APPENDIX 3.3:

HUMAN HEALTH RISK ASSESSMENT FOR THE SHELL PIERRE RIVER MINE UPDATE

PREPARED BY: INTRINSIK ENVIRONMENTAL SCIENCES

Table of Contents

List of Tables

List of Figures

List of Attachments

1.0 Introduction

The Environmental Impact Assessment (EIA) of Shell's Jackpine Mine Expansion (JME) and Pierre River Mine (PRM) was submitted to the Alberta Energy Resources Conservation Board (now Alberta Energy Regulator [AER]) and Alberta Environment and Sustainable Resource Development (ESRD) in December 2007. The Human Health Risk Assessment (HHRA) and Wildlife Health Risk Assessment (WHRA) for JME and PRM were presented in EIA, Volume 3, Sections 5.3 and 5.4 and are referred to in this appendix as the EIA HHRA and EIA WHRA, respectively.

This appendix and Attachment A provide additional information to the EIA HHRA and EIA WHRA, specifically as it relates to the potential effects associated with the PRM alone, as requested in the Joint Review Panel (JRP) Supplemental Information Request (SIR) 5 under Methods. In this JRP SIR, the JRP notes that "*In order to determine the significance of effects from the Pierre River Mine Project, the Panel requires information on effects of the Pierre River Mine Project only, without inclusion of the Jackpine Mine Expansion Project … [and in order] … to determine the effects of only the Pierre River Mine Project, the environmental consequences should be calculated for each Key Indicator Resource (KIR) using the effects within the Pierre River Mine LSA or some such reasonable spatial area as determined and rationalized by the proponent.*"

Further, in JRP SIR 8 under Methods, the JRP notes that "*additional projects and activities have been disclosed and/or occurred since the cumulative effects assessment was completed* [in the EIA]*, and thus an update is required to account for these projects.*"

The HHRA and WHRA describe the potential short-term (acute) and long-term (chronic) health risks to people and wildlife associated with chemical emissions from the PRM and in combination with existing, approved and planned regional developments. The updated assessment cases are referred to as the 2013 Base Case, 2013 PRM Application Case and 2013 PDC. In light of the distinct characteristics of the two assessments, the findings of the 2013 HHRA are presented herein (i.e., as part of the main appendix), while the findings of the 2013 WHRA are presented as an attachment to this appendix.

2.0 Assessment Methods Update

The impact assessment of the 2013 PRM Application Case and the 2013 PDC resulted in air and water quality predictions that differ from those that formed the basis of the EIA HHRA (i.e., EIA Volume 3, Section 5.3). As such, the health risks were re-examined using the updated air quality and water quality predictions.

Unless stated otherwise, the assessment methods used for the 2013 HHRA are consistent with those employed in the EIA HHRA. The HHRA followed a conventional risk assessment paradigm developed over time by such regulatory agencies as Health Canada, United States Environmental Protection Agency and the Canadian Council of Ministers of the Environment. This approach is also consistent with Alberta Health and Wellness' *Guidance on Human Health Risk Assessment for Environmental Impact Assessment in Alberta* (AHW 2011).

Certain aspects of the EIA HHRA have changed since the EIA was filed. Some key differences that were adopted in the 2013 HHRA include:

- Description of the existing conditions (Section 2.1), including exposure and health studies;
- Problem Formulation (Section 2.2), including changes to the final list of chemicals of potential concern and revisions to the consumption rates;
- Exposure assessment (Section 2.3), including a re-evaluation of how the physical-chemical characteristics were used to identify the non-volatile, potentially bioaccumulative chemicals; and
- Toxicity assessment (Section 2.4), including an update of any new health-based exposure limits.

These sections of the HHRA are described further below.

2.1 Existing Conditions

The existing conditions related to health were described in the EIA, Volume 3, Section 5.2.9.

The PRM will be situated in an area that was once part of the former Northern Lights Health Region (NLHR) (i.e., prior to the amalgamation of the provincial health regions under the auspices of the 'single' Alberta Health Services system). The NLHR spanned Alberta's border with the North West Territories, extending south just beyond Rainbow Lake in the west and past Conklin in the east. Although the NLHR is considerably larger than the PRM HHRA study area, information on the overall health status in the region can still be used in a broad sense as many of the communities that fall inside the NLHR are comparable to those in the vicinity of the PRM.

It is important that the data concerning the current health status in the region (i.e., baseline health data) be interpreted with a certain degree of caution. Baseline health data are publicly available on a relatively broad scale, wherein information is presented for the primary urban centres or for large geographic areas. No specific baseline health data are currently publicly available for the community nearest to the PRM (i.e., Fort McKay). Although some of the information presented below may not be specifically relevant to residents inside the HHRA study area, it can still be useful for "identifying critical receptors as well as in interpreting the HHRA in the context of population baseline, project and cumulative risks" (AHW 2011).

2.1.1 Exposure and Health Effects Studies

The Alberta Oil Sands Community Exposure and Health Effects Assessment Program (AOSCEHEAP) was a joint industry, government and community initiative that was established to investigate possible links between air quality and human health outcomes in the Fort McMurray region (AHW 2000). Results from this program showed that:

- Chemical air concentrations were generally low in the Fort McMurray region, compared to air quality guidelines, regardless of whether they were measured indoors or outdoors.
- Air concentrations were not significantly different in Fort McMurray compared to the reference location (Lethbridge, AB), despite the high degree of oil and gas development in the Fort McMurray region.

- No significant differences in health status were found between the Fort McMurray and Lethbridge regarding physician visits or prevalence of disease.

The AOSCEHEAP report and the more recent information from the Wood Buffalo Environmental Association (WBEA) suggest the following (WBEA 2007):

- Nitrogen dioxide (NO_2) concentrations were low compared to air quality guidelines, although levels have increased since the AHW (2000) study. Indoor concentrations were lower than outdoor concentrations. The most important exposure sources were local, suggesting that regional development has had little influence.
- Sulphur dioxide (SO_2) concentrations were low compared to air quality guidelines, and in general, outdoor air concentrations were similar to the AHW (2000) levels. Indoor concentrations were lower than outdoor levels. The most important exposure sources were determined to be local, followed by regional sources.
- Measured outdoor $PM_{2.5}$ air concentrations were less than the Canada-Wide Standard (CWS) of 30 µg/m³. However, $PM_{2.5}$ outdoor concentrations did not play an important role in personal exposure. The most important exposure source was personal activity and indoor sources.
- Ozone (O_3) indoor and personal concentrations were lower than the 1-hour Alberta Ambient Air Quality Objective (AAAQO) of 160 µg/m³ and 8-hour CWS of 125 µg/m³; outdoor ambient levels were an order of magnitude higher with the most important exposure source being naturally occurring background sources.
- Indoor concentrations were the predominant factor affecting personal exposure to volatile organic compounds (including but not limited to benzene, ethylbenzene, hexane, toluene and xylenes).

Kindzierski et al. (2010) conducted a trend analysis of air quality data between 1998 and 2007 from WBEA. Through their analysis, Kindzierski et al. (2010) concluded that “there is little or no pattern to the changes in concentrations of various air pollutants across the oil sands region over the past 10 years”. The authors did note increasing hourly concentrations of nitrogen oxides at the Fort McMurray Patricia McInnes and Fort McKay ambient monitoring stations. In contrast, decreasing hourly concentrations were observed for $PM_{2.5}$ at all of the community air monitoring stations (Fort McMurray, Fort McKay and Fort Chipewyan). No trends were apparent for any of the other chemicals.

Finally, the overall air quality at Fort Chipewyan appeared to be unique when compared to the other stations. According to Kindzierski et al. (2010), Fort Chipewyan appears to be far enough away from the oil sands development that “it is only slightly influenced by regional development and activity that is influencing, to varying degrees, many of the monitoring stations in the airshed.”

In February 2009, the Alberta Cancer Board (ACB) published a study on the incidence of cancer in the community of Fort Chipewyan. The study was completed in response to concerns from a local physician and the community that cancer rates appeared to be higher than expected in Fort Chipewyan. A cluster investigation was conducted based on the guidelines from the U.S. Centre for Disease Control and Prevention. Specifically, the purpose of the study was to determine if there was an elevated rate of

cholangiocarcinoma (a rare cancer of the bile duct) and whether there was an elevated rate of cancers overall in Fort Chipewyan based on data observed in the community from 1995 to 2006.

The overall findings of the ACB (2009) study are described as:

- incidence rates of cholangiocarcinoma were within the expected range;
- overall cancer rate was higher in Fort Chipewyan than expected;
- cancers of the blood and lymphatic system, biliary tract and soft tissue were higher than expected; and
- colon and lung cancer rates were within expected ranges.

The ACB indicated that the increased rates were based on a small number of cases and could be due to chance, increased detection or increased risk in the community.

The ACB indicated that further investigation would be required to determine whether the difference between the observed and expected cancer rates was due to chance or if there was an actual increased risk associated with living in Fort Chipewyan. The study was not designed to determine the cause of any of the cancers experienced in Fort Chipewyan. Further analysis was recommended by the ACB to determine whether risk factors such as lifestyle, family history, occupational exposures and/or environmental exposures are contributing to the observed cancer incidence.

In 2009, Alberta Health and Wellness conducted a health risk assessment of mercury in fish collected as part of the Regional Aquatics Monitoring Program (RAMP) in the oil sands (AHW 2009a). As part of the health risk assessment, Alberta Health and Wellness investigated the concentrations of mercury in various fish species collected from the water bodies of the RAMP area and, in turn, characterized the potential health risks associated with these concentrations. In addition, Alberta Health and Wellness discussed the overall benefits of fish consumption. The existing advisory to restrict or limit the consumption of walleye, northern pike and whitefish from certain lakes and rivers in the RAMP area was confirmed by the findings of the health risk assessment. The results of the study further indicated that concentrations of mercury in fish in the water bodies of the RAMP area were within the ranges for the same fish species from other water bodies in Alberta, Canada or the United States. As such, the health risks posed to the residents of the oil sands region do not appear to be higher than those posed to individuals who eat fish from other parts of the country.

In their 2010 Royal Society of Canada report on the oil sands, the expert panel concluded that “there is currently no credible evidence of environmental contaminant exposures from oil sands reaching Fort Chipewyan at levels expected to cause elevated human cancer rates” (RSCEP 2010).

In 2012, Kevin Percy, the executive director of WBEA, along with a number of other scientists authored a book titled, “Alberta Oil Sands: Energy, Industry and the Environment”. In the book, the authors summarize the results of WBEA’s 2008-2012 air monitoring network in the Athabasca Oil Sands Region and describe the significant environmental indicators of air quality and the terrestrial environment in the region (WBEA 2013). A chapter within the book, titled, “Air Quality in the Athabasca Oil Sands Region 2011”, reports that AAAQOs were not exceeded for criteria air contaminants including SO_2, NO_2,

and CO in 2011. However, during a severe forest fire event in May and June of 2011 near Fort McKay and Fort McMurray, $PM_{2.5}$ and O_3 concentrations exceeded their respective 24-h AAAQO and 1-h AAAQO. Even so, air quality remained at a low risk to health based on the reported hourly air quality hazard index (AQHI) calculated by ESRD using WBEA continuous monitoring data from four community stations. Results indicate that 96% to 99.3% of the time the AQHI values were considered in the low risk to health category, while 0.18% to 2.1% of the time the AQHI values were in the moderate risk category. The hours categorized in the high or very high risk categories occurred during the most intense smoke periods. According to model predictions, air quality is most significantly affected within 20 km of industrial emissions sources. These effects on air quality are rapidly reduced thereafter (Percy 2012).

A more recent study by Kurek et al. (2013) suggested that PAH concentrations in sediment in six lakes north of Fort McMurray may be linked to oil sands developments. Canadian interim sediment quality guidelines (CISQGs) were exceeded for PAHs in one of the six lakes; however, in general, PAH concentrations remained similar to other remote lakes and much lower than those of urbanized catchments.

Researchers at the University of Calgary are scheduled to lead a health study funded in part by the provincial and federal governments to examine incidences of cancer as well as other health issues in the First Nations' oil sands communities. Further information detailing potential links between oil sands developments and human health effects is expected to result from this study.

2.1.2 General Health Indicators

In addition to the quality of a person's ambient environment (e.g., air, water, etc.), many other factors play a role in determining a person's overall health. Collectively, these factors are referred to as health determinants and include such things as income and social status, social support networks, education, employment and working conditions, physical environment, biology and genetics, personal health practices and coping skills and access to health services, to name a few.

Research shows that Canadians in rural, remote and northern communities generally have a lower health status relative to other Canadians (CPHI 2006). This applies across a number of indicators, including lifestyle related illnesses, injuries and cardiovascular diseases.

The recent Royal Society of Canada report on the oil sands further indicates that the health status in the Northern Lights Health Region is worse than the provincial average for several non-environmental indicators, such as substance-related disorders, sexually transmitted infections, prevalence of diabetes, and mortality due to homicide as well as mortality rates due to motor vehicle collisions. The report also highlights the fact that the Northern Lights Health Region has the lowest availability of doctors. These indicators are typical of what the Royal Society of Canada report refers to as a "boomtown effect" (RSCEP 2010).

2.1.3 Cancer and Respiratory Disease

Mortality data for the most common cancers and respiratory diseases are available from the Government of Alberta's Interactive Health Data Application (IHDA) (AHW 2013). Alberta Health and

Wellness has designed this site to provide information on health status and determinants of health. The IHDA contains health indicators derived from various sources, in topics such as demographics, mortality, chronic and infectious disease, and children's health (AHW 2013).

The mortality rates (per 100,000 population) for lung, colorectal, breast and prostate cancer for both males and females in the NLHR are presented as three year rolling averages from 2000 to 2011 (see Table 2-1).

Table 2-1 Mortality Cancer Rates per 100,000 Population in the Northern Lights Health Region During Three-Year Periods From 2000 to 2011 (Provincial Averages are in Parentheses)

Years	Lung				Colorectal				Breast		Prostate	
	Female		Male		Female		Male		Female		Male	
2000 to 2002	43.2 (32.5)	=	59.7 (50.3)	=	16.9 (11.9)	=	16.2 (19.0)	=	29.3 (23.5)	=	22.6 (29.2)	=
2001 to 2003	43.7 (32.3)	=	56.6 (51.0)	=	13.9 (12.2)	=	13.8 (18.6)	<<	26.7 (24.2)	=	21.1 (28.5)	=
2002 to 2004	40.0 (33.6)	=	47.8 (51.2)	=	16.1 (12.5)	=	7.6 (18.9)	<<	19.3 (22.4)	=	42.3 (27.6)	+
2003 to 2005	38.1 (33.3)	=	48.6 (50.5)	=	22.8 (12.7)	+	6.2 (19.9)	=	12.6 (21.6)	<	60.6 (26.4)	++
2004 to 2006	44.6 (33.5)	=	57.6 (48.1)	=	30.7 (12.6)	+	20.7 (19.9)	=	8.3 (20.4)	<<	50.9 (25.0)	+
2005 to 2007	41.8 (33.2)	=	73.6 (47.4)	+	23.1 (12.4)	+	18.6 (18.8)	=	6.1 (20.6)	<<	42.3 (24.2)	+
2006 to 2008	38.9 (33.6)	=	66.4 (45.1)	+	12.0 (11.8)	=	17.8 (18.6)	=	2.3 (20.0)	<<	21.1 (23.5)	=
2007 to 2009	31.9 (33.3)	=	54.9 (45.5)	=	1.6 (11.6)	<<	16.6 (19.4)	=	9.9 (19.6)	<	18.8 (22.8)	=
2008 to 2010	26.5 (33.2)	=	52.2 (43.6)	=	3.0 (11.7)	<<	24.0 (19.3)	=	19.1 (18.7)	=	10.0 (22.4)	<<
2009 to 2011	20.9 (32.8)	<	58.6 (42.5)	+	8.7 (11.7)	=	23.3 (18.9)	=	18.5 (18.3)	=	15.9 (21.0)	=

Legend to statistical significance ratings (AHW 2013):

++	Regional mortality rate is significantly higher than the provincial average
+	Regional mortality rate is slightly higher than the provincial average
=	Regional mortality rate is similar to provincial average
<	Regional mortality rate is slightly lower than the provincial average
<<	Regional mortality rate is significantly lower than the provincial average

Much like the cancer mortality rates, additional asthma and Chronic Obstructive Pulmonary Disease (COPD) mortality data are available from the Alberta IHDA from 2000 until 2011 (AHW 2012). The mortality rates (per 100,000 population) for asthma and COPD in the NLHR are presented as three year rolling averages from 2000 to 2011 (see Table 2-2).

The extent to which the PRM and other industrial sources in the region will influence a number of these health indices will be addressed through the findings of this HHRA.

Table 2-2 Mortality Rates per 100,000 Population for COPD and Asthma in the Northern Lights Health Region During Three-Year Periods From 2000 to 2011 (Provincial Average are Presented in Parentheses)

Year	COPD Mortality		Asthma Mortality	
2000 to 2002	45.9(26.5)	+	0.4(0.7)	=
2001 to 2003	51.4(25.9)	++	0.4(0.7)	=
2002 to 2004	42.7(25.2)	+	0.4(0.7)	=
2003 to 2005	41.5(24.8)	+	0(0.7)	=
2004 to 2006	43.6(24.8)	++	0(0.6)	=
2005 to 2007	46.2(25.2)	++	0(0.6)	=
2006 to 2008	39.4(25.4)	+	0(0.6)	=
2007 to 2009	29.1(24.9)	=	0(0.6)	=
2008 to 2010	26.0(23.9)	=	0.3(0.5)	=
2009 to 2011	29.8(23.5)	=	0.3(0.5)	=

Legend to the statistical significance ratings (AHW 2013):

++	Regional ASMR significantly higher than provincial average
+	Regional ASMR slightly higher than provincial average
=	Regional ASMR similar to provincial average
<	Regional ASMR slightly lower than provincial average
<<	Regional ASMR significantly lower than provincial average

Note: ASMR = age standardized mortality rate.

2.2 Problem Formulation

The Problem Formulation of the HHRA was presented in EIA, Volume 3, Section 5.3.2.1.

The Problem Formulation is the planning stage of the HHRA and lends focus to the assessment through the identification of the Chemicals of Potential Concern (COPC) associated with the PRM, the characterization of people potentially 'at risk' in the region, and the confirmation of the relevant exposure pathways. Since filing the EIA, certain aspects of the Problem Formulation have changed, as described in the following sections.

2.2.1 Identification of the Chemicals of Potential Concern

Consistent with AHW's guidance on conducting HHRAs in Alberta (AHW 2011), the 2013 HHRA considered only those chemicals expected to be emitted from the PRM. As such, the 2013 HHRA differs from the EIA HHRA in that the 2013 HHRA did not evaluate chlorinated volatile organic compounds, as these chemicals are not expected to be emitted from the PRM. It is important to note that once COPC for the PRM were identified, other, non-PRM-related sources of the same COPC were included in the estimates of exposure to ensure that cumulative health risks were adequately characterized in the 2013 HHRA.

Specifically, the chemicals in the air quality emissions inventory that were excluded from the 2013 HHRA are as follows:

- Twenty-two VOCs:
  - chlorinated VOCs include: 1,1,1,2-tetrachloroethane; 1,1,1-trichloroethane; 1,1,2,2-tetrachloroethane; 1,1,2-trichloroethane, 1,1-dichloroethane; 1,2-dichloroethane; 1,2-dichloropropane; 1,3-dichloropropene; 1-chloronaphthalene; 2-chloronaphthalene; carbon tetrachloride; chlorobenzene; chloroethane; chloroform; methylene chloride; vinyl chloride
  - other VOCs: biphenyls; ethylene dibromide; isopropanol group; methanol; phenol; styrene
- Three metals: antimony; strontium; tin
- One sulphur containing compound: thiophene group

Table 2-3 provides the list of COPC to be carried forward in the 2013 PRM HHRA. Note that the naming of COPC from the air emissions profile may be different than the water emissions profile. For example, "acenaphthenes/acenaphthylenes" in the air emissions profile is referred to as PAH group 4 in the water emissions profile. In some instances the chemical constituents which make up the group may vary slightly.

Table 2-3 Chemicals of Potential Concern Assessed in the 2013 PRM Human Health Risk Assessment

Chemical Category	COPC Identified from Air Emissions Profile	COPC Identified from Water Emissions Profile
CACs	CO	—
	NO_2	—
	$PM_{2.5}$	—
	SO_2	—
Organic Compounds	1,3-Butadiene	—
	1-Pentene	—
	Acenaphthenes/Acenapthylenes	PAH group 4
	Acetaldehyde	—
	Acetone	—
	Acrolein	—
	Aliphatic aldehydes group	—
	Aliphatic C_5-C_8 group	—
	Aliphatic C_9-C_{16} group	—
	Aliphatic C_{17}-C_{34} group	—
	Anthracene/phenanthrenes and substituted	PAH group 5
	Aromatic C_9-C_{16} group	—
	Benzene	—
	—	PAH group 6[(1)]
	Carcinogenic PAH group 1	PAH group 1
	Carcinogenic PAH group 2	PAH group 2
	Carcinogenic PAH group 3	PAH group 3
	Cumene	—
	Cyclohexane	—
	Dichlorobenzene	—

Table 2-3 Chemicals of Potential Concern Assessed in the 2013 PRM Human Health Risk Assessment (continued)

Chemical Category	COPC Identified from Air Emissions Profile	COPC Identified from Water Emissions Profile
Organic Compounds (continued)	Ethylacetylene	—
	Ethylbenzene	—
	Ethylene	—
	Fluorenes/Fluoranthenes and substituted	PAH group 7
	Formaldehyde	—
	Hexane	—
	Methyl ethyl ketone group	—
	Naphthalene and substituted naphthalenes	PAH group 8
	—	Phenol
	Propylene	—
	Propylene oxide	—
	Pyrenes and substituted pyrenes	PAH group 9
	Toluene	—
	Trimethylbenzenes	—
	Xylenes	—
Metals and Minerals	Aluminum	Aluminum
	—	Antimony
	Arsenic	Arsenic
	Barium	Barium
	Beryllium	Beryllium
	—	Boron
	Cadmium	Cadmium
	Chromium	Chromium
	Cobalt	Cobalt
	Copper	Copper
	Lead	Lead
	—	Lithium
	Manganese	Manganese
	Mercury	Mercury
	Molybdenum	Molybdenum
	Nickel	Nickel
	Selenium	Selenium
	Silver	Silver
	—	Strontium
	—	Thallium
	—	Uranium
	Vanadium	Vanadium
	Zinc	Zinc
Sulphur Compounds	1-Hexanethiol	—
	COS	—
	CS_2 group	—
	H_2S	—
Other	—	Ammonia
	—	Naphthenic acids

Notes:
(1) PAH group 6 is assessed as the biphenyl group.
— = not applicable.

2.2.2 Characterization of People Potentially at Risk

The people potentially at risk were described in EIA, Volume 3, Section 5.3.2.1, pages 5-36 to 5-47, and in greater detail in EIA, Volume 3, Appendix 3-12.

People potentially at risk include sensitive or susceptible individuals who receive the highest exposure to the PRM emissions. In this regard, consideration was given to:

- Those people who are known or anticipated to spend time near the PRM;
- The lifestyles and physical characteristics of the individuals in the area; and
- The sensitivity or susceptibility of individuals in the area (e.g., children, elderly, individuals with compromised health).

