hydrocarbon in the Suncor froth treatment tailings and interpreted them in terms of the availability for vapourization of diluent. NRCAN has also indicated that there is nothing in this report that supports the use of only 50% of the diluent in the pond will contribute to VOCs and, therefore, DCEL is potentially underestimating VOC concentrations in air. Assuming that this is true, discuss the impacts to the results of the HHRA with respect to VOCs.

Response

c) To address this comment, DCEL conservatively assumed that a doubling in the VOC emissions would result in a doubling of the ground level air concentrations at all locations. Also refer to SIR responses 11(a) and 11(b) above.