In the EIA HHRA, people were assigned to one of the following 'lifestyle' categories:

- Transients: included all occasional or seasonal visitors to the area, in recognition of the notion that people may use the area in the immediate vicinity of the PRM for recreational or traditional activities such as hunting, trapping and plant gathering. As such, the HHRA included an assessment of potential health risks to people at the location where the maximum ground-level air concentrations attributable to the PRM were predicted to occur;
- Cabin residents: included people who use the cabins located near the PRM as temporary residences while engaged in traditional activities;
- Aboriginal residents: included all permanent First Nations and Métis or subsistence residents of the neighbouring communities;
- Community residents: included all permanent non-subsistence residents of the neighbouring communities; and
- Workers: included all workers who reside at nearby industrial camp sites, including Shell's workers who may live on-site at the Jackpine Mine Camp and the Pierre River Village throughout the course of their employment.

With the exception of the PRM maximum fenceline location, where people are expected to spend only short periods of time, these assumptions were applied to all the discrete receptor locations in the 2013 HHRA. In other words, no special distinction was made between the cabin dwellers and the community residents, as the HHRA now assumes that everyone in the region could engage in a subsistence lifestyle wherein they would receive all of their dietary requirements from local food sources. Therefore, the PRM maximum fenceline location, cabins, communities and industrial camp sites were assigned to the following three 'receptor' groups:

- Transients: includes the locations alongside the PRM fenceline where the maximum ground-level air concentrations attributable to the PRM are predicted to occur. The health risks for the single (1) location in this group are based on short-term exposures only.
- Aboriginals: includes all locations other than the industrial camp sites. Individuals within this group are assumed to use the cabins on a permanent basis. As well, this group is assumed to be actively engaged in traditional use of the neighbouring lands. The HHRA assumed that all residents of the

area were part of the Aboriginal group. The health risks for the 23 locations in this group are based on both short-term and long-term exposures.

- Workers: includes all industrial camp locations and worksites, wherein workers are assumed to reside throughout the terms of their employment. The health risks for the four (4) locations in this group are based on both short-term and long-term exposures.

Although people would likely only occupy the cabins during traditional or recreational activities, the actual time spent at these locations could not be definitively determined. As such, it was conservatively assumed that people would maintain permanent residency at the cabins and other locations for their entire lifetimes.

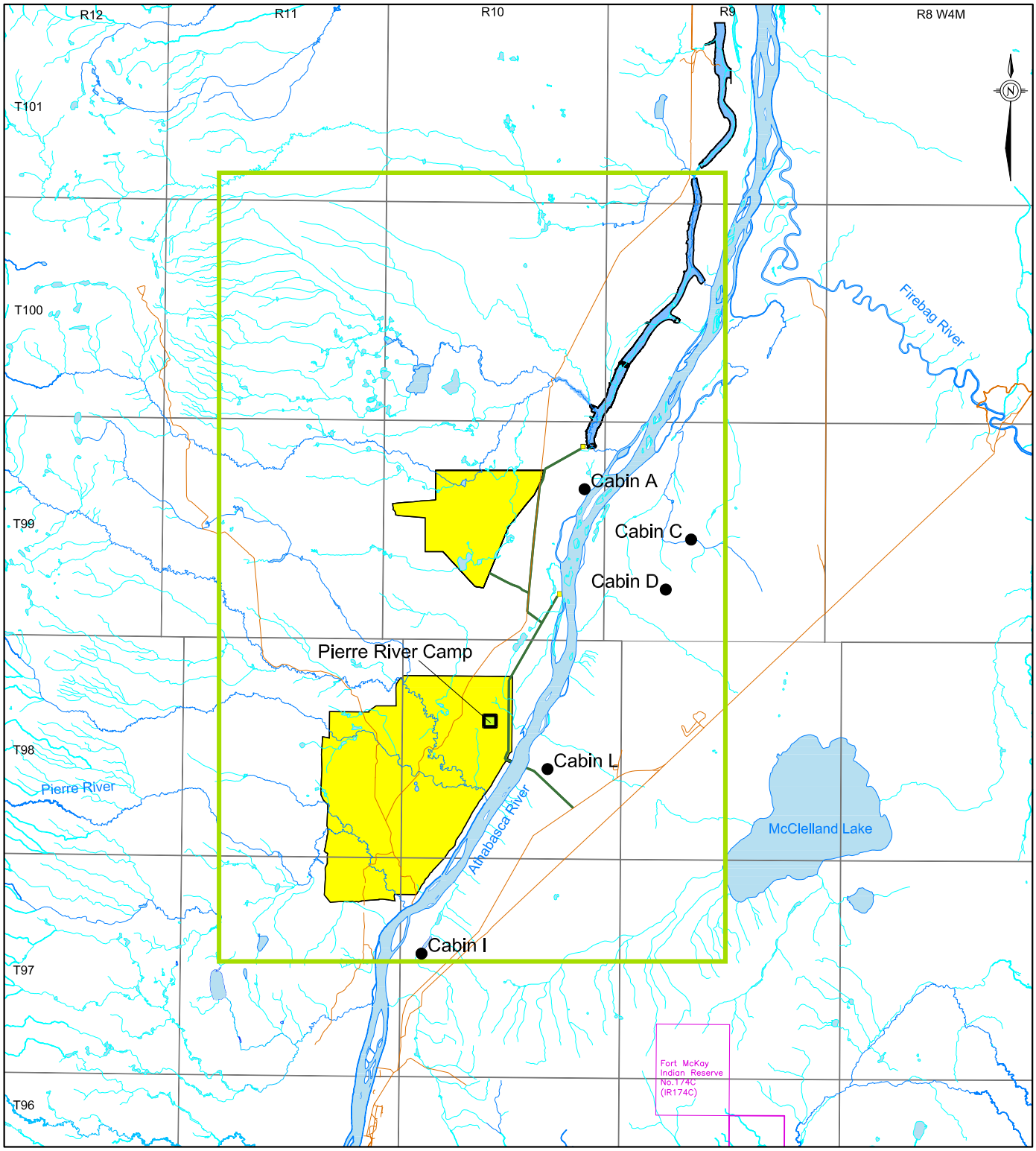
All receptor locations that were assessed in the EIA HHRA are being assessed in the 2013 HHRA with the exception of the maximum along the JME fenceline. In addition, the lifestyle category is slightly rearranged and assumptions of the lifestyle categories are different than those used in the EIA HHRA. Table 2-4 presents a comparison of the receptor groups in the EIA HHRA and in the 2013 HHRA.

Table 2-4 Lifestyle Categories and Corresponding Receptor Locations for the EIA HHRA and the 2013 HHRA

EIA HHRA			2013 HHRA		
Lifestyle Category	**Count**	**Location**	**Lifestyle Category**	**Count**	**Location**
Transient	2	Maximum along JME fenceline; Maximum along PRM fenceline	Transient	1	Maximum along PRM fenceline
Cabin resident	12	Cabin A, Cabin B, Cabin C, Cabin D, Cabin E, Cabin F, Cabin G, Cabin H, Cabin I, Cabin J, Cabin K, Cabin L	Cabin resident[(1)]	12	Cabin A, Cabin B, Cabin C, Cabin D, Cabin E, Cabin F, Cabin G, Cabin H, Cabin I, Cabin J, Cabin K, Cabin L
Aboriginal resident	11	Anzac, Clearwater (IR175), Conklin, Descharme Lake (SK), Fort Chipewyan, Fort McKay, Fort McMurray, Janvier/Chard (IR194), La Loche (SK), Namur River (IR174A), Poplar Point (IR201G)	Community resident[(1)]	11	Anzac, Clearwater (IR175), Conklin, Descharme Lake (SK), Fort Chipewyan, Fort McKay, Fort McMurray, Janvier/Chard (IR194), La Loche (SK), Namur River (IR174A), Poplar Point (IR201G)
Community resident	7	Anzac, Conklin, Descharme Lake, Fort Chipewyan, Fort McKay, Fort McMurray, La Loche	Community resident[(2)]	n/a	n/a
Worker	4	Jackpine Mine camp, Pierre River Mine camp, Oil Sands Lodge, PTI camp	Worker	4	Jackpine Mine camp, Pierre River Mine camp, Oil Sands Lodge, PTI camp

Notes:
[(1)] In the 2013 HHRA, Cabin resident and Community resident were assessed as part of the Aboriginal Group.
[(2)] The community receptor locations are assessed as part of the Aboriginal Group in the 2013 HHRA.
n/a = not applicable.

Twelve cabins and four worker camps were identified near to the PRM, many of which are located within the LSA where maximum Project-related changes in environmental quality are expected to occur (Figure 2-1). Eleven communities within the RSA are presented in Figure 2-2.



LEGEND

- RIVER OR STREAM
- PUBLIC ROADWAY
- PIERRE RIVER MINING AREA
- AIR LOCAL STUDY AREA
- CABIN
- WORKER CAMP

5 0 5
SCALE KILOMETRES

REFERENCE

ALBERTA DIGITAL DATA OBTAINED FROM ALTALIS LTD. (SEPTEMBER 2004.) USED UNDER LICENSE.
PROJECTION: TRANSVERSE MERCATOR DATUM: NAD 83 COORDINATE SYSTEM: UTM ZONE 12

PROJECT

PIERRE RIVER MINE PROJECT

TITLE

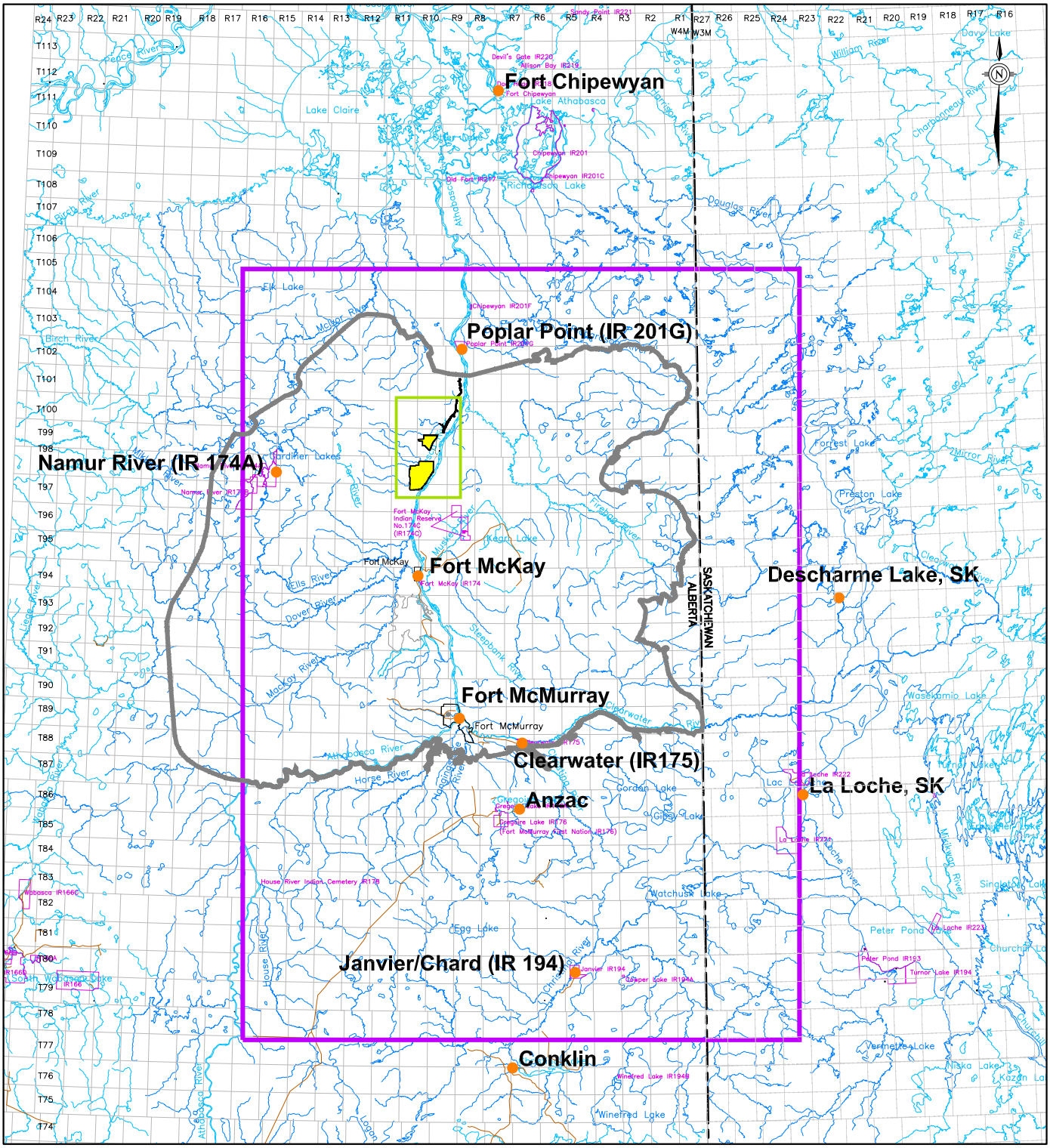
LOCATIONS WITHIN THE LOCAL STUDY AREA AT WHICH PEOPLE RESIDE OR VISIT

Shell Canada Limited

PROJECT	13.1346.0001.6105		FILE No.	134600016105A003
DESIGN	MA	22 Aug. 2013	SCALE AS SHOWN	REV. 0
CADD	PSR	22 Aug. 2013		
CHECK	MA	22 Aug. 2013		
REVIEW	WES	22 Aug. 2013		

FIGURE: 2-1

L:\2013\1346\13-1346-0001\6100\6105\Report_A\ Drawing file: Fig5.3-1_134600016105A003_LSA-PRV.dwg Jul 03, 2013 - 3:19pm



LEGEND

- RIVER OR STREAM
- PUBLIC ROADWAY
- PIERRE RIVER MINING AREA
- AIR LOCAL STUDY AREA
- AIR REGIONAL STUDY AREA
- TERRESTRIAL RESOURCES AIR EMISSIONS EFFECTS STUDY AREA
- REGIONAL COMMUNITIES

REFERENCE

ALBERTA NTDB DATA OBTAINED FROM GEOMATICS CANADA, AUGUST 2001. NAD 83 ZONE 12. SHEETS 74D, E AND 74L IN NAD 27 ZONE 12. SASKATCHEWAN NTDB DATA OBTAINED FROM ISC, AUG. 2001. NAD 83 ZONE 13. ALL DATA CONVERTED TO NAD 83 UTM ZONE 12.

PROJECT

PIERRE RIVER MINE PROJECT

TITLE

COMMUNITIES WITHIN THE REGIONAL STUDY AREA AT WHICH PEOPLE RESIDE

Shell Canada Limited

PROJECT	13.1346.0001.6105		FILE No.	1346000161105A002	
DESIGN	MA	22 Aug. 2013	SCALE	AS SHOWN	REV. 0
CADD	PSR	22 Aug. 2013	FIGURE: 2-2		
CHECK	MA	22 Aug. 2013			
REVIEW	WES	22 Aug. 2013			

L:\2013\1346\13-1346-0001\6100\6105\Report_A\Fig2-2_1313460001610SA002_CRSAPR.dwg Oct 02, 2013 - 3:55pm

Physical Characteristics of People in the Region

Potentially long-term exposed individuals residing in the LSA may be exposed through multiple pathways. All age classes (life stages) were considered in a multiple pathway exposure assessment. The five receptor life stages that were included in the HHRA are consistent with Health Canada guidance (Health Canada 2010a):

- Infant (0 to 6 months = 0.5 years);
- Toddler (7 months to 4 years = 4.5 years);
- Child (5 to 11 years = 7 years);
- Adolescent (12 to 19 years = 8 years);
- Adult (20 to 80 years = 60 years).

For the assessment of carcinogens, a 'composite individual' who represents all life stages (e.g., from infant to adult) was used to represent cumulative exposure over an 80-year lifetime.

General physical characteristics of potentially chronically exposed people residing in the LSA were obtained from Health Canada (2010a). General characteristics that were common to all individuals are summarized in Table 2-5.

Table 2-5 General Physical Characteristics Assumed for the Multiple Pathway Exposure Assessment

Physical Characteristic[(1)]	Life Stage (Health Canada 2010a)					
	Infant	Toddler	Child	Adolescent	Adult	Adult Worker
Inhalation rate (m^3/d)	2.2	8.3	14.5	15.6	16.6	33.6
Soil ingestion rate (g/d)	0.02	0.08	0.02	0.02	0.02	0.1
Water ingestion rate (L/d)	0.3	0.6	0.8	1.0	1.5	n/a
Body Weight (kg)	8.2	16.5	32.9	59.7	70.7	70.7
Lifetime Adjustment Factor (i.e., for carcinogenic exposures)	0.0063	0.056	0.088	0.1	0.75	0.75
Arms and legs body surface area (cm^2)	1,460	2,580	4,550	7,200	8,220	8,220
Hand surface area (cm^2)	320	430	590	800	890	890
Total surface area (cm^2)	3,620	6,130	10,140	15,470	17,640	17,640
Soil adherence factor – hands only (g/cm^2/d)	0.0001	0.0001	0.0001	0.0001	0.0001	0.001
Soil adherence factor – other than hands (g/cm^2/d)	0.00001	0.00001	0.00001	0.00001	0.00001	0.0001

Notes:
(1) Food consumption rates for the Aboriginal group are described in the sections below.
n/a = not applicable. The adult worker is not assumed to be drinking water from the local surface water.

It was assumed for the purpose of the 2013 HHRA that Aboriginal residents would:

- Be present at any given location 24 hours per day, 365 days per year over an 80-year lifespan. This includes the cabin and community locations.
- Practice a subsistence lifestyle, such that all traditional and non-traditional foods would be obtained from local sources.
- Get all their drinking water from local (surface) waterbodies.
- Swim in local water bodies during the summer months.

The consumption rates of traditional and non-traditional foods assumed for the Aboriginal group are presented in Table 2-6. The differences between the consumption rates used for the 2013 HHRA and those applied to the EIA HHRA are due to the current assumption that the area residents will get all of their food from local sources (e.g., fish, game, fruit and vegetables).

Table 2-6 Food Consumption Rates for the Aboriginal Group

Physical Characteristics	Consumption Rate [grams/day]					Source
	Infant	Toddler	Child	Teen	Adult	
Moose	0	65	95	133	206	Health Canada 2010a; Wein et al. 1989
Snowshoe hare	0	14	20	28	43	Health Canada 2010a; Wein et al. 1989
Ruffed grouse	0	7	10	14	21	Health Canada 2010a; Wein et al. 1989
Fish	0	20	33	40	40	Health Canada 2007
Wild mint or Labrador tea leaves	0	1	1	3	3	Wein 1989; Wein et al. 1991
Cattail roots	0	1	1	3	3	Wein 1989; Wein et al. 1991
Garden root vegetables	0	105	161	227	188	Health Canada 2010a
Garden leafy vegetables	0	67	98	120	137	Health Canada 2010a
Fruits, including wild berries	0	5	11	19	23	Wein 1989
Breast milk	664	0	0	0	0	O'Connor and Richardson 1997

Assumed consumption rates for wild game were based on Health Canada's food ingestion rates for Canadian Aboriginal populations (Health Canada 2010a) in combination with the frequency of consumption reported for native Canadians near Wood Buffalo National Park (Wein et al. 1989). Food consumption patterns were obtained by repeated 24-hour food recall surveys: two surveys were completed between late August and mid-November 1986; and, two surveys were completed between late April and mid-July 1987. One hundred and seventy-eight individuals over 12 years of age were interviewed. Large mammals constituted 76% of the wild game consumed by the 120 native households interviewed, small mammals constituted 16%, and upland birds 8%. For example, using Health Canada's (2010a) adult ingestion rate of 270 grams per day of wild game, it was assumed that adult residents would consume 205 grams of moose per day (270 grams/day × 0.76).

With respect to fish consumption rates for the HHRA, Health Canada (2007) assumes an adult subsistence consumption rate of 40 grams of fish per day for a 'heavy consumer'. This value was obtained from a Market Facts of Canada (1991) study on national seafood consumption and a Bureau of Chemical Safety (BCS) evaluation of current intake rates by Canadian consumers (BCS 2004). The BCS

study considered the information provided in multiple studies and recommended subsistence consumption rates that took into consideration sport, subsistence and Aboriginal fish eaters. Similar fish consumption rates have been reported in the 1997 diet and activity survey conducted in Swan Hills by Alberta Health and Wellness where the 'medium consumer' was reported to ingest 47 grams of fish per day (AHW 1997), and in a 1999 survey conducted by Health Canada of an Aboriginal population in the Lesser Slave Lake region of Alberta where a moderate consumer was reported to consume 46 grams of fish per day on average (AHW 2009b). Due to the lack of data of fish consumption for children, Health Canada assumes that children will have the same consumption frequency as adults but portion sizes would be smaller. The portion size for a toddler and child is assumed to be 50% and 83% of the adult's portion size respectively. The fish consumption rates for the toddler and child 'heavy consumer' are 20 grams of fish per day (40 g/day × 50%) and 33 grams of fish per day (40 g/day × 83%), respectively (Health Canada 2007). For the purpose of the HHRA, it was assumed that teens would consume the same amount of fish as an adult.

Plant and vegetable consumption rates were segregated into traditional aboveground plants (e.g., wild mint and Labrador tea leaves) and belowground plants (e.g., cattail root), as well as garden aboveground vegetables (e.g., lettuce) and belowground vegetables (e.g., potatoes). Wein (1989) reported a consumption rate of 134 grams per day, which was adjusted by the frequency of 2% (i.e., 7 days in 365 days) at which wild mint and Labrador tea leaves were reportedly consumed in the Aboriginal households interviewed (Wein et al. 1991). From this, an adult consumption rate of 3 grams per day was assumed for traditional aboveground plants (e.g., wild mint and Labrador tea leaves). Wein et al. (1991) reported that wild roots were seldom eaten in native households that were interviewed and thus did not provide consumption data for wild roots. As a result, it was assumed for the HHRA that the consumption rates for traditional belowground and aboveground plants were equivalent (i.e., 3 g/day).

Health Canada provided vegetable (root and other) ingestion rates for the Canadian general population based on 24-hour recall data collected in 1970 and 1972 as part of the Nutrition Canada Survey (Health Canada 1994, 2010a). The dietary survey involved a statistically representative sample of the Canadian population, personal interviews conducted by trained interviewers, and physical models of meal portions to assist in determining food portion sizes for some 180 different foods. Summary data were provided by Health Canada for vegetable (root and other) 'eaters only', which exclude individuals reporting no vegetable consumption. Using statistics for 'eaters only' ensures that the consumption rates of the individuals who consume the majority of the vegetables were not under estimated. Health Canada's vegetable ingestion rates were assumed to assess potential health risks associated with the consumption of garden vegetables.

The fruit or berry consumption rates were based on information presented in Wein (1989) that estimated adult populations in the area consumed 23 grams of berries per day. Fruit or berry consumption rates for earlier life stages were based on the body weight ratios. For example, the child fruit consumption rate is based on a ratio of the child to adult body weight (i.e., 0.47 = 32.9 kg/70.7 kg) multiplied by the adult consumption rate (i.e., 0.47 × 23 g/d = 11 g/d). The plant consumption rates assumed for Aboriginal peoples over all life stages are listed in Table 2-6.

Infant consumption rates for solid foods (i.e., fruit and vegetables, fish, game meat) were assumed to be zero for the purposes of the HHRA. The infant consumption rates for aboveground and belowground produce presented in the Health Canada (2010a) guidance are based on O'Connor and Richardson (1997), which is based on data obtained in a Nutrition Canada Survey (NCS) between 1970 and 1972. This information may not be reflective of current infant food consumption data. For example, a study by Health Canada in 1994 (Health Canada 1994a) states the following:

> "In Canada, infant feeding practices have changed dramatically over the last 30 years (Tanaka et al. 1987; Health and Welfare Canada 1991). Recent studies indicate that a majority of Canadian mothers breast-feed; breast-feeding initiation rates are close to 80%, with 30% still breast-feeding their infants after 6 months. The intake of breast milk peaks between 4 to 6 months of age. Solid foods are introduced to approximately 50% of infants by 4 months of age, and 89.5% by 6 months of age. To reflect these practices, estimation of total daily intake is generally based on the assumption that a typical infant is exclusively breast-fed up to 6 months of age, after which foods are consumed in the quantities determined in the NCS."

As such, the assumption applied in the HHRA that infants rely upon breast milk or a similar alternative for the first six months of life appears to be reasonable. This is further supported by the infant consumption rates for game and fish meat of zero in the most recent Health Canada (2010a) guidance document. Therefore, the HHRA assumed that infant consumption rates for berries, vegetables, traditional plants, game meat and fish were zero. Therefore, infant consumption relied solely on breast milk.

The workers were represented by discrete locations corresponding to the four camp sites. It was assumed that these camp sites would be occupied by adult workers only. Although workers would likely only reside at the camps during their years of employment, it was conservatively assumed that they would maintain permanent residency at the housing complexes over their entire adult life (assumed to be 60 years, that is ages 20 through 80) (Health Canada 2010a). It was further assumed that workers would obtain all of their food and water from the camps, which in turn would obtain all food from off-site (commercial) sources. Consistent with the EIA HHRA, the consumption of local foods was not considered for the worker group in the 2013 HHRA.

2.2.3 Identification of Exposure Pathways

The identification of exposure pathways was described in EIA, Volume 3, Section 5.3.2.1, pages 5-47 to 5-48. The exposure pathways selected for the 2013 HHRA were consistent with those originally presented, with the following exceptions:

- Ingestion of municipal drinking water was excluded for the worker group in the 2013 HHRA; and
- Dermal (skin) contact with soil was included for the worker group in the 2013 HHRA.

A summary of the exposure pathways assessed for the different groups is shown in Table 2-7.

Figures 2-3 and 2-4 present the conceptual model of exposure pathways for the Aboriginal receptor and the Worker receptor, respectively.

Table 2-7 Exposure Pathways Assessed in the 2013 Human Health Risk Assessment

Exposure Pathway	Receptor Groups		
	Transient	Aboriginal	Worker
Inhalation			
Inhalation of air	✓	✓	✓
Inhalation of dust	×	✓	✓
Ingestion			
Ingestion of soil (inadvertent)	×	✓	✓
Ingestion of water[(1)]	×	✓	×
Ingestion of local country foods (fruits and vegetables)	×	✓	×
Ingestion of local natural foods (berries, cattails and tea leaves)	×	✓	×
Ingestion of local fish	×	✓	×
Ingestion of local game	×	✓	×
Dermal contact			
Dermal contact with soil	×	✓	✓
Dermal contact while swimming	×	✓	×

Notes:
(1) Includes drinking surface water and incidental ingestion while swimming.
✓ Exposure pathway is applicable to the receptor group.
× Exposure pathway is not applicable to the receptor group.

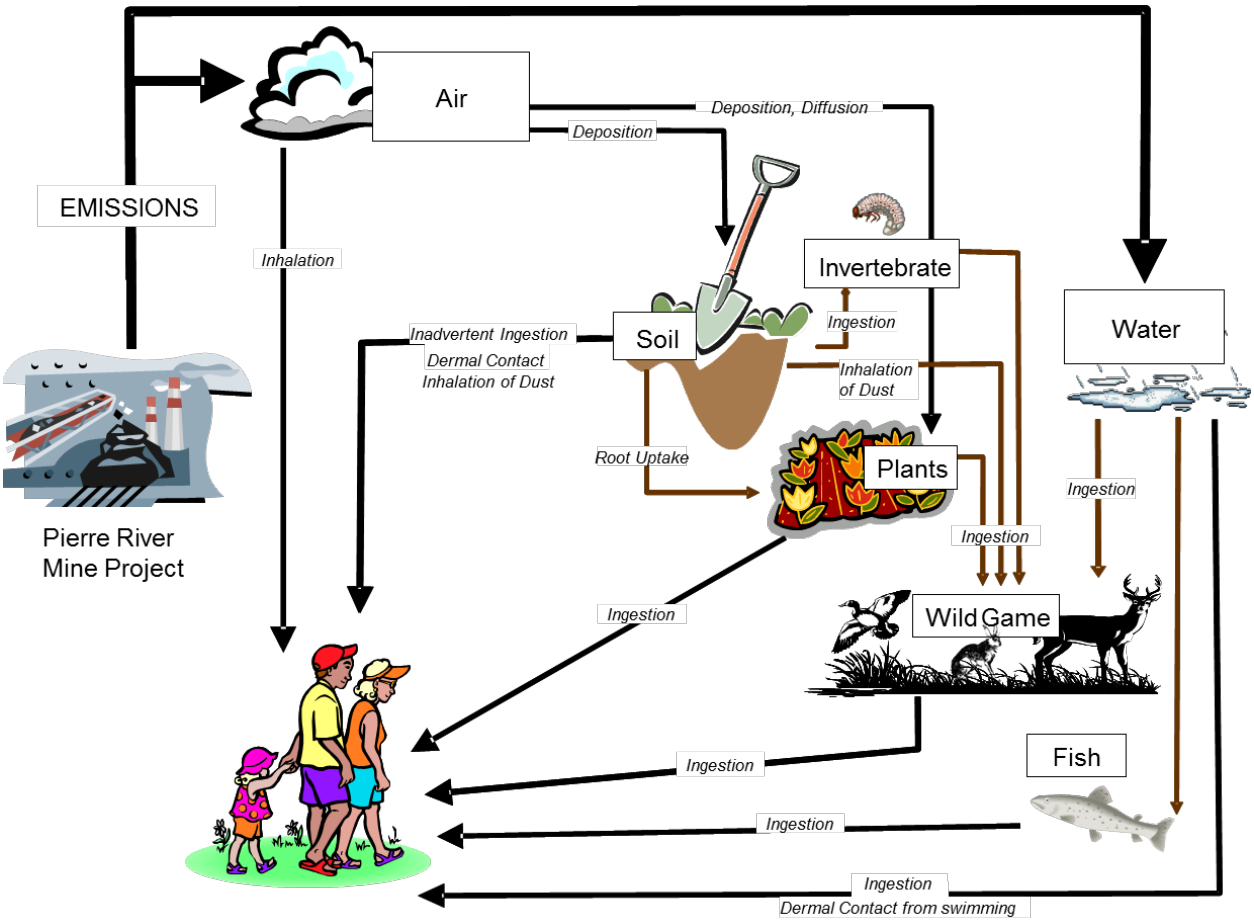


Figure 2-3 Conceptual Model of Exposure Pathways for the Aboriginal Group

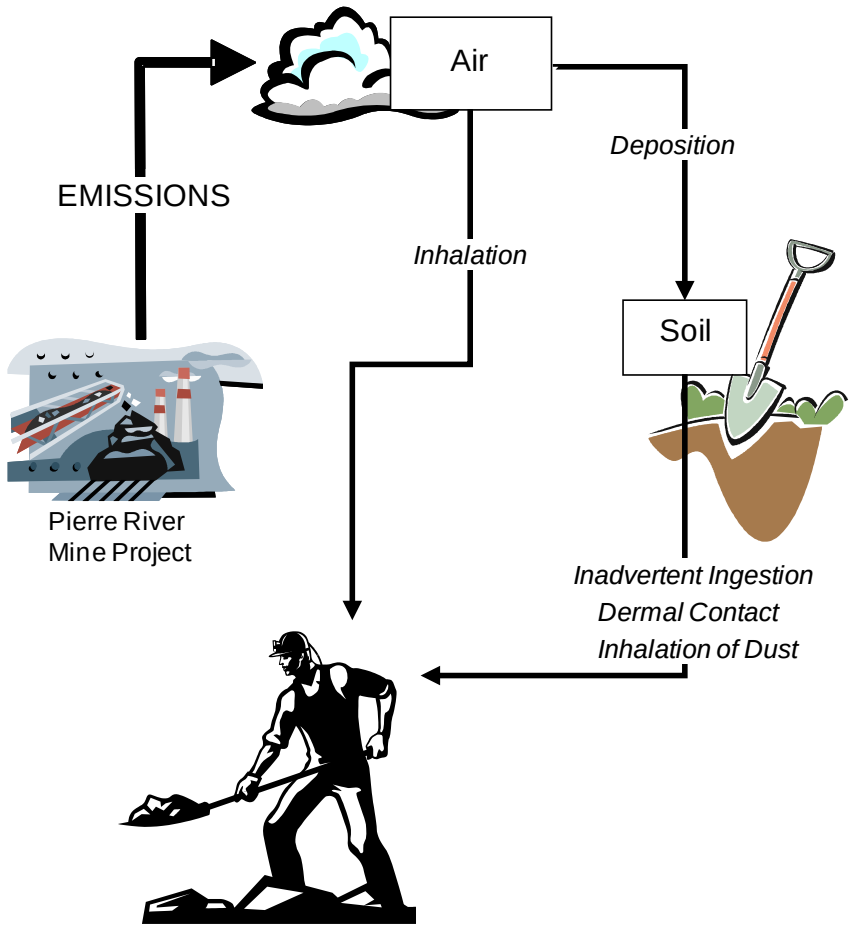


Figure 2-4 Conceptual Model of Exposure Pathways for the Worker Group

2.3 Exposure Assessment

The Exposure Assessment was described in EIA, Volume 3, Section 5.3.2.2.

For the most part, the approach for the Exposure Assessment of the 2013 HHRA remains consistent with what was presented in the EIA HHRA. The 2013 HHRA does differ, however, from the original Exposure Assessment in how it identifies those chemicals emitted by PRM that, although emitted into air, could potentially be deposited nearby and possibly persist or accumulate sufficient quantities for people to be exposed via secondary routes of exposure related to soil, food and water pathways. Another notable difference from the EIA HHRA is the exclusion of indoor air quality from the 2013 HHRA.

2.3.1 Inhalation Exposure Assessment

The 2013 HHRA did not make an adjustment for indoor air quality in the Inhalation Exposure Assessment. The exclusion of indoor air concentrations from the 2013 HHRA stems from information that arose out of the SIR process, wherein the uncertainty related to the inclusion of indoor air quality was highlighted. The EIA HHRA assumed that people would spend most of their time indoors (Volume 3,

Section 5.3). This assumption was questioned by AESRD in their SIRs on the EIA HHRA. As a result, further analysis was conducted based on the assumption that the Aboriginal receptor group would spend all of their time outdoors (see PRM SIR Round 1, Volume 2: SIR 77; SIR 78; SIR 82; SIR 89; SIR 90; SIR 91; SIR 102). Consistent with this, indoor air concentrations were excluded from the current assessment.

All other aspects of the Inhalation Exposure Assessment are consistent with those described in the EIA HHRA.

Predicted air concentrations were based on air dispersion modelling results described in the Air Quality Assessment (Appendix 3.2). The modelling results consisted of the predicted ground-level air concentrations for each chemical emitted from the PRM that could occur at the discrete receptor locations identified in Table 2.4.

Predicted air concentrations were estimated for various averaging periods. Acute averaging periods consisted of 1-hour or 24-hour averaging periods for all COPC and 10-minute averaging period for sulphur dioxide and 8-hour averaging period for carbon monoxide. The various acute averaging periods for COPC were provided in order to evaluate the correct exposure concentration averaging period with the averaging period associated with the acute inhalation exposure limits. On an acute basis, the peak (1^{st} highest) 10-minute, 1-hour, 8-hour, and 24-hour ground-level air concentrations were used to evaluate potential acute health risks. However, a unique averaging period was provided for $PM_{2.5}$ using the 98^{th} percentile (8^{th} highest) of 24-hour concentrations based on CCME guidance (CCME 2000).

Chronic averaging period was based on annual averages for all COPC. Chronic health risks were assessed using the maximum annual ground-level air concentrations.

2.3.2 Multiple Pathway Exposure Assessment

In order to assess the potential health risks associated with possible secondary exposure pathways, it was necessary to identify those chemicals emitted by PRM that, although only emitted into air, would be expected to deposit in the vicinity and possibly persist or accumulate in the environment in sufficient quantities for people to be exposed via soil, food and water pathways. For this purpose, two categories of chemicals emitted from PRM were identified:

- Gaseous chemicals, which are unlikely to contribute to human exposure via secondary pathways (e.g., CO, NO_2, SO_2, and H_2S). In addition, the health effects of these gaseous chemicals are strictly related to inhalation (i.e., these compounds act at the point of contact). Accordingly, the gaseous chemicals were removed from further consideration in the multiple pathway assessment and only evaluated in the inhalation assessment.
- Non-gaseous chemicals, which may deposit in the vicinity of the PRM, and persist or accumulate in the environment in sufficient quantities for residents and workers to be exposed via secondary pathways (e.g., metals, organic compounds, and sulphur compounds [with the exception of H_2S]). The potential occurrence of these non-gaseous chemicals in the secondary pathways of exposure required further consideration.

To identify the non-gaseous chemicals that could deposit nearby and possibly persist or accumulate in the environment, consideration was given to the intrinsic properties of the chemicals that influence their fate and persistence in the environment, and subsequently their potential occurrence in the secondary pathways of exposure. This was accomplished via the process outlined below. Due to their non-volatile, persistent and accumulative nature, metals emitted by the PRM were automatically examined in both the inhalation and multiple pathway assessments.

Comparison of Physical-Chemical Properties with Established Criteria for Volatility. The purpose of this step is to identify the chemicals emitted by PRM that are non-volatile and thus have the potential to accumulate in environmental media other than air, in accordance with the following criteria from the US EPA (2003):

- molecular weight ≥200 g/mol (or 2.0E+02 g/mol)
- Henry's Law Constant ≤0.00001 atm-m³/mol (or 1.0E-05 atm-m³/mol)
- vapour pressure ≤0.001 mmHg (or 1.0E-03 mmHg)

Comparison of Physical-Chemical Properties with Established Criteria for Bioaccumulation. The purpose of this step is to identify the chemicals emitted by the PRM that have the potential to accumulate in living organisms, in accordance with the following criterion from Environment Canada (2007):

- octanol-water partitioning coefficient (Log K_{ow}) ≥5

Fugacity Modelling. Fugacity modelling was completed to determine the potential relative apportionment of the chemicals emitted by PRM in environmental compartments other than air, and subsequently the chemicals' potential occurrence in the secondary pathways of exposure. Fugacity model results were based on the 'Level III' fugacity model developed by the US EPA (2011a) that adheres to methods developed by MacKay et al. (1992, 1993). If a chemical was found to partition in soil, water or sediment more than 5%, there may be a 'realistic presence' of the chemical in environmental media other than air (Boethling et al. 2009; Environment Canada 2003).

Physical-chemical properties (i.e., molecular weight, Henry's Law Constant, vapour pressure, and octanol-water partitioning coefficient) were adopted from Syracuse Research Corporation (SRC 2011). If a physical-chemical property was not available from SRC (2011), the EPI Suite program developed by the US EPA (2011a) was searched. For the aliphatic and aromatic hydrocarbon groups, however, physical-chemical properties were sourced from CCME (2008), whenever possible.

The premise of this exercise is that if a chemical emitted to the air does not meet any of these criteria, the potential for the chemical to deposit in the vicinity of PRM and persist or accumulate in the environment is negligible, and only limited opportunity exists for exposure via secondary pathways. Accordingly, these chemicals were removed from further consideration in the multiple pathway assessment and only evaluated in the inhalation assessment. However, if a chemical meets any one of these criteria, sufficient opportunity could be presented for exposure via secondary pathways, and the chemical was evaluated in both the inhalation and multiple pathway assessments.

Table 2-8 summarizes the relevant physical-chemical properties and fugacity model results for each of the chemicals released from PRM, and identifies those chemicals to be included in the multiple pathway assessment. The findings of the exercise indicate 17 chemicals were identified to be carried forward into the multiple pathway assessment in addition to the 17 metals automatically included in the multiple pathway screening from the air emissions inventory for a total of 34 chemicals (or chemical groups) eligible for inclusion in the multiple pathway assessment, provided that defensible exposure limits are available.

Table 2-8 Identification of Chemicals of Potential Concern for the Multiple Exposure Pathway Assessment

Chemical[1][2][3]	CAS #	Volatility[4]			Bioaccumulation	Fugacity			Included in the Multiple Pathway Assessment?
		Molecular Weight (g/mol)	Henry's Law Constant (atm-m³/mol)	Vapour Pressure (mm Hg)	Log Kow	Soil (%)	Water (%)	Sediment (%)	
CRITERIA:		≥2.0E+02	≤1.0E-05	≤1.0E-03	≥5.0	≥5	≥5	≥5	
Organic Compounds									
1,3-Butadiene	106-99-0	5.4E+01	7.4E-02	2.1E+03	2.0	0.01	0.03	0.0001	No
1-Pentene	109-67-1	7.0E+01	4.0E-01	6.4E+02	2.7	0.006	0.005	0.00002	No
Acenaphthenes / acenaphthylenes (methyl acenaphthene)	58548-38-2	1.7E+02	3.8E-04	2.1E-03	4.6	**10**	**11**	**5**	**Yes**
Acetaldehyde	75-07-0	4.4E+01	6.7E-05	9.0E+02	-0.34	2	**10**	0.02	**Yes**
Acetone	67-64-1	5.8E+01	4.0E-05	2.3E+02	-0.24	3	**12**	0.02	**Yes**
Acrolein	107-02-8	5.6E+01	1.2E-04	2.7E+02	-0.01	1	**8**	0.02	**Yes**
Aliphatic aldehydes (crotonaldehyde)	4170-30-3	7.0E+01	**9.7E-06**	3.0E+01	0.6	**6**	**14**	0.026	**Yes**
Aliphatic C_5-C_8 group	n/a	9.1E+01	9.9E-01	1.6E+02	3.8	0.008	0.002	0.00002	No
Aliphatic C_9-C_{16} group	n/a	1.6E+02	5.7E+00	1.8E+00	5.7	0.03	0.0004	0.0001	**Yes**
Aliphatic C_{17}-C_{34} group	n/a	**2.7E+02**	1.2E+02	**8.4E-04**	**6.9**	4	0.08	2	**Yes**
Anthracene/phenanthrenes and substituted (phenanthrene)	85-01-8	1.8E+02	4.2E-05	**1.2E-04**	4.5	**40**	**9**	**12**	**Yes**
Aromatic C_9-C_{16} group	n/a	1.3E+02	5.3E-03	1.77+00	3.6	2	0.5	0.05	No
Benzene	71-43-2	7.8E+01	5.6E-03	9.5E+01	2.1	0.3	0.5	0.005	No
Carcinogenic PAH group 1 (7,12-Dimethylbenz(a)anthracene)	57-97-6	**2.6E+02**	**3.8E-06**	**6.8E-07**	**5.8**	**80**	0.5	**18**	**Yes**
Carcinogenic PAH group 2 (indeno(1,2,3-cd)	193-39-5	**2.8E+02**	**3.5E-07**	**1.3E-10**	**6.7**	**80**	0.5	**19**	**Yes**
Carcinogenic PAH group 3 (benzo(g,h,i)perylene)	191-24-2	**2.8E+02**	**3.3E-07**	**1.0E-10**	**6.6**	**80**	0.5	**19**	**Yes**
Cumene	98-82-8	1.2E+02	1.2E-02	4.5E+00	3.7	0.5	0.2	0.005	No
Cyclohexane	110-82-7	8.4E+01	1.5E-01	9.7E+01	3.4	0.01	0.02	0.0001	No
Dichlorobenzenes (1,2-dichlorobenzene)	95-50-1	1.5E+02	1.9E-03	1.4E+00	3.4	2	1	0.03	No
Ethylacetylene	107-00-6	5.4E+01	2.1E-02	1.4E+03	1.5	0.03	0.1	0.0004	No
Ethylbenzene	100-41-4	1.1E+02	8.0E-03	1.0E+01	3.2	0.5	0.3	0.005	No
Ethylene	74-85-1	2.8E+01	2.3E-01	5.2E+04	1.1	0.005	0.01	0.00002	No
Fluorenes/fluoranthenes and substituted (fluoranthene)	206-44-0	**2.0E+02**	**8.9E-06**	**9.2E-06**	**5.2**	**65**	4	**18**	**Yes**
Formaldehyde	50-00-0	3.0E+01	**3.0E-07**	3.9E+03	0.35	**61**	**23**	0.04	**Yes**
Hexane	110-54-3	8.6E+01	1.8E+00	1.5E+02	3.9	0.004	0.001	0.000006	No
Methyl ethyl ketone group	78-93-3	7.2E+01	5.7E-05	9.1E+01	0.3	2	**11**	0.02	**Yes**

Table 2-8 Identification of Chemicals of Potential Concern for the Multiple Exposure Pathway Assessment (continued)

Chemical[1][2][3]	CAS #	Volatility[4]			Bioaccumulation	Fugacity			Included in the Multiple Pathway Assessment?
		Molecular Weight (g/mol)	Henry's Law Constant (atm-m³/mol)	Vapour Pressure (mm Hg)	Log Kow	Soil (%)	Water (%)	Sediment (%)	
CRITERIA:		≥2.0E+02	≤1.0E-05	≤1.0E-03	≥5.0	≥5	≥5	≥5	
Naphthalene and substituted naphthalenes (indole)	120-72-9	1.2E+02	**5.3E-07**	1.2E-02	2.1	**78**	**6**	0.2	**Yes**
Propylene	115-07-1	4.2E+01	2.0E-01	8.7E+03	1.8	0.005	0.01	0.00003	No
Propylene oxide	75-56-9	5.8E+01	7.0E-05	5.4E+02	0.03	2	**10**	0.02	**Yes**
Pyrenes and substituted pyrenes (picene)	213-46-7	**2.8E+02**	**4.9E-07**	**3.7E-09**	**7.1**	**80**	0.5	**19**	**Yes**
Toluene	108-88-3	9.2E+01	7.0E-03	2.8E+01	2.7	0.3	0.3	0.003	No
Trimethylbenzene (1,2,3-trimethylbenzene)	526-73-8	1.2E+02	4.4E-03	1.7E+00	3.7	1	0.6	0.02	No
Xylenes (o-xylene)	95-47-6	1.1E+02	5.2E-03	6.6E+00	3.1	0.6	0.4	0.007	No
Sulphur Compounds									
1-Hexanethiol	111-31-9	1.2E+02	1.1E-02	4.2E+00	3.2	0.2	0.2	0.002	No
COS	463-58-1	6.0E+01	6.1E-01	9.4E+03	-1.3	0.004	0.004	0.000007	No
Carbon disulphide group	75-15-0	7.6E+01	1.4E-02	3.6E+02	1.9	0.03	0.2	0.0004	No

Notes:

-**Bold** values indicate that the physical-chemical parameter meets or exceeds the pre-established criterion, and the chemical is eligible for inclusion in the multiple pathway assessment, provided that defensible exposure limits are available.

-Physical-chemical parameters for all COPC were obtained from the following sources in the order of priority: SRC (2011), US EPA (2011a) (i.e. EPISuite). The exception is for aliphatic and aromatic hydrocarbons where physical-chemical parameters were obtained from CCME (2008), Table B.1.

n/a = not applicable.

[1] CACs and H_2S were not included in the physical-chemical screening as these chemicals predominantly exist in air and therefore they strictly relate to inhalation exposures. Metals were not included in the physical-chemical screening because metals were automatically included in the multiple exposure pathway assessment.

[2] $PM_{2.5}$ was excluded from the screening as it is a mixture for which the physical-chemical properties and fugacity are not known.

[3] Chemicals in parentheses represent the chemical constituent within the group that was used as the surrogate chemical for the physical-chemical screening

[4] With scientific notation, values are written are expressed either to the negative power (i.e., E-x) or to the positive power (i.e., E+x). For example, molecular weight for 1,3-butadiene is 5.4E+01 which is equivalent to 54.

In addition to the 34 chemicals identified for inclusion in the multiple pathway assessment from the air emissions inventory, several other chemicals or chemical groups where added into the multiple pathways for the following reasons:

- Although the aromatic C_9-C_{16} group did not screen on based on physical-chemical parameters identified from CCME (2008) for the group as a whole, some of the chemical constituents within that group screen on as an individual (for example, acenaphthenes/acenaphthylenes is a constituent of the aromatic C_9-C_{16} group but screens on for fugacity modeling);
- Aromatic C_{17}-C_{34} group was created in order to assess those chemical constituents that fall into this hydrocarbon group for the assessment of chemicals for multiple pathway exposure;
- COPC identified from the water emissions inventory were automatically included in the multiple pathway assessment. These chemicals were identified previously in Table 2-3. Many of the chemicals emitted to water were also identified in the air emissions inventory. However, there are 10 chemicals emitted to water that were not emitted into air: ammonia, antimony, biphenyls, boron, lithium, naphthenic acids, phenol, thallium, strontium and uranium;
- Chromium VI is not a COPC identified in the air emissions profile, however, chromium VI can be formed from chromium in the ambient air;
- Methyl mercury is not a COPC identified in the air emissions profile, however, mercury can be bio-transformed to methyl mercury in the aquatic environment.

The final list of 48 chemicals assessed through multiple pathways of exposure is presented in Table 2-9.

Table 2-9 Chemicals Selected for Inclusion in the Multiple Pathway Assessment

Chemical Category	Chemical
Organic Compounds	Acetaldehyde
	Acenaphthenes/Acenaphthylenes
	Acetone
	Acrolein
	Aliphatic aldehydes
	Aliphatic C_9-C_{16} group
	Aliphatic C_{17}-C_{34} group
	Anthracene/phenanthrenes and substituted
	Aromatic C_9-C_{16} group[(1)]
	Aromatic C_{17}-C_{34} group[(2)]
	Biphenyls[(3)]
	Carcinogenic PAH group 1
	Carcinogenic PAH group 2
	Carcinogenic PAH group 3
	Fluorenes/fluoranthenes and substituted
	Formaldehyde
	Methyl ethyl ketone group
	Naphthalene
	Phenol[(3)]
	Propylene oxide
	Pyrene

Table 2-9 Chemicals Selected for Inclusion in the Multiple Pathway Assessment (continued)

Chemical Category	Chemical
Metals and minerals	Aluminum
	Antimony[3]
	Arsenic
	Barium
	Beryllium
	Boron[3]
	Cadmium
	Chromium
	Chromium VI[4]
	Cobalt
	Copper
	Lead
	Lithium[3]
	Manganese
	Mercury
	Methyl mercury[5]
	Molybdenum
	Nickel
	Selenium
	Silver
	Strontium[3]
	Thallium[3]
	Uranium[3]
	Vanadium
	Zinc
Other	Ammonia[3]
	Naphthenic acids[3]

Notes:

[1] The aromatic C_9-C_{16} group did not screen on based on physical-chemical parameters from CCME (2008). However chemical constituents within this group screened on and therefore the aromatic C_9-C_{16} group was carried forward into the multiple pathway assessment.

[2] The aromatic C_{17}-C_{34} group was created for those chemical constituents that are non-carcinogenic PAHs.

[3] These COPC are predicted to emit into the water from the PRM and are automatically included in the multiple pathway assessment but were not identified in the air emissions inventory. All COPC emitted into water was automatically evaluated in the multiple pathway assessment.

[4] Potential of conversion of chromium total or chromium III to chromium VI may be possible in air or surface water. Therefore it was assumed that chromium VI was a COPC in the multiple pathway assessment. It was assumed that chromium VI makes up 8.3% of total chromium concentrations in soil and plants (Fengxiang et al. 2004), and 100% in water (Government of Canada 1994).

[5] Although the Project will not emit methyl mercury directly to the environment, the release of inorganic mercury into surface water may cause bio-transformation to methyl mercury. On this basis, methyl mercury, in addition to inorganic mercury, was identified as a COPC in the multiple pathway assessment of the HHRA.

Table 2-9, which presents the chemicals identified for inclusion in the multiple pathway assessment, differs slightly than the one identified in the EIA HHRA in that:

- The 2013 HHRA did not include the following chemicals identified in the air emissions inventory in the physical-chemical screening because these chemicals were not emitted into air by PRM: chlorinated volatile organic compounds, biphenyls, ethylene dibromide, isopropanol group, methanol, phenol, styrene, antimony, strontium, tin, and thiophene group. However, biphenyls,

phenol, antimony, and strontium were included in the multiple pathway assessment because these chemicals were identified in the water emissions inventory;
- Due to the changes in how the physical-chemical characteristics were screened, the 2013 HHRA list includes acetaldehyde, acrolein, aliphatic aldehydes, formaldehyde, methyl ethyl ketone group, propylene oxide and pyrene, which the EIA HHRA did not;
- The 2013 HHRA does not include the aliphatic C_5-C_8 group or aromatic C_9-C_{16} group, which the EIA HHRA did.

The differences between the lists of COPC included in the multiple pathway assessment are due to the updated approach that was used for the physical-chemical screening in the 2013 HHRA.

2.4 Toxicity Assessment

The Toxicity Assessment was described in EIA, Volume 3, Section 5.3.2.3.

The health-based exposure limits presented in the EIA HHRA were derived from a number of different regulatory agencies. These agencies review their limits on a periodic basis. Therefore, with the passage of time, some of the exposure limits used in the EIA HHRA have since changed. As a result, the 2013 HHRA had to re-evaluate the available regulatory exposure limits to ensure the Toxicity Assessment was up to date. Other than the update of the exposure limits and the resultant changes to the chemical mixtures assessment, all other aspects of the Toxicity Assessment remain the same.

2.4.1 Exposure Limits Used in the Human Health Risk Assessment

For the purpose of the 2013 HHRA, reliance was placed on exposure limits developed or recommended by regulatory or reputable scientific agencies as criteria (e.g., objectives, guidelines or standards) for the protection of human health. The 2013 HHRA exposure limits were obtained from:

- Alberta Environment and Sustainable Resource Development (ESRD)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- American Conference of Governmental Industrial Hygienists (ACGIH)
- Canadian Council of Ministers of the Environment (CCME)
- Health Canada and Environment Canada
- Netherlands National Institute of Public Health and the Environment (RIVM)
- California's Office of Environmental Health Hazard Assessment (OEHHA)
- Ontario Ministry of the Environment (OMOE)
- Texas Commission of Environmental Quality (TCEQ)
- United States Environmental Protection Agency (US EPA)
- World Health Organization (WHO)

For inclusion in the 2013 HHRA, exposure limits were required to be:

- protective of the health of the general public based on current scientific knowledge of the health effects associated with exposure to the COPC;

- protective of sensitive individuals (i.e., children and the elderly) through the incorporation of uncertainty or safety factors;
- established or recommended by reputable scientific or regulatory authorities; and
- supported by adequate documentation.

When these criteria were satisfied by more than one objective, guideline or standard, the most scientifically defensible exposure limit was typically selected.

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 exposure limits on the basis of duration of exposure. The two exposure limit durations used in the HHRA can be described as follows:

- Acute Exposure Limit: the amount or dose of a chemical that can be tolerated without evidence of adverse health effects on a short-term basis. These limits are routinely applied to conditions in which exposures extend over several hours or several days only.
- Chronic Exposure Limit: the amount of a chemical that is expected to be without effect, even when exposure occurs continuously or regularly over extended periods, lasting for periods of at least a year, and possibly extending over an entire lifetime.

In the chronic assessment, further distinction must be made between the exposure limits developed for the primary inhalation pathway and the secondary exposure pathways.

Most of the chemicals identified in the air emissions inventory were evaluated either as individual chemicals (e.g., benzene) or as chemical constituents within a pre-defined chemical group (e.g., carcinogenic PAH group). Additionally, several of the chemicals were assessed both as an individual chemical (e.g., hexane) and as part of an aliphatic or aromatic group (e.g., in this case, the aliphatic C_5-C_8 group). In these instances, the exposure limit identified for the individual chemical was lower (i.e., more conservative) than the exposure limit for the aliphatic or aromatic group as a whole.

In the EIA HHRA, the three carcinogenic PAH groups were assessed individually. For the 2013 HHRA, the three groups were assessed together as benzo(a)pyrene equivalents and evaluated using two distinct approaches:

- For the first approach (Approach 1), a mixture of carcinogenic PAHs was evaluated based on its benzo(a)pyrene content. The use of benzo(a)pyrene as an indicator of the potency of the mixture is based on the World Health Organization's (WHO) review of air quality guidelines for PAHs (WHO 2000). Benzo(a)pyrene was chosen as the indicator PAH as its toxicity is best characterized out of all the carcinogenic PAH compounds.
- In the second approach (Approach 2), the mixture of carcinogenic PAHs was evaluated by summing each individual PAH's toxic equivalency to benzo(a)pyrene (i.e., the toxic equivalency quotient approach). The toxic equivalencies of the PAH groups were determined using Potency Equivalency Factors (PEF) that have been adopted by Health Canada (2010a). A search of chemical constituents within each carcinogenic PAH group was identified for PEF values from Health Canada (2010a).

Chemical constituents within each carcinogenic PAH group with the highest PEF value was used to represent the PEF for the group:

- Carcinogenic PAH group 1 was assigned a PEF of 10 based on 7,12-dimethylbenz(a)anthracene
- Carcinogenic PAH group 2 was assigned a PEF of 1.0 based on four PAHs with the highest PEF of 1.0 assigned to 5,9 and 5,11-dimethylchrysenes; 7-methylbenzo(a)anthracene; and 8-methylbenzo(a)anthracene
- Carcinogenic PAH group 3 was assigned a PEF of 0.01 based on benzo(g,h,i)perylene and chrysene
- Fluorene/fluoranthene group was assigned a PEF of 0.001 based on fluoranthene
- Anthracene/phenanthrene group was assigned a PEF of 0.01 based on four PAHs with the highest PEF assigned to 1,4-dimethylphenathrene; 2,9,10-trimethylanthracene; 2,3,9,10-trimethylanthracene; and 9,10-dimethylanthracene
- Pyrenes/substituted pyrenes group was assigned a PEF of 0.001 based on 2-methylfluoranthene

When compared to the approach taken in the EIA HHRA, the 'amalgamation' of the carcinogenic PAH groups in the 2013 HHRA is a more conservative way of assessing the potential carcinogenic PAHs.

Approach 2 is consistent with the relative potency approach described by the US EPA (2002), in which the carcinogenic potencies of PAHs are scaled to an index compound (benzo(a)pyrene) using toxic equivalency factors (which are analogous to PEFs) and then added together to calculate the total cancer risk for the mixture. This approach permits the evaluation of the mixture when limited data are available for most of the mixture components.

The health-based exposure limits used in the 2013 HHRA are presented in Table 2-10.

2.4.2 Chemical Mixtures

Given that chemical exposures rarely occur in isolation, the potential health effects associated with simultaneous exposures to the COPC were assessed in the EIA HHRA and the 2013 HHRA. In accordance with HHRA guidance from Health Canada, additive interactions were assumed (Health Canada 2010a). Additive interactions apply most readily to chemicals that are structurally similar, that act toxicologically through similar mechanisms or that affect the same target tissue in the body (i.e., share commonality in effect) (Health Canada 2010a).

The critical endpoints of the exposure limits used in the 2013 HHRA provided the basis for an individual chemical's inclusion in a chemical mixture. For example, the acute inhalation exposure limit for formaldehyde is based on its ability to cause eye and nasal irritation, thus formaldehyde was included in both the acute inhalation 'eye irritants' and 'nasal irritants' mixtures. For details concerning the critical effects of the chemicals included in each of the mixtures, see Table 2-10.

The chemical constituents of the mixtures are listed in Table 2-11. The original mixtures table was presented in EIA, Volume 3, Section 5.3, Table 5.3-13. Any differences between the chemical mixtures of the EIA HHRA and 2013 HHRA are due to changes in the critical endpoints that form the basis of the exposure limits of the COPC assigned to the various chemical mixtures.

Table 2-10 Exposure Limits for the Chemicals of Potential Concern

Chemical of Potential Concern [(1)]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
Criteria Air Contaminants												
CO	1-Hour	40,000	Hypoxia	US EPA	—	—	—	—	N/A	N/A	N/A	N/A
	8-Hour	10,000	Hypoxia	US EPA								
NO_2	1-Hour	300	Respiratory irritation	ESRD	RfC	100	Respiratory irritation	US EPA	N/A	N/A	N/A	N/A
$PM_{2.5}$	24-Hour	30	—	CCME	RfC	12	—	CARB	N/A	N/A	N/A	N/A
SO_2	10-Minute	500	Respiratory irritation	WHO	—	—	—	—	N/A	N/A	N/A	N/A
	1-Hour	450	Respiratory irritation	ESRD								
Organics												
1,3-Butadiene	24-Hour	15	Reproductive / developmental effects	US EPA	RsC	0.3	Leukemia	US EPA	N/A	N/A	N/A	N/A
					RfC	2	Reproductive / developmental effects	US EPA				
1-Pentene	See aliphatic C_5-C_8 group				See aliphatic C_5-C_8 group				N/A	N/A	N/A	N/A
Acenaphthenes / acenaphthylenes (acenaphthene)	See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group				RfD	60	Liver effects	US EPA
									See aromatic C_9-C_{16} group			
Acetaldehyde	1-Hour	470	Eye, nasal and respiratory irritation	OEHHA	RsC	17.2	Nasal tumours	HC	—	—	—	—
					RfC	390	Nasal irritation	HC				
Acetone	4-Hour	62,000	Neurological effects	ATSDR	RfC	31,000	Neurological effects	ATSDR	RfD	900	Immunological effects; kidney effects; reproductive / developmental effects	US EPA
Acrolein	1-Hour	2.5	Eye, nasal and respiratory irritation	OEHHA	RfC	0.35	Nasal irritation	OEHHA	RfD	0.5	—	US EPA
Aliphatic aldehyde group (methacrolein and crotonaldehyde)	1-Hour	53	Eye irritation	TCEQ	RfC	1.2	Eye and nasal irritation	TCEQ	RfD	1	Gastrointestinal effects	US EPA
Aliphatic C_2-C_4 group (ethylene and propylene)	4-Hour	570,000	Liver effects	TCEQ	RfC	3,000	Nasal irritation; kidney effects	OEHHA	N/A	N/A	N/A	N/A
Aliphatic C_5-C_8 group (pentane)	1-Hour	200,000	—	TCEQ	RfC	18,400	Neurological effects	CCME	N/A	N/A	N/A	N/A
Aliphatic C_9-C_{16} group	—	—	—	—	RfC	200	Neurological effects	MA DEP	RfD	100	Kidney effects; liver effects	TPHCWG
Aliphatic C_{17}-C_{34} group	—	—	—	—	—	—	—	—	RfD	2,000	Liver effects	TPHCWG

Table 2-10 Exposure Limits for the Chemicals of Potential Concern (continued)

Chemical of Potential Concern [1]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
Anthracene / phenanthrenes and substituted (anthracene)	See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group; benzo(a)pyrene group				RfD	300	—	US EPA
									See aromatic C_9-C_{16} group, aromatic C_{17}-C_{34} group; benzo(a)pyrene group			
Aromatic C_9-C_{16} group (naphthalene)	1-Hour	2,000	Eye irritation	ACGIH	RfC	50	Kidney effects; liver effects	MA DEP	RfD	40	Kidney effects; liver effects	TPHCWG
Aromatic C_{17}-C_{34} group	—	—	—	—	—	—	—	—	RfD	30	Kidney effects	TPHCWG
Benzene	1-Hour	580	Immunological effects	TCEQ	RsC	1.3	Leukemia	US EPA	N/A	N/A	N/A	N/A
					RfC	9.8	Hematological effects; immunological effects	ATSDR				
Benzo(a)pyrene group 2 (using approach 1)[2]	—	—	—	—	RsC	0.00012	Lung tumours	WHO	N/A	N/A	N/A	N/A
Benzo(a)pyrene group 2 (using approach 2) [2]	—	—	—	—	RsC	0.32	Lung tumours	HC	RsD	0.0014	Gastrointestinal tumours	USE PA
Biphenyl	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	50	Kidney effects	US EPA
Carcinogenic PAH group 1	—	—	—	—	See benzo(a)pyrene group				See aromatic C_{17}-C_{34} group; benzo(a)pyrene group			
Carcinogenic PAH group 2	—	—	—	—	See benzo(a)pyrene group				See aromatic C_{17}-C_{34} group; benzo(a)pyrene group			
Carcinogenic PAH group 3	—	—	—	—	See benzo(a)pyrene group				See aromatic C_{17}-C_{34} group; benzo(a)pyrene group			
Cumene	See aromatic C_9-C_{16} group				RfC	400	Adrenal gland effects; kidney effects	US EPA	N/A	N/A	N/A	N/A
					See aromatic C_9-C_{16} group							
Cyclohexane	See aliphatic C_5-C_8 group				RfC	6,000	Reproductive / developmental effects	US EPA	N/A	N/A	N/A	N/A
					See aliphatic C_5-C_8 group							
Dichlorobenzenes	1-Hour	3,000	Eye and nasal irritation	TCEQ	RfC	60	Nasal irritation	ATSDR	N/A	N/A	N/A	N/A
Ethylacetylene	See aliphatic C_2-C_4 group				See aliphatic C_2-C_4 group				N/A	N/A	N/A	N/A
Ethylbenzene	1-Hour	21,700	Neurological effects	ATSDR	RfC	260	Kidney effects	ATSDR	N/A	N/A	N/A	N/A
Ethylene	See aliphatic C_2-C_4 group				See aliphatic C_2-C_4 group				N/A	N/A	N/A	N/A
Fluorenes/fluoranthenes and substituted (fluorene)	See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group; benzo(a)pyrene group				RfD	40	Kidney effects; liver effects; spleen effects	US EPA
									See aromatic C_9-C_{16} group; benzo(a)pyrene group			
Formaldehyde	1-Hour	50	Eye and nasal irritation	ATSDR	RsC	0.8	Nasal tumours	US EPA	RfD	150	Gastrointestinal effects; kidney effects	HC
					RfC	11	Eye, nasal and respiratory	TCEQ				

Table 2-10 Exposure Limits for the Chemicals of Potential Concern (continued)

Chemical of Potential Concern [(1)]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
							irritation					
Hexane	See aliphatic C_5-C_8 group				RfC	670	Neurological effects	TCEQ	N/A	N/A	N/A	N/A
					See aliphatic C_5-C_8 group							
Methyl ethyl ketone group (methyl ethyl ketone)	1-Hour	59,000	Neurological effects	TCEQ	RfC	5,000	Reproductive / developmental effects	US EPA	RfD	600	Reproductive / developmental effects	US EPA
Naphthalene and substituted naphthalenes (naphthalene)	1-Hour	2,000	Eye Irritation	ACGIH (adjusted)	RfC	3	Nasal irritation	US EPA	RfD	20	—	HC
	See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group			
Phenol	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	300	Reproductive / developmental effects	US EPA
Propylene	See aliphatic C_2-C_4 group				See aliphatic C_2-C_4 group				N/A	N/A	N/A	N/A
Propylene oxide	1-Hour	3,100	Nasal irritation	OEHHA	RsC	3	Nasal tumours	US EPA	RsD	0.04	Stomach tumours	US EPA
					RfC	30	Nasal irritation	US EPA				
Pyrenes and substituted pyrenes (pyrene)	See aromatic C_9-C_{16} group				See aromatic C_9-C_{16} group; benzo(a)pyrene group				RfD	30	Kidney effects	US EPA
									See aromatic C_9-C_{16} group; aromatic C_{17}-C_{34} group; benzo(a)pyrene group			
Toluene	1-Hour	15,000	Eye and nasal irritation; neurological effects	TCEQ	RfC	5,000	Neurological effects	US EPA	N/A	N/A	N/A	N/A
Trimethylbenzenes	1-Hour	690,000	Neurological effects	US EPA	RfC	5	Neurological effects	US EPA	N/A	N/A	N/A	N/A
Xylenes	1-Hour	7,400	Respiratory irritation; neurological effects	TCEQ	RfC	610	Eye and nasal irritation; neurological effects	TCEQ	N/A	N/A	N/A	N/A
Metals and Minerals												
Aluminum	—	—	—	—	RfC	5	Neurological effects	US EPA	RfD	143	Kidney effects; liver effects; neurological effects; reproductive / developmental effects	WHO
Antimony	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	0.2	Kidney effects; liver effects	HC

Table 2-10 Exposure Limits for the Chemicals of Potential Concern (continued)

Chemical of Potential Concern [1]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
Arsenic	1-Hour	0.2	Reproductive / developmental effects	OEHHA	RsC	0.0016	Lung tumours	HC	RsD	0.006	Bladder, liver, and lung tumours	HC
									RfD	0.3	—	US EPA
Barium	—	—	—	—	RfC	1.0	Cardiovascular effects; haematological effects	RIVM	RfD (food and soil)	200	Kidney effects	ATSDR
									RfD (water)	16	Cardiovascular effects	HC
Beryllium	—	—	—	—	RsC	0.004	Lung tumours	US EPA	RfD	2	Gastrointestinal effects	US EPA
					RfC	0.007	Respiratory irritation	OEHHA				
Boron	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	200	Reproductive / developmental effects	US EPA
Cadmium	24-Hour	0.03	Nasal and respiratory irritation	ATSDR	RsC	0.002	Lung tumours	OEHHA	RfD (food and soil)	1	Kidney effects	US EPA
					RfC	0.01	Kidney effects	ATSDR	RfD (water)	0.5	Kidney effects	US EPA
Chromium (chromium III)	1-Hour	12	Respiratory irritation	TCEQ	RfC	0.14	Respiratory irritation	TCEQ	RfD	1,500	—	US EPA
Chromium VI [3]	—	—	—	—	RsC	0.00013	Lung tumours	HC	RfD	1.0	Gastrointestinal effects	ATSDR
					RfC	0.1	Respiratory irritation	US EPA				
Cobalt	—	—	—	—	RfC	0.1	Respiratory irritation	ATSDR	RfD	1.4	Cardiovascular effects	RIVM
Copper	1-Hour	100	Respiratory irritation	OEHHA	RfC	1	Respiratory irritation; immunological effects	RIVM	RfD (birth to 4 years)	90	Liver effects	HC
									RfD (5 years +)	100	Liver effects	HC
Lead [4]	—	—	—	—	—	—	—	—	—	—	—	—
Lithium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	—	—	—	—

Table 2-10 Exposure Limits for the Chemicals of Potential Concern (continued)

Chemical of Potential Concern [1]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
Manganese	—	—	—	—	RfC	0.09	Neurological effects	OEHHA	RfD (soil and water)	47	Neurological effects	US EPA
									RfD (food)	140	Neurological effects	US EPA
Mercury	1-Hour	0.6	Neurological effects; reproductive / developmental effects	OEHHA	RfC	0.03	Neurological effects	OEHHA	RfD	0.3	Kidney effects	US EPA
Methyl mercury [5]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	0.1	Neurological effects; reproductive / developmental effects	US EPA
Molybdenum	—	—	—	—	RfC	12	—	RIVM	RfD	5	—	US EPA
Nickel	1-Hour	0.2	Immunological effects	OEHHA	RsC	0.0077	Lung tumours	HC	RfD	11	Reproductive / developmental effects	OEHHA
					RfC	0.014	Nasal and respiratory irritation	OEHHA				
Selenium	—	—	—	—	RfC	20	Liver effects; neurological effects	OEHHA	RfD	5	Liver effects; neurological effects	US EPA
Silver	—	—	—	—	RfC	0.4	N/A	ACGIH (adjusted)	RfD	5	N/A	US EPA
Strontium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	600	Reproductive / developmental effects	US EPA
Thallium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	—	—	—	—
Uranium	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	RfD	0.6	Kidney effects	HC
Vanadium	1-Hour	30	Respiratory irritation	OEHHA	RfC	0.1	Respiratory irritation	ATSDR	RfD	2	Reproductive / developmental effects	RIVM
Zinc	1-Hour	250	Respiratory irritation	ACGIH (adjusted)	—	—	—	—	RfD	300	N/A	US EPA

Table 2-10 Exposure Limits for the Chemicals of Potential Concern (continued)

Chemical of Potential Concern [(1)]	Acute Inhalation Exposure Limit				Chronic Inhalation Exposure Limit				Chronic Multiple Pathway Exposure Limit			
	Duration	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/m³]	Critical Effect	Agency	Type	Value [µg/kg bw/day]	Critical Effect	Agency
Sulphur Compounds												
1-Hexanethiol	—	—	—	—	—	—	—	—	N/A	N/A	N/A	N/A
COS	See CS_2 group								N/A	N/A	N/A	N/A
CS_2	6-Hour	6,200	Reproductive / developmental effects	OEHHA	RfC	100	Neurological effects	HC	N/A	N/A	N/A	N/A
H_2S	1-Hour	98	Respiratory irritation	ATSDR	RfC	2	Nasal irritation	US EPA	N/A	N/A	N/A	N/A
Thiophenes	—	—	—	—	—	—	—	—	N/A	N/A	N/A	N/A
Other												
Ammonia	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	—	—	—	—
Naphthenic acids [(6)]	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	—	—	—	—

Notes:

[(1)] Chemicals within the brackets refer to the surrogate assumed to be representative of toxicity of the chemical group. For identification of the most appropriate surrogate, preference was given to a chemical constituent of the group.

[(2)] Potential chronic inhalation health risks associated with benzo(a)pyrene and the other carcinogenic PAHs were evaluated using two different approaches

[(3)] For the inhalation assessment, it was assumed that chromium VI makes up 18% of total chromium emissions from combustion of diesel fuels (US EPA 2005). In the multiple pathway assessment, it was assumed that chromium VI makes up 8.3% of total chromium concentrations in soil and plants (Fengxiang et al. 2004), and 100% in water (Government of Canada 1994).

[(4)] The current state of the science prevents the use of any of the available exposure limits for lead. See Section 3.5 for further detail.

[(5)] Although the Project will not emit methyl mercury directly to the environment, it might release inorganic mercury into surface water. Bio-transformation of inorganic mercury species to methylated organic species in water bodies can occur in sediment and in the water column. On this basis, methyl mercury, in addition to mercury, was identified as a COPC in the multiple pathway assessment of the HHRA.

[(6)] Although an oral exposure limit was not identified for naphthenic acids, nor was an appropriate surrogate compound, a semi-quantitative assessment was conducted to address historical concerns expressed over the potential risks associated with naphthenic acids (Section 3.6).

— = No value available, or no information available

N/A= Not applicable.

Table 2-11 Potential Additive Interactions of the Chemicals of Potential Concern

Assessment	Critical Effect	Toxicant Designation	Chemicals of Potential Concern
Acute Inhalation	Eye irritation	Eye irritants [(1)]	Acetaldehyde, Acrolein, Aliphatic aldehyde group, Aromatic C_9-C_{16} group, Dichlorobenzenes, Formaldehyde, Naphthalene and substituted naphthalenes, Toluene
	Nasal irritation	Nasal irritants	Acetaldehyde, Acrolein, Cadmium, Dichlorobenzenes, Formaldehyde, Propylene oxide, Toluene
	Respiratory irritation	Respiratory irritants [(2)]	Acetaldehyde, Acrolein, Cadmium, Chromium, Copper, H_2S, NO_2, SO_2, Vanadium, Xylenes, Zinc
	Immunological effects	Immunotoxicants	Benzene, Nickel
	Neurological effects	Neurotoxicants	Acetone, Ethylbenzene, Mercury, Methyl ethyl ketone group, Toluene, Trimethylbenzenes, Xylenes
	Reproductive / developmental effects	Reproductive / developmental toxicants	1,3-Butadiene, Arsenic, CS_2 group, Mercury
Chronic Inhalation	Eye irritation	Eye irritants	Aliphatic aldehyde group, Formaldehyde, Xylenes
	Nasal irritation	Nasal irritants	Acetaldehyde, Acrolein, Aliphatic aldehyde group, Aliphatic C_2-C_4 group, Dichlorobenzenes, Formaldehyde, H_2S, Naphthalene and substituted naphthalenes, Nickel, Propylene oxide, Xylenes
	Respiratory irritation	Respiratory irritants	Beryllium, Chromium, Chromium VI, Cobalt, Copper, Formaldehyde, Nickel, NO_2, Vanadium
	Haematological effects	Hematotoxicants	Barium, Benzene
	Immunological effects	Immunotoxicants	Benzene, Copper
	Kidney effects	Renal toxicants [(3)]	Aliphatic C_2-C_4 group, Aromatic C_9-C_{16} group, Cadmium, Cumene, Ethylbenzene
	Liver effects	Hepatotoxicants	Aromatic C_9-C_{16} group, Selenium
	Neurological effects	Neurotoxicants [(3)]	Acetone, Aliphatic C_5-C_8 group, Aliphatic C_9-C_{16} group, Aluminum, CS_2 group, Hexane, Manganese, Mercury, Selenium, Toluene, Trimethylbenzenes, Xylenes
	Reproductive / developmental effects	Reproductive / developmental toxicants	1,3-Butadiene, Cyclohexane, Methyl ethyl ketone group
	Nasal tumours	Nasal carcinogens	Acetaldehyde, Formaldehyde, Propylene oxide
	Lung tumours	Lung carcinogens [(4)]	Arsenic, Benzo(a)pyrene group, Beryllium, Cadmium, Chromium VI, Nickel
	Leukemia	Leukemogens	1,3-Butadiene, Benzene

Table 2-11 Potential Additive Interactions of the Chemicals of Potential Concern (continued)

Assessment	Critical Effect	Toxicant Designation	Chemicals of Potential Concern
Chronic Multiple Pathway	Cardiovascular effects	Cardiovascular toxicants	Barium, Cobalt
	Gastrointestinal effects	Gastrointestinal toxicants	Aliphatic aldehyde group, Beryllium, Chromium VI, Formaldehyde
	Kidney effects	Renal toxicants [3]	Acetone, Aliphatic C_9-C_{16} group, Aluminum, Antimony, Aromatic C_9-C_{16} group, Aromatic C_{17}-C_{34} group, Barium, Biphenyls, Cadmium, Fluorenes/fluoranthenes and substituted, Formaldehyde, Mercury, Pyrenes and substituted pyrenes, Uranium
	Liver effects	Hepatotoxicants [3]	Acenaphthenes / acenaphthylenes, Aliphatic C_9-C_{16} group, Aliphatic C_{17}-C_{34} group, Aluminum, Antimony, Aromatic C_9-C_{16} group, Copper, Fluorenes/fluoranthenes and substituted, Selenium
	Neurological effects	Neurotoxicants	Aluminum, Manganese, Methyl mercury, Selenium
	Reproductive / developmental effects	Reproductive / developmental toxicants	Acetone, Aluminum, Boron, Methyl mercury, Methyl ethyl ketone group, Nickel, Phenol, Strontium, Vanadium

Notes:

[1] Naphthalene was not added to the eye irritants mixture in the acute inhalation assessment as it was already used as the surrogate for the aromatic C_9-C_{16} group.

[2] The highest risk estimate of the averaging times (i.e., 10-minute and 1-hour) for SO_2 was used in the prediction of the potential health risks for the respiratory irritants mixture in the acute inhalation assessment.

[3] Because some COPC were assessed both individually and as part of a chemical group, the corresponding risk estimates were likely exaggerated due to the 'double counting' of these chemicals in the mixtures. Examples include:

- cumene and the aromatic C_9-C_{16} group in the chronic inhalation renal toxicants;
- hexane and the aliphatic C_5-C_8 group in the chronic inhalation neurotoxicants;
- fluorenes/fluoranthenes and substituted and the aromatic C_9-C_{16} group in the chronic multiple pathway hepatotoxicants and renal toxicants; and
- pyrene and substituted pyrenes, the aromatic C_9-C_{16} group and the aromatic C_{17}-C_{34} group in the chronic multiple pathway renal toxicants.

[4] The highest risk estimate of the two approaches for assessing the benzo(a)pyrene group was used in the prediction of the potential health risks for the lung carcinogens mixture in the chronic inhalation assessment.

2.5 Risk Characterization

The assessment methods for characterizing the health risks were consistent with those described in EIA, Volume 3, Section 5.3.2.4. To assist in the interpretation of the results, the pertinent aspects of the risk characterization step of the HHRA are reproduced below.

Risk estimates are presented as potential PRM-specific effects and cumulative effects for both acute and chronic exposures. The potential health risks associated with COPC emissions from the PRM are expressed as risk quotients for the non-cancer (i.e., non-carcinogenic) COPC and as Incremental Lifetime Cancer Risks (ILCRs) for the carcinogenic COPC.

2.5.1 Non-Cancer Risk Estimates

Risk quotients are calculated by comparing the predicted levels of exposure for the non-carcinogenic COPC with their respective exposure limits developed by regulatory or scientific authorities. The chronic Risk Quotients (RQs) for three of the assessment cases (i.e., 2013 Base, 2013 PRM Application and 2013 PDC) are calculated as follows:

$$\text{Risk Quotient} = \frac{\text{Predicted Exposure } (\mu g/m^3 \text{ or } \mu g/kg \text{ bw/day})}{\text{Exposure Limit } (\mu g/m^3 \text{ or } \mu g/kg \text{ bw/day})}$$

Interpretation of the RQ values proceeded as follows:

- RQ ≤ 1.0: indicates that the estimated exposure is less than or equal to the exposure limit (i.e., the assumed safe level of exposure). Risk quotients less than or equal to 1.0 are associated with low health risks, even in sensitive individuals given the level of conservatism incorporated in the derivation of the exposure limits and the risk estimates.
- RQ >1.0: indicates that the exposure estimate exceeds the exposure limit. This suggests an elevated level of risk, the consequence of which must be balanced against the degree of conservatism incorporated in the risk assessment.

2.5.2 Cancer Risk Estimates

Regulatory agencies such as Health Canada, ESRD and the US EPA assume that any level of long-term exposure to carcinogenic chemicals is associated with some 'hypothetical cancer risk'. On this basis, Health Canada and ESRD have specified an incremental (i.e., over and above background) lifetime cancer risk of 1 in 100,000, which these agencies consider acceptable, tolerable or essentially negligible (AHW 2011; Health Canada 2010a). Because this assumed 'acceptable' cancer risk level was specifically developed to address cancer risks over and above background cancer incidence, a portion of which includes background exposure to environmental pollutants, background exposures were not included in the assessment of potential health risks for non-threshold (i.e., carcinogenic) chemicals.

Health Canada (2010a) requires that carcinogens be assessed on an incremental basis, and mandates an 'acceptable' incremental lifetime cancer risk of 1 in 100,000. For the purposes of this assessment,

incremental lifetime cancer risks have been determined for PRM alone as well as the incremental contribution of the planned future emission sources.

The incremental lifetime cancer risks were calculated for PRM alone and planned future emission sources as follows:

$$\text{ILCR} = \frac{\text{Incremental Exposure (µg/m}^3\text{ or µg/kg bw/day)}}{\text{Carcinogenic Exposure Limit (µg/m}^3\text{ or µg/kg bw/day)}}$$

Interpretation of these ILCRs was based on comparison of the ILCR associated with the PRM alone against the Health Canada (2010a) *de minimus risk* level of 1 in 100,000 (i.e., one extra cancer case in a population of 100,000 people).

3.0 Results

As previously stated, health effects are dependent, in part, on the duration of exposure. Similarly, the pathway of exposure can also influence the potential health effects elicited by a chemical exposure.

In recognition of the influence of duration and pathway of exposure, risk estimates are discussed below in the context of:

- acute inhalation;
- chronic inhalation; and
- chronic multiple pathway.

The risk estimates are presented in scientific notation as many of the calculated numerical values are well below 1.0. For instance, the acute RQ for a person exposed to the peak hourly dichlorobenzenes air concentration along the PRM fenceline under the 2013 Base Case is 2.6E-07, which is equivalent to an RQ of 0.00000026 (Table 3-1).

The discussion of the results focuses on those risk estimates that exceed 1.0 (presented in bold in the tables), as these could signify potential health risks. Where risk estimates do not exceed 1.0 (i.e., where the predicted exposures are less than the exposure limits), the predicted risk values are presented in the tables but are not discussed further.

3.1 Acute Inhalation

Acute inhalation risk estimates, expressed as RQ values, were based on assumed exposure periods that range from a few minutes to 24 hours. The maximum RQ values for the PRM fenceline, Aboriginal group (cabin and community locations) and worker group are presented in Table 3-1 to Table 3-4. Note that the risks for the Aboriginal group are “teased out” for the cabin locations and community locations in Table 3-2 and Table 3-3, respectively.

Most of the RQ values are below 1.0, indicating that the predicted air concentrations for those COPC are less than their exposure limits. Adverse health effects are therefore not expected to result from acute exposure to these COPC.

Risk quotients are predicted to be greater than 1.0 for the following:

- Along the PRM fenceline:
  - aromatic C_9-C_{16} group for the 2013 PDC only;
  - eye irritants for all three assessment cases;
  - nasal irritants for all three assessment cases; and
  - respiratory irritants for all three assessment cases.
- Cabin locations:
  - acrolein for all three assessment cases;
  - aliphatic aldehyde group for all three assessment cases;
  - aromatic C_9-C_{16} group for the 2013 PDC only;
  - eye irritants for all three assessment cases;
  - nasal irritants for all three assessment cases; and
  - respiratory irritants for all three assessment cases.
- Community locations:
  - acrolein for the 2013 PDC only;
  - eye irritants for all three assessment cases;
  - nasal irritants for all three assessment cases; and
  - respiratory irritants for all three assessment cases.
- Worker locations:
  - SO_2 (10-minute) for all three assessment cases;
  - acrolein for all three assessment cases;
  - eye irritants for all three assessment cases;
  - nasal irritants for all three assessment cases; and
  - respiratory irritants for all three assessment cases.

The significance of each exceedance is discussed in the following sections.

Table 3-1 Acute Inhalation Risk Quotients – Pierre River Mine Fenceline

Chemical of Potential Concern(		Averaging Time[1]	Risk Quotients[2]		
			2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminant	CO	1-hour	2.2E-02	2.6E-02	2.7E-02
		8-hour	6.3E-02	9.0E-02	9.4E-02
	NO_2	1-hour	4.9E-01	5.1E-01	4.8E-01
	$PM_{2.5}$	24-hour CWS Stat	6.2E-01	7.9E-01	7.9E-01
	SO_2	10-minute	4.6E-01	4.6E-01	4.8E-01
		1-hour	3.1E-01	3.1E-01	3.3E-01
Organics	1,3-Butadiene	24-hour	6.4E-03	9.7E-03	9.7E-03
	Acetaldehyde	1-hour	6.2E-02	6.8E-02	6.7E-02
	Acetone	1-hour	2.5E-04	2.7E-04	2.7E-04
	Acrolein	1-hour	9.5E-01	1.0E+00	1.0E+00
	Aliphatic aldehyde group	1-hour	8.2E-01	9.0E-01	8.9E-01
	Aliphatic C_2-C_4 group	1-hour	7.7E-05	1.6E-03	1.6E-03
	Aliphatic C_5-C_8 group	1-hour	1.2E-02	4.5E-02	5.3E-02
	Aromatic C_9-C_{16} group	1-hour	7.7E-02	8.1E-02	**1.5E+00**
	Benzene	1-hour	1.7E-02	7.1E-02	7.1E-02
	Dichlorobenzenes	1-hour	2.6E-07	8.4E-07	8.6E-07
	Ethylbenzene	1-hour	4.6E-03	4.7E-03	1.4E-01
	Formaldehyde	1-hour	3.1E-01	3.4E-01	3.4E-01
	Methyl ethyl ketone group	1-hour	1.5E-04	1.7E-04	1.6E-04
	Naphthalene and substituted naphthalenes	1-hour	5.8E-05	8.1E-05	8.6E-05
	Propylene oxide	1-hour	1.6E-06	1.4E-05	1.4E-05
	Toluene	1-hour	1.5E-02	1.5E-02	7.4E-02
	Trimethylbenzenes	1-hour	5.0E-05	5.0E-05	2.1E-04
	Xylenes	1-hour	1.8E-02	1.9E-02	5.4E-01
Sulphur Compounds	CS_2 group	1-hour	2.8E-04	5.4E-04	1.9E-03
	H_2S	1-hour	5.1E-02	8.3E-02	1.9E-01
Metals	Arsenic	1-hour	1.8E-03	3.8E-03	4.0E-03
	Cadmium	24-hour	1.8E-01	2.6E-01	2.8E-01
	Chromium	1-hour	7.1E-04	7.2E-04	7.9E-04
	Copper	1-hour	2.6E-05	3.4E-05	3.6E-05
	Mercury	1-hour	6.0E-04	1.7E-03	1.7E-03
	Nickel	1-hour	5.9E-02	6.0E-02	6.6E-02
	Vanadium	1-hour	2.3E-04	3.0E-04	3.1E-04
	Zinc	1-hour	2.9E-04	4.5E-04	4.7E-04
Mixtures[3]	Eye irritants	–	**2.2E+00**	**2.5E+00**	**3.9E+00**
	Nasal irritants	–	**1.5E+00**	**1.7E+00**	**1.8E+00**
	Respiratory irritants	–	**2.2E+00**	**2.4E+00**	**3.1E+00**
	Immunotoxicants	–	7.6E-02	1.3E-01	1.4E-01
	Neurotoxicants	–	3.9E-02	4.1E-02	7.6E-01
	Reproductive / developmental toxicants	–	9.1E-03	1.6E-02	1.7E-02

Notes:
[1] Based on the peak (1st highest) predicted air concentration, unless otherwise noted.
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
– = Not applicable.

Table 3-2 Acute Inhalation Risk Quotients – Aboriginal Group (Cabin Locations)

Chemical of Potential Concern		Averaging Time[1]	Risk Quotients[2]		
			2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	CO	1-hour	3.5E-02	3.5E-02	3.6E-02
		8-hour	7.7E-02	7.9E-02	8.1E-02
	NO_2	1-hour	6.2E-01	6.3E-01	5.9E-01
	$PM_{2.5}$	24-hour CWS Stat	8.6E-01	8.7E-01	9.0E-01
	SO_2	10-minute	4.7E-01	4.7E-01	5.0E-01
		1-hour	3.2E-01	3.2E-01	3.4E-01
Organics	1,3-Butadiene	24-hour	9.1E-03	9.3E-03	8.6E-03
	Acetaldehyde	1-hour	9.7E-02	9.7E-02	9.5E-02
	Acetone	1-hour	3.9E-04	3.9E-04	3.8E-04
	Acrolein	1-hour	**1.5E+00**	**1.5E+00**	**1.5E+00**
	Aliphatic aldehyde group	1-hour	**1.3E+00**	**1.3E+00**	**1.2E+00**
	Aliphatic C_2-C_4 group	1-hour	4.2E-04	4.2E-04	4.3E-04
	Aliphatic C_5-C_8 group	1-hour	2.3E-02	2.3E-02	5.0E-02
	Aromatic C_9-C_{16} group	1-hour	5.4E-01	5.4E-01	**1.6E+00**
	Benzene	1-hour	4.1E-02	4.1E-02	8.0E-02
	Dichlorobenzenes	1-hour	3.3E-07	3.3E-07	7.7E-07
	Ethylbenzene	1-hour	5.0E-02	5.0E-02	1.5E-01
	Formaldehyde	1-hour	4.9E-01	4.9E-01	4.8E-01
	Methyl ethyl ketone group	1-hour	2.4E-04	2.4E-04	2.3E-04
	Naphthalene and substituted naphthalenes	1-hour	8.9E-05	8.9E-05	9.2E-05
	Propylene oxide	1-hour	1.9E-06	5.2E-06	5.2E-06
	Toluene	1-hour	2.7E-02	2.7E-02	7.9E-02
	Trimethylbenzenes	1-hour	7.4E-05	7.4E-05	2.2E-04
	Xylenes	1-hour	1.9E-01	1.9E-01	5.8E-01
Sulphur Compounds	CS_2 group	1-hour	6.3E-04	6.4E-04	7.6E-04
	H_2S	1-hour	7.0E-02	7.0E-02	8.9E-02
Metals	Arsenic	1-hour	1.6E-03	1.6E-03	3.5E-03
	Cadmium	24-hour	2.9E-01	3.0E-01	3.2E-01
	Chromium	1-hour	7.0E-04	7.0E-04	7.7E-04
	Copper	1-hour	4.0E-05	4.0E-05	7.2E-05
	Mercury	1-hour	4.7E-04	5.1E-04	1.5E-03
	Nickel	1-hour	5.8E-02	5.9E-02	6.5E-02
	Vanadium	1-hour	2.2E-04	2.2E-04	2.7E-04
	Zinc	1-hour	2.4E-04	2.5E-04	5.2E-04
Mixtures[3]	Eye irritants	-	**3.4E+00**	**3.4E+00**	**4.3E+00**
	Nasal irritants	-	**2.3E+00**	**2.3E+00**	**2.3E+00**
	Respiratory irritants	-	**3.0E+00**	**2.9E+00**	**3.1E+00**
	Immunotoxicants	-	9.6E-02	9.6E-02	1.2E-01
	Neurotoxicants	-	2.7E-01	2.7E-01	8.1E-01
	Reproductive / developmental toxicants	-	1.1E-02	1.1E-02	1.4E-02

Notes:
[1] Based on the peak (1st highest) predicted air concentration, unless otherwise noted.
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
– = Not applicable.

Table 3-3 Acute Inhalation Risk Quotients – Aboriginal Group (Community Locations)

Chemical of Potential Concern		Averaging Time[1]	Risk Quotients[2]		
			2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	CO	1-hour	8.4E-02	8.4E-02	8.2E-02
		8-hour	1.9E-01	1.9E-01	1.9E-01
	NO_2	1-hour	4.4E-01	4.4E-01	4.5E-01
	$PM_{2.5}$	24-hour CWS Stat	9.2E-01	9.3E-01	9.4E-01
	SO_2	10-minute	5.4E-01	5.4E-01	5.5E-01
		1-hour	3.6E-01	3.6E-01	3.7E-01
Organics	1,3-Butadiene	24-hour	5.2E-03	5.2E-03	7.1E-03
	Acetaldehyde	1-hour	4.6E-02	4.7E-02	6.9E-02
	Acetone	1-hour	1.8E-04	1.9E-04	2.8E-04
	Acrolein	1-hour	7.0E-01	7.2E-01	**1.1E+00**
	Aliphatic aldehyde group	1-hour	6.1E-01	6.2E-01	9.1E-01
	Aliphatic C_2-C_4 group	1-hour	9.9E-05	1.0E-04	2.1E-04
	Aliphatic C_5-C_8 group	1-hour	8.0E-03	8.0E-03	9.8E-03
	Aromatic C_9-C_{16} group	1-hour	2.4E-02	2.5E-02	7.5E-02
	Benzene	1-hour	5.4E-02	5.4E-02	1.4E-01
	Dichlorobenzenes	1-hour	2.4E-07	2.4E-07	6.0E-07
	Ethylbenzene	1-hour	7.5E-04	7.6E-04	6.9E-03
	Formaldehyde	1-hour	2.3E-01	2.4E-01	3.5E-01
	Methyl ethyl ketone group	1-hour	1.1E-04	1.2E-04	2.6E-04
	Naphthalene and substituted naphthalenes	1-hour	4.3E-05	4.5E-05	4.7E-05
	Propylene oxide	1-hour	9.5E-07	9.7E-07	1.8E-06
	Toluene	1-hour	4.6E-03	4.6E-03	5.6E-03
	Trimethylbenzenes	1-hour	1.5E-05	1.5E-05	1.8E-05
	Xylenes	1-hour	7.5E-03	7.5E-03	2.7E-02
Sulphur Compounds	CS_2 group	1-hour	2.3E-04	2.4E-04	2.9E-04
	H_2S	1-hour	1.3E-02	1.3E-02	1.4E-02
Metals	Arsenic	1-hour	1.4E-03	1.4E-03	1.5E-03
	Cadmium	24-hour	1.9E-01	1.9E-01	1.9E-01
	Chromium	1-hour	8.1E-04	8.1E-04	8.2E-04
	Copper	1-hour	1.9E-05	1.9E-05	2.3E-05
	Mercury	1-hour	6.0E-04	6.0E-04	6.7E-04
	Nickel	1-hour	8.5E-02	8.5E-02	8.5E-02
	Vanadium	1-hour	2.2E-04	2.2E-04	2.3E-04
	Zinc	1-hour	3.5E-04	3.5E-04	3.6E-04
Mixtures[3]	Eye irritants	-	**1.6E+00**	**1.6E+00**	**2.4E+00**
	Nasal irritants		**1.2E+00**	**1.2E+00**	**1.5E+00**
	Respiratory irritants	-	**1.9E+00**	**2.0E+00**	**2.0E+00**
	Immunotoxicants	-	9.4E-02	9.4E-02	1.8E-01
	Neurotoxicants	-	1.4E-02	1.4E-02	3.8E-02
	Reproductive / developmental toxicants	-	7.4E-03	7.5E-03	8.6E-03

Notes:
[1] Based on the peak (1st highest) predicted air concentration, unless otherwise noted.
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
– = Not applicable.

Table 3-4 Acute Inhalation Risk Quotients – Worker Group

Chemical of Potential Concern		Averaging Time[1]	Risk Quotients[2]		
			2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	CO	1-hour	2.6E-02	2.6E-02	3.2E-02
		8-hour	9.6E-02	9.6E-02	1.2E-01
	NO_2	1-hour	5.6E-01	5.6E-01	5.6E-01
	$PM_{2.5}$	24-hour CWS Stat	9.2E-01	9.3E-01	9.3E-01
	SO_2	10-minute	**1.3E+00**	**1.3E+00**	**1.3E+00**
		1-hour	8.7E-01	8.7E-01	8.9E-01
Organics	1,3-Butadiene	24-hour	1.2E-02	1.2E-02	1.4E-02
	Acetaldehyde	1-hour	7.0E-02	7.1E-02	7.8E-02
	Acetone	1-hour	2.8E-04	2.8E-04	3.1E-04
	Acrolein	1-hour	**1.1E+00**	**1.1E+00**	**1.2E+00**
	Aliphatic aldehyde group	1-hour	9.2E-01	9.3E-01	**1.0E+00**
	Aliphatic C_2-C_4 group	1-hour	2.2E-04	2.3E-04	3.3E-04
	Aliphatic C_5-C_8 group	1-hour	1.2E-02	1.2E-02	1.4E-02
	Aromatic C_9-C_{16} group	1-hour	4.3E-02	4.3E-02	2.3E-01
	Benzene	1-hour	1.3E-02	1.3E-02	1.9E-02
	Dichlorobenzenes	1-hour	9.3E-07	9.3E-07	8.8E-07
	Ethylbenzene	1-hour	1.2E-03	1.2E-03	2.2E-02
	Formaldehyde	1-hour	3.5E-01	3.6E-01	3.9E-01
	Methyl ethyl ketone group	1-hour	1.7E-04	1.7E-04	1.9E-04
	Naphthalene and substituted naphthalenes	1-hour	7.5E-05	7.5E-05	1.2E-04
	Propylene oxide	1-hour	1.1E-05	1.1E-05	1.6E-05
	Toluene	1-hour	9.0E-03	9.0E-03	1.2E-02
	Trimethylbenzenes	1-hour	3.0E-05	3.0E-05	3.3E-05
	Xylenes	1-hour	1.2E-02	1.2E-02	8.3E-02
Sulphur Compounds	CS_2 group	1-hour	6.1E-04	6.2E-04	8.6E-04
	H_2S	1-hour	9.2E-02	1.6E-01	1.6E-01
Metals	Arsenic	1-hour	3.1E-03	3.1E-03	4.3E-03
	Cadmium	24-hour	4.8E-01	4.8E-01	5.8E-01
	Chromium	1-hour	1.2E-03	1.2E-03	1.2E-03
	Copper	1-hour	5.6E-05	5.6E-05	5.7E-05
	Mercury	1-hour	1.3E-03	1.3E-03	1.8E-03
	Nickel	1-hour	1.2E-01	1.2E-01	1.2E-01
	Vanadium	1-hour	3.3E-04	3.3E-04	3.3E-04
	Zinc	1-hour	5.2E-04	5.2E-04	5.2E-04
Mixtures[3]	Eye irritants	-	**2.5E+00**	**2.5E+00**	**2.8E+00**
	Nasal irritants	-	**2.0E+00**	**2.0E+00**	**2.3E+00**
	Respiratory irritants	-	**3.2E+00**	**3.3E+00**	**3.3E+00**
	Immunotoxicants		1.4E-01	1.4E-01	1.4E-01
	Neurotoxicants	-	2.3E-02	2.3E-02	1.2E-01
	Reproductive / developmental toxicants	-	1.7E-02	1.7E-02	2.1E-02

Notes:
[1] Based on the peak (1st highest) predicted air concentration, unless otherwise noted.
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
– = Not applicable.

3.1.1 Acrolein

The acute RQ values for acrolein, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 for the Aboriginal group (cabin and community locations) and the worker group, but not at any locations along the PRM fenceline where the maximum PRM-related air concentrations would be expected to occur. At the maximum cabin location (Cabin K), the peak 1-hour acrolein concentrations are predicted to be 3.7 µg/m^3 for the 2013 Base and 2013 PRM Application cases, and 3.6 µg/m^3 for the 2013 PDC. These peak air concentrations are associated with the maximum RQ of 1.5 predicted for the cabin locations, under all three assessment cases. Peak hourly air concentrations also exceed the acute exposure limit of 2.5 µg/m^3 for acrolein at Cabin J where peak 1-hour acrolein air concentrations are predicted to be 3.1 µg/m^3 for the 2013 Base and 2013 PRM Application cases, and 2.8 µg/m^3 for the 2013 PDC.

At the maximum of the community locations, the peak 1-hour acrolein concentrations are predicted to be 1.8 µg/m^3 for the 2013 Base and 2013 PRM Application cases (Fort McKay), and 2.7 µg/ m^3 for the 2013 PDC (Fort McMurray). Based on these peak predicted concentrations, the maximum RQ values for the community locations are below 1.0 for the 2013 Base and 2013 PRM Application cases, but increase to 1.1 at Fort McMurray for the 2013 PDC. At the maximum worker location (Jackpine Mine Camp), the peak 1-hour acrolein concentrations are predicted to be 2.7 µg/m^3 for the 2013 Base and 2013 PRM Application cases, and 3.0 µg/m^3 for the 2013 PDC. These peak predicted air concentrations for the worker group are associated with the maximum RQ values of 1.1 for the 2013 Base and 2013 PRM Application cases, and 1.2 for the 2013 PDC. The predicted peak 1-hour air concentrations are below the acute exposure limit for acrolein at all other locations assessed in the 2013 HHRA.

As the 2013 Base Case peak acrolein air concentrations were not predicted to change at these locations under the 2013 PRM Application Case, the incremental changes in peak predicted air concentrations as a result of Project emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to acrolein at these locations. For the 2013 PDC, RQ values were predicted to increase at the maximum of the community locations and industrial camp sites. The predicted increase in the peak acrolein air concentrations in Fort McMurray can be attributed to the projected increase in the Fort McMurray population, while the predicted increase at the Jackpine Mine Camp is the result of planned future developments in the region.

Use of the peak predicted air concentrations is conservative, as these concentrations result from rare and extreme meteorological conditions of a short-lived nature. Alberta ESRD recommends that the eight highest predicted 1-hour concentrations for each location in a single year be disregarded, as they are considered to be outliers (AENV 2009). The 9th highest value may therefore be a more reasonable concentration to consider for the purposes of the 2013 HHRA, as the acrolein concentrations are expected to be equal to or lower than this value 99.9% of the time.

The 9th highest hourly acrolein concentrations are not predicted to exceed the acute exposure limit of 2.5 µg/m^3 at any locations assessed in the 2013 HHRA, other than the Jackpine Mine Camp for the 2013

PDC. For the acute RQ values predicted using the 9th highest hourly acrolein concentrations, refer to Table 3-5.

Table 3-5 Comparison of Acute Inhalation Risk Quotients for Acrolein Based on the Peak and 9th Highest 1-Hour Concentrations

Receptor Group	Risk Quotients[(1)]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	9.5E-01	1.0E+00	1.0E+00	8.1E-01	9.1E-01	8.7E-01
Cabin Locations	**1.5E+00**	**1.5E+00**	**1.5E+00**	9.4E-01	9.7E-01	8.5E-01
Community Locations	7.0E-01	7.2E-01	**1.1E+00**	5.3E-01	5.4E-01	8.5E-01
Worker Camp Sites	**1.1E+00**	**1.1E+00**	**1.2E+00**	1.0E+00	1.0E+00	**1.1E+00**

Notes:
(1) Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

For the locations at which peak 1-hour air concentrations are predicted to exceed the acute exposure limit for acrolein, attention was given to the likelihood of such an exceedance occurring. Frequency analysis of the predicted 1-hour acrolein air concentrations for 1995 and 2002 was completed for each of these locations; the higher frequency of the two years is presented in Table 3-6. Note that the values in Table 3-6 do not provide any indication as to the level of the exceedance; they simply acknowledge that an exceedance was predicted.

Table 3-6 Predicted Likelihood of Exceeding the 1-Hour Acute Inhalation Exposure Limit for Acrolein

Location	Frequency of Exceedance [%][(1)]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Cabin J	0.057	0.057	0.034
Cabin K	0.068	0.080	0.068
Fort McMurray	0	0	0.034
Jackpine Mine Camp	0.13	0.13	0.47

Notes:
(1) Values represent the number of hours, as a percentage, that the exposure limit was exceeded on an annual basis.

Two acute studies of human exposure to acrolein are available, and form the basis of the acute exposure limit of 2.5 µg/m³ used in the 2013 HHRA. The lowest-observable-adverse-effect-level (LOAEL) determined from a study by Darley et al. (1960) was 140 µg/m³. For the second study, Weber-Tschopp et al. (1977) identified a LOAEL of 160 µg/m³. Mild eye irritation was reported at these concentrations. At increasing concentrations (i.e., above 140 and 160 µg/m³), nasal and respiratory irritation was also reported by the exposed subjects. The results of these two studies suggest that the maximum predicted peak hourly concentration (3.7 µg/m³) and 9th highest hourly concentration (2.9 µg/m³) are well below the concentrations at which irritation has been reported in humans. There is a margin of safety (ratio of

the potential effect concentration to exposure concentration) of 38 to 55 between the LOAELs and the maximum predicted hourly air concentrations for acrolein. These comparisons with the toxicological thresholds suggest that the overall potential for adverse effects in association with short-term exposure to acrolein is likely low at the locations assessed in the 2013 HHRA.

3.1.2 Aliphatic Aldehyde Group

The acute RQ values for the aliphatic aldehyde group, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 for the Aboriginal group (cabin locations only). At the maximum cabin location (Cabin K), the peak 1-hour aliphatic aldehyde concentrations are predicted to be 68 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 66 µg/m³ for the 2013 PDC. These peak air concentrations are associated with the maximum predicted RQ values of 1.3 for the cabin locations under the 2013 Base and 2013 PRM Application cases, and 1.2 under the 2013 PDC. Peak hourly aliphatic aldehyde concentrations also exceed the exposure limit of 53 µg/m³ at Cabin J where peak 1-hour air concentrations are predicted to be 56 µg/m³, 57 µg/m³ and 51 µg/m³ for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. The acute RQ values for the aliphatic aldehyde group are below 1.0 at all other locations assessed in the 2013 HHRA, even along the PRM fenceline where the maximum PRM-related air concentrations would be expected to occur.

As the 2013 Base Case peak air concentrations for the aliphatic aldehyde group are predicted to increase only slightly, if at all, at these locations under the 2013 PRM Application Case and 2013 PDC, the incremental changes in peak predicted air concentrations as a result of PRM emissions and emissions from planned future developments are essentially negligible, and the PRM and other planned developments will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to the aliphatic aldehydes at these locations.

As previously discussed, use of the 9th highest value may be a more reasonable concentration to consider for the purposes of the 2013 HHRA, as the aliphatic aldehyde concentrations are expected to be equal to or lower than this value 99.9% of the time. The 9th highest hourly aliphatic aldehyde concentrations are not predicted to exceed the acute exposure limit of 53 µg/m³ at any locations assessed in the 2013 HHRA. For the acute RQ values predicted using the 9th highest hourly aliphatic aldehyde concentrations, refer to Table 3-7.

Table 3-7 Comparison of Acute Inhalation Risk Quotients for the Aliphatic Aldehyde Group Based on the Peak and 9th Highest 1-Hour Concentrations

Receptor Group	Risk Quotients[(1)]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	8.2E-01	9.0E-01	8.9E-01	8.1E-01	8.3E-01	7.3E-01
Cabin Locations	**1.3E+00**	**1.3E+00**	**1.2E+00**	6.9E-01	7.9E-01	7.5E-01
Community Locations	6.1E-01	6.2E-01	9.1E-01	4.6E-01	4.6E-01	7.3E-01
Worker Camp Sites	9.2E-01	9.3E-01	1.0E+00	8.7E-01	8.7E-01	9.9E-01

Notes:
(1) Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

For the locations at which peak 1-hour air concentrations were predicted to exceed the exposure limit of 53 µg/m³ for the aliphatic aldehyde group, attention was given to the likelihood of such an exceedance occurring. Frequency analysis of the predicted 1-hour aliphatic aldehyde air concentrations for 1995 and 2002 was completed for each of these locations; the higher frequency of the two years is presented in Table 3-8. Note that the values in Table 3-8 do not provide any indication as to the level of the exceedance; they simply acknowledge that an exceedance was predicted.

Table 3-8 Predicted Likelihood of Exceeding the 1-Hour Acute Inhalation Exposure Limit for the Aliphatic Aldehyde Group

Location	Frequency of Exceedance [%][(1)]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Cabin J	0.023	0.023	0
Cabin K	0.023	0.034	0.023

Notes:
(1) Values represent the number of hours, as a percentage, that the exposure limit was exceeded on an annual basis.

In total, 15 aliphatic aldehydes were evaluated as part of the aliphatic aldehyde group using methacrolein as the surrogate in the acute inhalation assessment. The constituents of the aliphatic aldehyde group are 3-methylbutanal, benzaldehyde, butanal, crotonaldehyde, decanal, dodecanal, heptanal, hexanal, isobutyraldehyde, methacrolein, nonanal, octanal, propanal (propionaldeyde), tridecanal and undecanal.

A tiered approach was used in the selection of the exposure limits. If a suitable exposure limit could not be identified from one of the regulatory agencies in the first tier, the search was then expanded to the second tier of agencies. For the aliphatic aldehydes, a suitable acute inhalation exposure limit was identified for methacrolein in the first tier. The acute inhalation exposure limit of 53 µg/m³ was derived by the TCEQ from a human study where the effects of methacrolein on eye irritation were evaluated (TCEQ 2009). The LOAEL determined from the study was 800 µg/m³ for increased eye blink frequency during 20 minutes of exposure to methacrolein. The results of this study suggest that the peak predicted hourly concentration of 68 µg/m³ is well below the concentrations at which irritation has been reported

in the scientific literature. There is a margin of safety of about 12 between the LOAEL of 800 µg/m³ and the peak predicted air concentration for the aliphatic aldehyde group of 68 µg/m³. The margin of safety is increased to 15-fold using the 9th highest predicted hourly concentration of 52 µg/m³. This comparison with the toxicological threshold for methacrolein suggests that the overall potential for adverse effects in association with short-term exposure to the aliphatic aldehydes is expected to be low at the locations assessed in the 2013 HHRA.

For added perspective, acute inhalation limits were identified for crotonaldehyde and propionaldehyde from the second tier of agencies. These include the United States Environmental Protection Agency (US EPA) 1-hour Acute Exposure Guideline Level 1 (AEGL-1) values of 550 µg/m³ for crotonaldehyde and 110,000 µg/m³ for propionaldehyde; both based on human exposure data (US EPA 2008, 2009, respectively). These limits were not selected in the current acute inhalation assessment of aliphatic aldehydes due to the presence of the more conservative (i.e., lower), first tier acute inhalation exposure limit for methacrolein. However, they do suggest that use of the methacrolein as the surrogate chemical for the aliphatic aldehydes group may overstate the actual health risks posed to the area residents.

The acute inhalation exposure limit for crotonaldehyde was based on chemical plant workers complaining of eye irritation. Crotonaldehyde concentrations measured near vats of chemicals at eight different locations within the plant ranged from less than 1,000 to 3,100 µg/m³, with an average concentration of 1,600 µg/m³. To account for differences in sensitivity and susceptibility between workers and members of the public, the US EPA applied a partial uncertainty factor of 3 to the average concentration, resulting in an acute exposure limit of 550 µg/m³. There is an 8-fold margin of safety between the peak predicted air concentration for the aliphatic aldehydes and the US EPA 1-hour AEGL-1 for crotonaldehyde.

For propionaldehyde, the acute inhalation exposure limit was based on a controlled chamber study in which twelve adult males were exposed to 320,000 µg/m³ of propionaldehyde for 30 minutes. Mild irritation of the mucosal surfaces (e.g., eye, nasal cavity, respiratory tract) was reported. To account for potential differences in sensitivity and susceptibility between study volunteers and members of the public, the US EPA applied a partial uncertainty factor of 3 to the LOAEL of 320,000 µg/m³, resulting in the acute exposure limit of 110,000 µg/m³. Comparison of the peak predicted air concentration of68 µg/m³ for the aliphatic aldehyde group with the US EPA 1-hour AEGL-1 for propionaldehyde suggests a 1,600-fold margin of safety.

3.1.3 Aromatic C_9-C_{16} Group

The acute RQ values for the aromatic C_9-C_{16} group, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 along the PRM fenceline and at the cabin locations under the 2013 PDC only. Along the PRM fenceline, the peak 1-hour concentration for the aromatic C_9-C_{16} group is predicted to be 150 µg/m³ for the 2013 Base Case, and 162 µg/m³ for the 2013 PRM Application Case, and 3,024 µg/m³ for the 2013 PDC. These peak air concentrations are associated with the maximum predicted RQ values along the PRM fenceline of 0.077 for the 2013 Base Case, 0.081 for the 2013 PRM Application Case, and 1.5 for the 2013 PDC. At the maximum cabin location (Cabin J), the

peak 1-hour concentrations are predicted to be 1,086 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 3,233 µg/m³ for the 2013 PDC. These peak hourly concentrations are associated with the 2013 Base Case and 2013 PRM Application Case RQ of 0.54, and the PDC RQ of 1.6. The peak hourly concentrations of the aromatic C_9-C_{16} group are below the exposure limit of 2,000 µg/m³ at all other locations assessed in the 2013 HHRA.

As the 2013 Base Case peak air concentrations for the aromatic C_9-C_{16} group were predicted to increase only slightly, if at all, at these locations under the 2013 PRM Application Case, the incremental changes in peak predicted air concentrations as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to aromatic C_9-C_{16} group at these locations. For the 2013 PDC, RQ values were predicted to increase along the PRM fenceline and the maximum of the cabin locations to values greater than 1.0. The predicted increase in the peak aromatic C_9-C_{16} group concentrations at these locations can be attributed to other planned future developments in the region.

As previously described, use of the 9th highest hourly value may be a more reasonable concentration to consider for the purposes of the 2013 HHRA, as concentrations of the aromatic C_9-C_{16} group are expected to be equal to or lower than this value 99.9% of the time. The 9th highest hourly aromatic C_9-C_{16} group concentrations are not predicted to exceed the acute exposure limit of 2,000 µg/m³ at any locations assessed in the 2013 HHRA, other than Cabin J for the 2013 PDC. For the acute RQ values predicted using the 9th highest hourly air concentration for the aromatic C_9-C_{16} group, refer to Table 3-9.

Table 3-9 Comparison of Acute Inhalation Risk Quotients for the Aromatic C_9-C_{16} Group Based on the Peak and 9th Highest 1-Hour Concentrations

Receptor Group	Risk Quotients[(1)]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	7.7E-02	8.1E-02	**1.5E+00**	3.1E-02	3.5E-02	5.2E-01
Cabin Locations	5.4E-01	5.4E-01	**1.6E+00**	4.7E-01	4.7E-01	**1.3E+00**
Community Locations	2.4E-02	2.5E-02	7.5E-02	1.8E-02	1.8E-02	4.6E-02
Worker Camp Sites	4.3E-02	4.3E-02	2.3E-01	3.4E-02	3.4E-02	1.1E-01

Notes:
(1) Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

For the locations at which peak 1-hour air concentrations were predicted to exceed the exposure limit of 2,000 µg/m³ for the aromatic C_9-C_{16} group, attention was given to the likelihood of such an exceedance occurring. Frequency analysis of the predicted 1-hour aromatic C_9-C_{16} group air concentrations for 1995 and 2002 was completed for each of these locations; the higher frequency of the two years is presented in Table 3-10. Note that the values in Table 3-10 do not provide any indication as to the level of the exceedance; they simply acknowledge that an exceedance was predicted.

Table 3-10 Predicted Likelihood of Exceeding the 1-Hour Acute Inhalation Exposure Limit for the Aromatic C_9-C_{16} Group

Location	Frequency of Exceedance [%][(1)]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	0	0	0.011
Cabin J	0	0	0.091

Notes:
(1) Values represent the number of hours, as a percentage, that the exposure limit was exceeded on an annual basis.

For the aromatic C_9-C_{16} group, chronic inhalation and oral exposure limits have been developed for the chemical group as a whole. On an acute basis, however, an inhalation exposure limit has not been developed for the group; therefore, a suitable surrogate was required. Acute inhalation exposure limits that met the pre-defined criteria (as described in Section 2.4.1) were identified for two suitable surrogates – naphthalene and trimethylbenzenes.

The acute inhalation exposure limit used in the 2013 HHRA of 2,000 µg/m³ was based on the Short-Term Exposure Limit (STEL) of 79,000 µg/m³ developed by the America Conference of Governmental Industrial Hygienists (ACGIH) for naphthalene. The ACGIH (1992) reports that eye irritation in workers has been observed at naphthalene air concentrations in excess of 79,000 µg/m³. This LOAEL forms the basis of the STEL. The STEL represents a 15-minute air concentration that should not be exceeded at any time during a workday (ACGIH 2013). The 15-minute STEL was adjusted to an equivalent 1-hour concentration using a modified Haber's Law, and a default uncertainty factor was applied to account for differences in sensitivity and susceptibility between workers and members of the public. The result is the acute inhalation exposure limit of 2,000 µg/m³ used in the 2013 HHRA to assess the potential short-term health risks associated with the aromatic C_9-C_{16} group. This suggests that the predicted 9th highest hourly concentration of 2,534 µg/m³ is well below the concentration at which irritation has been reported in the scientific literature. There is a margin of safety of about 31 between the LOAEL (79,000 µg/m³) and the predicted maximum air concentration for the aliphatic aldehyde group (2,534 µg/m³). This comparison with the toxicological threshold for naphthalene suggests that the overall potential for adverse effects in association with short-term exposure to the aromatic C_9-C_{16} group is likely low at the locations assessed in the 2013 HHRA.

The adjusted STEL for naphthalene of 2,000 µg/m³ was selected for use in the 2013 HHRA over the US EPA 1-hour AEGL-1 for trimethylbenzenes of 690,000 µg/m³, despite both limits being supported by the pre-defined criteria, because the adjusted STEL is almost 350 times lower (i.e., more conservative) than the US EPA 1-hour AEGL-1. Comparison of the predicted 9th highest hourly concentration for the aromatic C_9-C_{16} group of 2,534 µg/m³ with the 1-hour AEGL-1 for trimethylbenzenes of 690,000 µg/m³ suggests a 270-fold margin of safety.

Based on the above information, the weight of evidence indicates a low potential for adverse health effects as a result of short-term exposure to the aromatic C_9-C_{16} group.

3.1.4 Sulphur Dioxide (SO_2)

The acute RQ values for SO_2 are predicted to exceed 1.0 at the maximum industrial camp site (Oil Sands Lodge) for the 10-minute averaging time only. The SO_2 air concentrations are predicted to be below the acute exposure limits of 500 µg/m³ (10-minute) and 450 µg/m³ (1-hour) at all other locations assessed in the 2013 HHRA. At the Oil Sands Lodge, the peak 10-minute SO_2 concentrations are predicted to be 647 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 663 µg/m³ for the 2013 PDC. These peak 10-minute air concentrations are associated with the maximum RQ of 1.3 for the industrial camp sites predicted for all three assessment cases. As there is no difference between the 2013 Base Case and 2013 PRM Application Case air concentrations, the PRM is not expected to increase the acute SO_2-related health risks at this location. For the 2013 PDC, the predicted increase in the peak 10-minute SO_2 concentrations at Oil Sands Lodge can be attributed to other planned future developments in the region.

As previously described, use of the 9th highest 10-minute value may be a more reasonable concentration to consider for the purposes of the 2013 HHRA. The 9th highest 10-minute SO_2 concentration is predicted to be 257 µg/m³. Frequency analysis of predicted air concentrations at this location suggests that 10-minute SO_2 concentrations could exceed the acute exposure limit about 0.03% of the time. This suggests that these exceedances are unlikely to occur.

A detailed discussion of the potential health effects at varying concentrations of SO_2 was presented in the EIA, Volume 3, pages 5-98 to 5-100 (see Table 5.3-28). The predicted 10-minute SO_2 concentration of 257 µg/m³ is at the low end of the range of air concentrations where possible modest, transient changes in lung function indices (detectable by spirometry) among asthmatics during moderate to strenuous exercise might occur. All changes in airway resistance would be fully reversible and subclinical in nature, with no evidence of wheezing, shortness of breath or other clinical signs. The PRM is not expected to have a material impact on these risks.

The current conclusions with respect to short-term exposure to SO_2 are consistent with those presented in the EIA HHRA.

3.1.5 Eye Irritants

The acute RQ values for the eye irritants, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 for all of the receptor groups. Along the PRM fenceline, the maximum RQ values are predicted to be 2.2, 2.5 and 3.9 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. At the maximum cabin location (Cabin K), the RQ values are predicted to be 3.4 for the 2013 Base and 2013 PRM Application cases, and 4.3 for the 2013 PDC. The acute RQ values for the eye irritants are predicted to exceed 1.0 at 11 of the 12 cabin locations; acute RQ values are less than 1.0 at Cabin B for all three assessment cases.

At the maximum of the community locations, the RQ values are predicted to be 1.6 for the 2013 Base and 2013 PRM Application cases (Fort McKay), and 2.4 for the 2013 PDC (Fort McMurray). Risk quotients for the eye irritants are less than 1.0 for all remaining community locations, under all three assessment

cases. At the maximum worker location (Jackpine Mine Camp), the RQ values are predicted to be 2.5 for the 2013 Base and 2013 PRM Application cases, and 2.8 for the 2013 PDC. Risk quotients were predicted to be above 1.0 for all other industrial camp sites.

As the 2013 Base Case RQ values are not predicted to change at these locations under the 2013 PRM Application Case, the exception being a slight increase in the 2013 PRM Application Case RQ values along the PRM fenceline, the incremental changes in RQ values as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to eye irritants mixture. The predicted increase in RQ values between the 2013 Base Case and the 2013 PDC can be attributed to the other planned future developments in the region.

As stated previously, it was assumed that there could potentially be an additive interaction among the eye irritants. The RQ values for the individual eye irritants were therefore summed to derive the RQ values for the eye irritants mixture. The constituents of the acute eye irritants mixture are acetaldehyde, acrolein, aliphatic aldehyde group, aromatic C_9-C_{16} group, dichlorobenzenes, formaldehyde, naphthalene and substituted naphthalenes and toluene. Of these, acrolein, the aliphatic aldehyde group and the aromatic C_9-C_{16} group were predicted to exceed their acute inhalation exposure limits. The degree of conservatism incorporated in the acute inhalation RQ values for these compounds or groups of compounds has been previously discussed (see Section 3.1.1 to Section 3.1.3). It follows that the principal contributors to the eye irritants risks are acrolein (25% to 44%), the aliphatic aldehyde group (25% to 44%), and the aromatic C_9-C_{16} group (1% to 41%). Together these compounds and groups contribute between 82% and 88% of the eye irritants risks. Because acrolein, the aliphatic aldehyde group and the aromatic C_9-C_{16} group are the principal contributors to the eye irritants risks, the interpretation of the predicted risks focuses on these three compounds or groups.

Risk quotients resulting from peak (1st highest) predicted hourly air concentrations for all chemicals were used to calculate the mixture RQ values. As previously described, the likelihood of achieving any of these peak concentrations is very low, and achieving them simultaneously is even lower (i.e., much less than 0.1%) as the actual occurrence of the peak predicted air concentrations will vary from chemical to chemical, dependent on such variables as emission rates, physical characteristics of the emission sources and ambient conditions (e.g., meteorology). The use of the peak predicted concentrations in the calculation of the mixture RQ values therefore likely overstates the actual combined risk. Use of the more representative 9th highest hourly acrolein, the aliphatic aldehyde group and the aromatic C_9-C_{16} group air concentrations yields maximum mixture RQ values approximately 4% to 21% lower; however, the combined RQ values for the eye irritants remain above 1.0 for all receptor groups (see Table 3-11).

Table 3-11 Comparison of Acute Inhalation Risk Quotients for the Eye Irritants Based on the Peak and 9th Highest 1-Hour Concentrations

Receptor Group	Risk Quotients[1]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	**2.2E+00**	**2.5E+00**	**3.9E+00**	**1.9E+00**	**2.2E+00**	**2.6E+00**
Cabin Locations	**3.4E+00**	**3.4E+00**	**4.3E+00**	**2.7E+00**	**2.8E+00**	**3.4E+00**
Community Locations	**1.6E+00**	**1.6E+00**	**2.4E+00**	**1.3E+00**	**1.3E+00**	**2.0E+00**
Worker Camp Sites	**2.5E+00**	**2.5E+00**	**2.8E+00**	**2.3E+00**	**2.3E+00**	**2.7E+00**

Notes:
[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

As the peak predicted 1-hour air concentrations for acrolein, the aliphatic aldehyde group and the aromatic C_9-C_{16} group are well below the concentrations at which eye irritation has been observed in humans, there are reasonable margins of safety between the peak predicted air concentrations and their respective effect thresholds. These margins of safety are increased when considering the more representative 9th highest hourly concentrations. Similar conservatism is incorporated in the prediction of the acute RQ values for each of the individual eye irritants. As such, summation of the RQ values for the eight constituents of the eye irritants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the actual combined risks.

3.1.6 Nasal Irritants

The acute RQ values for the nasal irritants, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 for all of the receptor groups. Along the PRM fenceline, the maximum RQ values are predicted to be 1.5, 1.7 and 1.8 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. At the maximum cabin location (Cabin K), the RQ values are predicted to be 2.3 for the three assessment cases. The acute RQ values for the nasal irritants are also predicted to exceed 1.0 at Cabin J, where RQ values of 2.0, 2.1 and 2.0 are predicted for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively.

At the maximum of the community locations, the RQ values are predicted to be 1.2 for the 2013 Base and 2013 PRM Application cases (Fort McKay), and 1.5 for the 2013 PDC (Fort McMurray). At Fort McKay, the RQ of 1.2 is predicted across all three assessment cases. At Fort McMurray, acute RQ values are predicted to be less than 1.0 for the 2013 Base and 2013 PRM Application cases (0.65). At the maximum worker location (Jackpine Mine Camp), the RQ values are predicted to be 2.0 for the 2013 Base and 2013 PRM Application cases, and 2.3 for the 2013 PDC. Risk quotients were predicted to be above 1.0 under all assessment cases at Oil Sands Lodge (1.7). Risk quotients for the nasal irritants are less than 1.0 for all remaining locations assessed in the 2013 HHRA.

As the 2013 Base Case RQ values are not predicted to change at these locations under the 2013 PRM Application Case, the exception being a slight increase in the 2013 PRM Application Case RQ values along the PRM fenceline and Cabin J, the incremental changes in RQ values as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to the nasal irritants. The predicted increase in RQ values between the 2013 Base Case and the 2013 PDC can be attributed to other future developments in the region.

As stated previously, it was assumed that there could potentially be an additive interaction among the nasal irritants. The RQ values for the individual nasal irritants were therefore summed to derive the RQ values for the nasal irritants mixture. The constituents of the acute nasal irritants mixture are acetaldehyde, acrolein, cadmium, dichlorobenzenes, formaldehyde, propylene oxide and toluene. Of these, acrolein was the only mixture component predicted to exceed its acute inhalation exposure limit. The degree of conservatism incorporated in the acute inhalation RQ values for acrolein has been previously discussed. It follows that the primary contributor to the nasal irritants mixture is acrolein (53% to 69%). Cadmium (3% to 26%) and formaldehyde (17% to 23%) are the next largest contributors to the nasal irritant risks; combined these three compounds represent 92% to 96% of the predicted nasal irritant risks. As such, the interpretation of the predicted risks focuses on these compounds.

Risk quotients resulting from peak (1st highest) predicted air concentrations for all chemicals were used to calculate the mixture RQ values. As described previously, the likelihood of achieving any of these peak concentrations is very low, and achieving them simultaneously is even lower (i.e., much less than 0.1%) as the actual occurrence of the peak predicted air concentrations will vary from chemical to chemical, dependent on such variables as emission rates, physical characteristics of the emission sources and ambient conditions (e.g., meteorology). Although conservatively assumed in the 2013 HHRA, in reality, the peak short-term air concentrations of all the nasal irritants are not expected to occur at precisely the same time at any given location. The use of the peak predicted concentrations in the calculation of the mixture RQ values therefore likely overstates the actual combined risk.

Use of the more representative 9th highest hourly acrolein and formaldehyde air concentrations yields mixture RQ values 4% and 30% lower for the various receptor groups (Table 3-12). Based on the 9th highest hourly acrolein and formaldehyde concentrations, the combined RQ values for the nasal irritants are less than 1.0 for the community locations under the 2013 Base and 2013 PRM Application cases. The RQ values for cadmium were not re-evaluated using the 9th highest air concentrations as cadmium was assessed on a 24-hour basis.

Table 3-12 Comparison of Acute Inhalation Risk Quotients for the Nasal Irritants Based on the Peak and 9th Highest 1-Hour Concentrations

Receptor Group	Risk Quotient[(1)]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	**1.5E+00**	**1.7E+00**	**1.8E+00**	**1.3E+00**	**1.6E+00**	**1.6E+00**
Cabin Locations	**2.3E+00**	**2.3E+00**	**2.3E+00**	**1.7E+00**	**1.7E+00**	**1.6E+00**
Community Locations	**1.2E+00**	**1.2E+00**	**1.5E+00**	9.4E-01	9.6E-01	**1.9E+00**
Worker Camp Sites	**2.0E+00**	**2.0E+00**	**2.3E+00**	**1.9E+00**	**1.9E+00**	**2.2E+00**

Notes:
[(1)] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

When evaluating the potential additivity of the nasal irritants, consideration must also be given to the toxicological effects on which the exposure limits for each of the mixture's components are based. The acute inhalation exposure limit for cadmium was derived from toxicological data obtained from mice, while, in contrast, the acute inhalation limits for acrolein and formaldehyde are human-based. There is some evidence that suggests that rodents may be more susceptible to the occurrence of nasal lesions than humans, resulting in the potential over prediction of the cadmium's contribution to the risk associated with the nasal irritants mixture.

Laboratory rodents (i.e., rats, mice, hamsters, guinea pigs) are typically obligate nose breathers, given the proximity of the epiglottis to the soft palate, which prevents breathing from the mouth (Harkema et al. 2006; Reznik 1990). In contrast, the structure of the nasal and oral cavities of humans permits both nasal and mouth breathing (Harkema et al. 2006; Reznik 1990). In addition, there are marked differences in air flow patterns between humans and rodents, primarily because of variation in the shape of the nasal turbinate structures (Harkema et al. 2006). Recent computational modelling has revealed that about 20 percent of inhaled air reaches the olfactory epithelium in rats, while only 3% of inhaled air reaches the olfactory epithelium in humans (Kimbell 2006). As a function of less inhaled air reaching the olfactory epithelium in humans than in rats, lesser amounts of a toxicant would be deposited in the nasal cavity of humans than in macrosmatic species such as the rat (Reznik and Stinson 1983). The result is a lower overall inhaled dose of the toxicant for the human relative to the rat (Harkema et al. 2006). For these reasons, rodent species may be more susceptible to nasal lesions than humans.

Furthermore, the acute exposure limits for acrolein and formaldehyde are based on the incidence of clinical nasal irritation in humans associated with air concentrations of 140 µg/m³ and 500 µg/m³, respectively. As the peak predicted 1-hour air concentration for acrolein of 3.7 µg/m³ and for formaldehyde of 24 µg/m³ are well below the concentrations at which nasal irritation has been observed in humans, there is a reasonable margin of safety between the peak predicted air concentrations and their respective effect thresholds. The margin of safety is increased for both

compounds when considering the more representative 9th highest hourly concentrations. Similar conservatism is incorporated in the prediction of the acute RQ values for each of the individual nasal irritants. As such, summation of the RQ values for the seven constituents of the nasal irritants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the combined risks.

3.1.7 Respiratory Irritants

The acute RQ values for the respiratory irritants, based on the peak (1st highest) hourly air concentrations, are predicted to exceed 1.0 for all of the receptor groups. Along the PRM fenceline, the maximum RQ values are predicted to be 2.2, 2.4 and 3.1 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. At the maximum cabin location (Cabin J), the RQ values are predicted to be 2.9 for the 2013 Base and 2013 PRM Application cases, and 3.1 for the 2013 PDC. The acute RQ values for the respiratory irritants are predicted to exceed 1.0 at 11 of the 12 cabin locations; acute RQ values are less than 1.0 at Cabin B for all three assessment cases.

At the maximum of the community locations (Fort McKay), the RQ values are predicted to be 2.0, 1.9 and 2.0 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. At Fort McMurray, acute RQ values are also predicted to exceed 1.0 for all three assessment cases. Acute RQ values of 1.1 are predicted at Fort McMurray under the 2013 Base and 2013 PRM Application cases, and 2.0 under the 2013 PDC. At the maximum worker location (Oil Sands Lodge), the RQ values are predicted to be 3.2 for the 2013 Base Case, and 3.3 for the 2013 PRM Application Case and 2013 PDC. Risk quotients were predicted to be above 1.0 for all other industrial camp sites.

As the 2013 Base Case RQ values are not predicted to change at these locations under the 2013 PRM Application Case, the exception being a slight increase in the 2013 PRM Application Case RQ values along the PRM fenceline and the Oil Sands Lodge, the incremental changes in RQ values as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with short-term exposure to respiratory irritants mixture. The predicted increase in RQ values between the 2013 Base Case and the 2013 PDC can be attributed to other future developments in the region.

As stated previously, it was assumed that there could potentially be an additive interaction among the respiratory irritants. The RQ values for the individual respiratory irritants were therefore summed to derive the RQ values for the respiratory irritants mixture. The constituents of the acute respiratory irritants mixture are acetaldehyde, acrolein, cadmium, chromium, copper, H_2S, NO_2, SO_2, vanadium, xylenes and zinc. Of these, acrolein and SO_2 are the only mixture components predicted to exceed their acute inhalation exposure limits. It follows that the primary contributor to the respiratory irritants mixture is acrolein (53% to 69%) and SO_2 (10% to 40%). The degree of conservatism incorporated in the acute inhalation RQ values for acrolein and SO_2 have been previously discussed. The next largest contributor is NO_2, representing 17% to 36% of the respiratory irritants risks. Together these three compounds represent 65% to 93% of the predicted respiratory irritant risks. As such, the interpretation of the predicted risks focuses on these compounds.

Risk quotients resulting from peak (1st highest) predicted air concentrations for all chemicals were used to calculate the mixture RQ values. As described previously, the likelihood of achieving any of these peak concentrations is very low, and achieving them simultaneously is even lower (i.e., much less than 0.1%) as the actual occurrence of the peak predicted air concentrations will vary from chemical to chemical, dependent on such variables as emission rates, physical characteristics of the emission sources and ambient conditions (e.g., meteorology). Although conservatively assumed in the 2013 HHRA, in reality, the peak short-term air concentrations of all the respiratory irritants are not expected to occur at precisely the same time at any given location. The use of the peak predicted concentrations in the calculation of the mixture RQ values therefore likely overstates the actual combined risk.

Use of the more representative 9th highest hourly acrolein, SO_2 and NO_2 air concentrations yields mixture RQ values 13% and 30% lower for the various receptor groups (Table 3-13). Based on the 9th highest hourly acrolein, SO_2 and NO_2 concentrations, the combined RQ values for the respiratory irritants remain above 1.0 for all receptor groups.

Table 3-13 Comparison of Acute Inhalation Risk Quotients for the Respiratory Irritants Based on the Peak and 9th Highest 1-Hour and 10-Minute Concentrations

Receptor Group	Risk Quotients[(1)]					
	Based on Peak (1st Highest) Concentrations			Based on 9th Highest Concentrations		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
PRM Fenceline	**2.2E+00**	**2.4E+00**	**3.1E+00**	**1.8E+00**	**2.1E+00**	**2.7E+00**
Cabin Locations	**2.9E+00**	**3.0E+00**	**3.1E+00**	**2.5E+00**	**2.5E+00**	**2.6E+00**
Community Locations	**1.9E+00**	**2.0E+00**	**2.0E+00**	**1.5E+00**	**1.5E+00**	**1.5E+00**
Worker Camp Sites	**3.2E+00**	**3.3E+00**	**3.3E+00**	**2.3E+00**	**2.3E+00**	**2.7E+00**

Notes:
[(1)] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

As part of the interpretation of the risks associated with the respiratory irritants mixture, consideration was given to the mechanism of action by which each of the constituent of the mixture elicit the respiratory effect. For example, acrolein is irritating to mucous membranes, and as a result, its respiratory irritant action is at the point of contact, in part due to interaction with sulphydryl groups within mucosa in the upper airway (TCEQ 2010a). The exposure limit selected for acrolein in the 2013 HHRA is based on subjective, clinical reports of respiratory irritation in exposed individuals. In comparison, the key adverse effect that can occur within minutes of SO_2 exposure is bronchoconstriction which occurs in the lungs, resulting from what is likely a combination of inflammatory and neurological mechanisms (US EPA 2010b). As well, the specific target tissues can differ between the respiratory irritants. Nitrogen dioxide can be inhaled deeply into the lungs, acting as a deep-lung irritant; whereas, SO_2 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). That is, the primary responses of these chemicals occur in different regions of the respiratory tract. Based on the above, assuming that the predicted risks of respiratory irritants are directly additive is likely conservative and results in an overestimate of the actual cumulative risks of experiencing respiratory irritation associated with short-term exposure to the these compounds.

3.2 Chronic Inhalation Results

Chronic inhalation health risks were estimated based on the assumption that residents and workers would be continuously exposed to maximum predicted annual average ground-level concentrations for an assumed lifespan of 80 years (Health Canada 2010a).

Separate assessments were completed for non-carcinogenic and carcinogenic exposures, reflecting the different approaches used in calculating and interpreting the risk estimates.

Chronic inhalation risks were evaluated for the Aboriginal group (i.e., cabin and community locations) and worker group only. The potential inhalation risks to a person active along the PRM fenceline was not evaluated on a chronic basis as it is intended to reflect worst-case exposure to a hypothetical, transient person who might be in the area when worst case emissions and meteorological conditions are occurring. As such, the chronic inhalation pathway is not considered relevant to the PRM fenceline.

3.2.1 Non-Carcinogens

Chronic inhalation health risks, expressed as RQ values, for the Aboriginal group (cabin and community locations) and worker group are presented in Tables 3-14 to 3-16. Note that the risks for the Aboriginal group are “teased out” for the cabin locations and community locations in Table 3-14 and Table 3-15, respectively.

Most of the RQ values are below 1.0, indicating that the predicted air concentrations for those COPC are less than their exposure limits. Adverse health effects are therefore not expected to result from chronic inhalation of these COPC.

Risk quotients were predicted to be greater than 1.0 for the following:

- Cabin locations:
  - aliphatic aldehyde group for all three assessment cases;
  - aromatic C_9-C_{16} group for all three assessment cases;
  - trimethylbenzenes for the 2013 PDC only;
  - eye irritants for all three assessment cases;
  - nasal irritants for all three assessment cases;
  - renal toxicants for all three assessment cases;
  - hepatotoxicants for all three assessment cases; and
  - neurotoxicants for all three assessment cases.
- Community locations:
  - aliphatic aldehyde group for all three assessment cases;
  - eye irritants for all three assessment cases; and

  - nasal irritants for all three assessment cases.
- Worker locations:
  - aliphatic aldehyde group for all three assessment cases;
  - H_2S for the 2013 PRM Application Case and 2013 PDC only;
  - eye irritants for all three assessment cases; and
  - nasal irritants for all three assessment cases.

The significance of each exceedance is discussed in the following sections.

Table 3-14 Chronic Inhalation Risk Quotients – Aboriginal Group (Cabin Locations)

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	NO_2	3.4E-01	3.6E-01	3.5E-01
	$PM_{2.5}$	5.1E-01	5.2E-01	5.5E-01
Organics	1,3-Butadiene	6.5E-03	6.8E-03	6.4E-03
	Acetaldehyde	4.5E-03	4.7E-03	4.5E-03
	Acetone	3.0E-05	3.1E-05	2.9E-05
	Acrolein	4.1E-01	4.3E-01	4.0E-01
	Aliphatic aldehyde group	**2.2E+00**	**2.3E+00**	**2.2E+00**
	Aliphatic C_2-C_4 group	1.3E-03	1.3E-03	1.3E-03
	Aliphatic C_5-C_8 group	1.5E-02	1.5E-02	2.2E-02
	Aliphatic C_9-C_{16} group	3.1E-01	3.1E-01	5.0E-01
	Aromatic C_9-C_{16} group	**1.6E+00**	**1.6E+00**	**2.4E+00**
	Benzene	2.0E-01	2.0E-01	1.2E-01
	Cumene	3.4E-02	3.4E-02	5.1E-02
	Cyclohexane	5.7E-03	5.7E-03	8.6E-03
	Dichlorobenzenes	9.4E-07	9.6E-07	1.6E-06
	Ethylbenzene	3.0E-01	3.0E-01	4.5E-01
	Formaldehyde	8.6E-02	8.9E-02	8.5E-02
	Hexane	1.6E-02	1.6E-02	2.3E-02
	Methyl ethyl ketone group	1.1E-04	1.1E-04	1.1E-04
	Naphthalene and substituted naphthalenes	2.8E-03	3.0E-03	3.4E-03
	Propylene oxide	6.7E-06	8.2E-06	1.2E-05
	Toluene	6.0E-03	6.1E-03	9.1E-03
	Trimethylbenzenes	7.7E-01	7.7E-01	**1.1E+00**
	Xylenes	1.7E-01	1.7E-01	2.6E-01
Sulphur Compounds	CS_2 group	1.5E-03	1.6E-03	3.0E-03
	H_2S	5.1E-02	6.3E-02	1.7E-01

Table 3-14 Chronic Inhalation Risk Quotients – Aboriginal Group (Cabin Locations) (continued)

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Metals	Aluminum	4.1E-04	4.1E-04	4.3E-04
	Barium	3.2E-04	3.3E-04	8.2E-04
	Beryllium	1.4E-04	1.5E-04	3.4E-04
	Cadmium	1.5E-01	1.5E-01	1.7E-01
	Chromium	2.7E-03	2.8E-03	4.2E-03
	Chromium VI	6.9E-04	7.0E-04	1.1E-03
	Cobalt	2.4E-03	2.5E-03	2.7E-03
	Copper	2.8E-04	2.9E-04	4.1E-04
	Manganese	3.1E-03	3.2E-03	3.9E-03
	Mercury	6.2E-04	6.3E-04	1.6E-03
	Molybdenum	7.5E-06	7.6E-06	1.8E-05
	Nickel	1.8E-02	1.8E-02	3.7E-02
	Selenium	3.0E-06	3.0E-06	3.0E-06
	Silver	5.9E-04	6.0E-04	6.3E-04
	Vanadium	2.1E-03	2.1E-03	4.8E-03
Mixtures[2]	Eye irritants	**2.4E+00**	**2.5E+00**	**2.5E+00**
	Nasal irritants	**2.9E+00**	**3.0E+00**	**3.1E+00**
	Respiratory irritants	4.5E-01	4.7E-01	4.9E-01
	Haematotoxicants	2.0E-01	2.0E-01	1.2E-01
	Immunotoxicants	2.0E-01	2.0E-01	1.2E-01
	Renal toxicants	**2.1E+00**	**2.1E+00**	**3.1E+00**
	Hepatotoxicants	**1.6E+00**	**1.6E+00**	**2.4E+00**
	Neurotoxicants	**1.3E+00**	**1.3E+00**	**2.0E+00**
	Reproductive / developmental toxicants	1.2E-02	1.3E-02	1.5E-02

Notes:
[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2] Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
- = Not applicable.

Table 3-15 Chronic Inhalation Risk Quotients – Aboriginal Group (Community Locations)

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	NO_2	3.0E-01	3.0E-01	3.2E-01
	$PM_{2.5}$	8.0E-01	8.1E-01	8.3E-01
Organics	1,3-Butadiene	6.1E-03	6.1E-03	7.1E-03
	Acetaldehyde	4.2E-03	4.2E-03	4.9E-03
	Acetone	2.8E-05	2.8E-05	3.2E-05
	Acrolein	3.8E-01	3.9E-01	4.4E-01
	Aliphatic aldehyde group	**2.0E+00**	**2.0E+00**	**2.4E+00**
	Aliphatic C_2-C_4 group	1.1E-03	1.2E-03	2.1E-03
	Aliphatic C_5-C_8 group	3.9E-03	3.9E-03	4.5E-03
	Aliphatic C_9-C_{16} group	9.3E-02	9.3E-02	1.1E-01
	Aromatic C_9-C_{16} group	7.6E-02	7.7E-02	1.1E-01
	Benzene	1.5E-01	1.5E-01	3.9E-01
	Cumene	3.0E-03	3.0E-03	3.9E-03
	Cyclohexane	2.0E-04	2.1E-04	3.0E-04
	Dichlorobenzenes	9.1E-07	9.1E-07	2.1E-06
	Ethylbenzene	3.0E-03	3.0E-03	8.9E-03
	Formaldehyde	8.0E-02	8.1E-02	9.4E-02
	Hexane	6.6E-03	6.6E-03	7.7E-03
	Methyl ethyl ketone group	1.2E-04	1.2E-04	1.8E-04
	Naphthalene and substituted naphthalenes	1.7E-03	1.8E-03	2.1E-03
	Propylene oxide	5.0E-06	5.3E-06	8.4E-06
	Toluene	9.1E-04	9.1E-04	1.1E-03
	Trimethylbenzenes	1.2E-01	1.2E-01	1.5E-01
	Xylenes	5.7E-03	5.7E-03	8.5E-03
Sulphur Compounds	CS_2 group	8.6E-04	8.8E-04	1.1E-03
	H_2S	4.6E-02	4.7E-02	5.2E-02
Metals	Aluminum	2.8E-04	2.8E-04	2.9E-04
	Barium	2.4E-04	2.4E-04	3.7E-04
	Beryllium	1.2E-04	1.2E-04	1.7E-04
	Cadmium	9.2E-02	9.3E-02	9.8E-02
	Chromium	2.2E-03	2.3E-03	2.6E-03
	Chromium VI	5.7E-04	5.7E-04	6.5E-04
	Cobalt	1.6E-03	1.6E-03	1.6E-03
	Copper	1.9E-04	1.9E-04	2.2E-04
	Manganese	2.3E-03	2.3E-03	2.4E-03
	Mercury	4.4E-04	4.5E-04	7.0E-04
	Molybdenum	6.3E-06	6.3E-06	9.0E-06
	Nickel	2.0E-02	2.0E-02	2.4E-02
	Selenium	2.7E-06	2.7E-06	2.7E-06
	Silver	3.7E-04	3.8E-04	3.8E-04
	Vanadium	1.9E-03	1.9E-03	2.6E-03

Table 3-15 Chronic Inhalation Risk Quotients – Aboriginal Group (Community Locations) (continued)

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Mixtures[2]	Eye irritants	**2.1E+00**	**2.1E+00**	**2.5E+00**
	Nasal irritants	**2.6E+00**	**2.6E+00**	**2.9E+00**
	Respiratory irritants	4.1E-01	4.1E-01	4.4E-01
	Haematotoxicants	1.5E-01	1.5E-01	3.9E-01
	Immunotoxicants	1.5E-01	1.5E-01	3.9E-01
	Renal toxicants	1.8E-01	1.8E-01	2.2E-01
	Hepatotoxicants	7.6E-02	7.7E-02	1.1E-01
	Neurotoxicants	2.4E-01	2.4E-01	2.9E-01
	Reproductive / developmental toxicants	6.4E-03	6.5E-03	7.3E-03

Notes:
[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2]Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.
- = Not applicable.

Table 3-16 Chronic Inhalation Risk Quotients – Worker Group

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	NO_2	3.5E-01	3.5E-01	4.3E-01
	$PM_{2.5}$	5.4E-01	5.5E-01	6.0E-01
Organics	1,3-Butadiene	7.1E-03	7.1E-03	1.1E-02
	Acetaldehyde	4.9E-03	4.9E-03	7.7E-03
	Acetone	3.2E-05	3.3E-05	5.1E-05
	Acrolein	4.4E-01	4.5E-01	7.0E-01
	Aliphatic aldehyde group	**2.4E+00**	**2.4E+00**	**3.7E+00**
	Aliphatic C_2-C_4 group	1.5E-03	1.5E-03	2.2E-03
	Aliphatic C_5-C_8 group	6.7E-03	6.7E-03	7.3E-03
	Aliphatic C_9-C_{16} group	1.7E-01	1.7E-01	1.9E-01
	Aromatic C_9-C_{16} group	1.4E-01	1.4E-01	2.0E-01
	Benzene	5.9E-02	5.9E-02	6.2E-02
	Cumene	4.8E-03	4.8E-03	7.9E-03
	Cyclohexane	3.9E-04	4.0E-04	6.1E-04
	Dichlorobenzenes	1.4E-06	1.5E-06	1.2E-06
	Ethylbenzene	4.1E-03	5.0E-03	2.8E-02
	Formaldehyde	9.3E-02	9.4E-02	1.5E-01
	Hexane	1.3E-02	1.3E-02	1.4E-02
	Methyl ethyl ketone group	1.2E-04	1.2E-04	1.8E-04
	Naphthalene and substituted naphthalenes	2.9E-03	2.9E-03	6.3E-03
	Propylene oxide	8.8E-06	1.7E-05	2.1E-05
	Toluene	1.9E-03	1.9E-03	2.1E-03
	Trimethylbenzenes	2.8E-01	2.8E-01	3.0E-01
	Xylenes	1.0E-02	1.0E-02	1.8E-02
Sulphur Compounds	CS_2 group	1.8E-03	3.2E-03	3.9E-03
	H_2S	1.1E-01	**1.1E+00**	**1.1E+00**
Metals	Aluminum	5.1E-04	5.1E-04	5.9E-04
	Barium	5.1E-04	5.2E-04	5.4E-04
	Beryllium	2.2E-04	2.3E-04	2.3E-04
	Cadmium	1.8E-01	1.8E-01	2.2E-01
	Chromium	3.3E-03	3.4E-03	4.1E-03
	Chromium VI	8.4E-04	8.5E-04	1.0E-03
	Cobalt	3.0E-03	3.0E-03	3.6E-03
	Copper	3.4E-04	3.4E-04	4.3E-04
	Manganese	3.9E-03	3.9E-03	4.6E-03
	Mercury	9.7E-04	9.9E-04	1.1E-03
	Molybdenum	1.2E-05	1.2E-05	1.2E-05
	Nickel	2.8E-02	2.8E-02	2.9E-02
	Selenium	3.1E-06	3.1E-06	3.1E-06
	Silver	7.3E-04	7.3E-04	8.7E-04
	Vanadium	3.3E-03	3.3E-03	3.2E-03

Table 3-16 Chronic Inhalation Risk Quotients – Worker Group (continued)

Chemicals of Potential Concern		Risk Quotients[1]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Mixtures[2]	Eye irritants	**2.5E+00**	**2.5E+00**	**3.9E+00**
	Nasal irritants	**3.0E+00**	**3.0E+00**	**4.8E+00**
	Respiratory irritants	4.7E-01	4.8E-01	6.1E-01
	Haematotoxicants	5.9E-02	6.0E-02	6.3E-02
	Immunotoxicants	5.9E-02	5.9E-02	6.3E-02
	Renal toxicants	2.6E-01	2.6E-01	4.2E-01
	Hepatotoxicants	1.4E-01	1.4E-01	2.0E-01
	Neurotoxicants	4.9E-01	4.9E-01	5.2E-01
	Reproductive / developmental toxicants	7.4E-03	7.4E-03	1.2E-02

Notes:
[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2]Note that addition of the individual constituents' RQ values might not equate to the RQ value provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each lifestyle category, regardless of the location at which exposure occurred.

3.2.1.1 Aliphatic Aldehyde Group

The chronic RQ values for the aliphatic aldehyde group are predicted to exceed 1.0 for the Aboriginal group (cabin and community locations) and the worker group. At the maximum cabin location (Cabin J), the annual average concentrations of the aliphatic aldehyde group are predicted to be 2.6 µg/m³, 2.7 µg/m³ and 2.6 µg/m³ for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. These annual average concentrations area associated with the maximum RQ values of 2.2, 2.3 and 2.2 predicted for the cabin locations under the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. The annual average concentrations also exceed the exposure limit of 1.2 µg/m³ at the following cabin locations:

- Cabin K, where annual average concentrations are predicted to be 2.3 µg/m³ for all three assessment cases.
- Cabin I, where annual average concentrations are predicted to be 1.4 µg/m³ for the 2013 Base Case, and 1.7 µg/m³ for the 2013 PRM Application Case and 2013 PDC.
- Cabin L, where annual average concentrations are predicted to be 1.2 µg/m³ for the 2013 Base Case, and 1.5 µg/m³ for the 2013 PRM Application Case and 2013 PDC.

At the maximum of the community locations, the annual average concentrations are predicted to be 2.4 µg/m³and 2.5 µg/m³ for the 2013 Base and 2013 PRM Application cases (at Fort McKay), and 2.8 µg/m³ for the 2013 PDC (at Fort McMurray). These annual average air concentrations are associated with the maximum RQ values for the community locations of 2.0 for the 2013 Base Case and 2013 PRM Application Case, and 2.4 for the 2013 PDC. At Fort McKay, the 2013 PDC annual average concentration is predicted to remain unchanged from the 2013 PRM Application Case of 2.5 µg/m³. At Fort McMurray,

the annual average concentrations of the aliphatic aldehyde group are not predicted to exceed the chronic exposure limit of 1.2 µg/m³ under the 2013 Base and 2013 PRM Application cases.

At the maximum worker location, the annual average concentrations are predicted to be 2.8 µg/m³ for the 2013 Base Case and 2.9 µg/m³ for the 2013 PRM Application Case (Oil Sands Lodge), and 4.4 µg/m³ for the 2013 PDC (Jackpine Mine Camp). These annual average concentrations are associated with the maximum RQ values for the workers of 2.4 for the 2013 Base Case and 2013 PRM Application Case, and 3.7 for the 2013 PDC. At Oil Sands Lodge, the 2013 PDC annual average concentration is predicted to be 2.8 µg/m³. At Jackpine Mine Camp, the predicted annual average concentrations of the aliphatic aldehyde group are 2.8 µg/m³ under the 2013 Base and 2013 PRM Application cases. The predicted annual average concentrations also exceed the exposure limit of 1.2 µg/m³ at:

- Pierre River Camp, where annual average concentrations are predicted to be 1.1 µg/m³ for the 2013 Base Case, 1.8 µg/m³ for the 2013 PRM Application Case, and 1.9 µg/m³ for the 2013 PDC; and
- PTI Camp, where annual average concentrations are predicted to be 1.3 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 1.5 µg/m³ for the 2013 PDC.

The predicted annual average concentrations of the aliphatic aldehyde group are below the chronic exposure limit of 1.2 µg/m³ at all other locations assessed in the 2013 HHRA.

The degree of conservatism incorporated into the chronic inhalation exposure limit needs to be considered in the interpretation of these RQ values. As was the case in the acute inhalation assessment, the 15 aliphatic aldehydes were evaluated as part of the aliphatic aldehyde group using methacrolein as the surrogate. The constituents of the aliphatic aldehyde group are 3-methylbutanal, benzaldehyde, butanal, crotonaldehyde, decanal, dodecanal, heptanal, hexanal, isobutyraldehyde, methacrolein, nonanal, octanal, propanal (propionaldeyde), tridecanal and undecanal.

The chronic inhalation exposure limit for the group was based on the TCEQ RfC of 1.2 µg/m³ for the respiratory effects of methacrolein. In the key study, rats were exposed to methacrolein concentrations of 0 (control) to 42,000 µg/m³ for 6 hours per day, 5 days per week for 13 weeks. No adverse effects were observed at the lowest exposure concentration of 2,800 µg/m³. Symptoms of eye irritation were observed at both 14,000 µg/m³ (only for the first six exposure days) and 42,000 µg/m³ (not limited to any part of the study). Changes to nasal epithelia and, to a lesser degree, laryngeal epithelia were observed in rats exposed to the highest exposure concentration. Following a recovery period, evidence of regeneration was observed in the respiratory tract of the animals. The TCEQ (2009) identified study NOAELs for two different endpoints: eye irritation (2,800 µg/m³) and respiratory irritation (14,000 µg/m³). These two NOAELs were adjusted for continuous exposure and converted to human equivalent concentrations. The TCEQ then incorporated a 300-fold uncertainty factor to account for differences in sensitivity between rats and humans, differences in sensitivity within the human population, use of subchronic data, and database uncertainties. In the end, the TCEQ calculated two distinct chronic RfCs: 9.3 µg/m³ for eye irritation and 1.2 µg/m³ for respiratory irritation. As the respiratory-based RfC resulted in the more conservative value, the TCEQ (2009) determined respiratory tract irritation to be the critical effect in the derivation of its RfC.

In addition to the TCEQ RfC, the US EPA has derived a chronic RfC of 8 µg/m³ for propionaldehyde based on nasal effects in rats. In the key study, rats were exposed to 0 (control) to 3,750 mg/m³ propionaldehyde for 6 hours per day, 7 days per week for 5 to 7 weeks. The study LOAEL was determined to be 360 mg/m³ for olfactory atrophy. In the derivation of the RfC, the US EPA completed benchmark-dose modelling, adjusted for continuous exposure, calculated a human equivalent concentration, and applied a cumulative uncertainty factor of 1,000 to account for differences in sensitivity between rats and humans, differences in sensitivity in the human population, use of subchronic data, and database uncertainties. This value was not selected for use in the 2013 HHRA due to the presence of the lower (i.e., more conservative) chronic inhalation exposure limit for methacolein. There is a 2-fold margin of safety between the maximum predicted annual air concentration for the aliphatic aldehydes of 4.4 µg/m³ (associated with the RQ of 3.7 for Jackpine Mine Camp) and the US EPA RfC for propionaldehyde.

As previously discussed, recent evidence suggests that rodents might be more susceptible to the occurrence of nasal lesions than humans as a result of higher doses reaching the critical target site or tissue in rodents (Harkema et al. 2006; Reznik 1990; Dorman et al. 1999; Reznik and Stinson 1983; Kimbell 2006). These differences suggest the potential over prediction of the risks associated with the aliphatic aldehyde group.

On this basis, it was concluded that the RQ values based on the methacrolein RfC represent a conservative estimate of the potential risk of adverse health effects associated with the aliphatic aldehyde group, and that adverse health effects as a result of long-term exposures to the aliphatic aldehydes are not expected.

3.2.1.2 Aromatic C_9-C_{16} Group

The chronic RQ values for the aromatic C_9-C_{16} group are predicted to exceed 1.0 at the maximum cabin location (Cabin J) for all three assessment cases. The annual average concentrations of the aromatic C_9-C_{16} group predicted at Cabin J are 79 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 120 µg/m³ for the 2013 PDC. These annual average air concentrations are associated with the maximum RQ values for the cabin locations of 1.6 for the 2013 Base and 2013 PRM Application cases, and 2.4 for the 2013 PDC. The annual average air concentrations of the aromatic C_9-C_{16} group are below the chronic inhalation exposure limit of 50 µg/m^3 at all other locations assessed in the 2013 HHRA.

As the 2013 Base Case annual average air concentrations for the aromatic C_9-C_{16} group are not predicted to change at this cabin location under the 2013 PRM Application Case, the incremental change in predicted air concentrations as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with long-term exposure to the aromatic C_9-C_{16} group at Cabin J. For the 2013 PDC, RQ values were predicted to increase at the maximum cabin location as a result of other planned future developments in the region.

The chronic inhalation exposure limit of 50 µg/m^3 is based on an RfC from the Massachusetts Department of Environmental Protection (MADEP 2003). The MADEP RfC is based on increased liver and kidney weights in male rats exposed to 0 (control) to 1,800 mg/m³ aromatic naphtha, which is primarily

composed of 9-carbon aromatic compounds, 6 hours per day, 5 days per week for 12 months. The MADEP identified a LOAEL of 1,800 mg/m³ for liver and kidney effects. In its derivation of the RfC, the MADEP adjusted a NOAEL of 900 mg/m³ for continuous exposure, and applied a cumulative uncertainty factor of 3,000 to account for the differences in sensitivity between rats and humans, differences in sensitivity in the human population, use of subchronic data, and database deficiencies. Although a human effect concentration could not be identified, comparison of the maximum predicted annual average air concentration of 120 µg/m³ for the aromatic C_9-C_{16} group (associated with the RQ of 2.4 for Cabin J) with the concentration at which adverse health effects have been observed in rats (1,800 mg/m³), suggests a potential 15,000-fold margin of safety.

The EIA HHRA used an exposure limit of 200 µg/m^3 from the Canadian Council of Ministers of the Environment (CCME 2008) for the aromatic C_9-C_{16} group. The CCME limit is based on the same study MADEP used to develop its RfC, the difference being that the CCME did not apply MADEP's uncertainty factor for the suggested deficiencies in the database. The CCME RfC of 200 µg/m³ was not used in the chronic inhalation effects assessment, as the MA DEP RfC represents a more conservative limit. None of the predicted aromatic C_9-C_{16} group air concentrations exceed the CCME RfC of 200 µg/m^3 (CCME 2008). There is almost a 2-fold margin of safety between the maximum predicted annual air concentration for the aromatic C_9-C_{16} group of 120 µg/m³ and the CCME RfC.

Based on the above information, it was concluded that adverse health effects as a result of long-term exposures to the aromatic C_9-C_{16} group are not expected.

3.2.1.3 Hydrogen Sulphide (H_2S)

The chronic RQ values for H_2S are predicted to exceed 1.0 at the maximum worker location (Pierre River Mine Camp) for the 2013 PRM Application Case and 2013 PDC only. The annual average concentrations of H_2S predicted at the Pierre River Mine Camp are 0.056 µg/m³ for the 2013 Base Case, and 2.1 µg/m³ for the 2013 PRM Application Case and 2013 PDC. The annual average air concentrations of H_2S are below the chronic inhalation exposure limit of 2 µg/m^3 at all other locations assessed in the 2013 HHRA, even along the PRM fenceline. In general, maximum PRM-related air concentrations are predicted to occur in close proximity to the PRM emission sources and predicted to decrease with increasing distance from these sources. As public access to the PRM Development Area will be restricted, adverse health effects would not be expected for members of the general public active even in the immediate vicinity of the PRM.

The chronic inhalation limit for H_2S was based on the RfC developed by the US EPA for incidence of nasal lesions reported in a rat inhalation study. In the key study, rats were exposed to 0 (control) to 111 mg/m³ of H_2S for 6 hours per day, 7 days per week for 10 weeks. The US EPA identified a LOAEL of 42 mg/m³ for olfactory loss. In the derivation of its RfC, the US EPA adjusted the NOAEL of 13.9 mg/m³ for continuous exposure, calculated a human equivalent concentration, and applied a cumulative uncertainty factor of 300 to account for differences in sensitivity between rats and humans, differences in sensitivity within the human population, and subchronic exposure duration. Comparison of the maximum annual average air concentration of 2.1 µg/m³ with the lowest concentration at which nasal

effects have been observed in rats (42,000 µg/m³) suggests that there is a low potential for adverse effects in workers from the long-term inhalation of H_2S.

As previously discussed, recent evidence suggests that rodents might be more susceptible to the occurrence of nasal lesions than humans as a result of higher doses reaching the critical target site or tissue in rodents (Harkema et al. 2006; Reznik 1990; Dorman et al. 1999; Reznik and Stinson 1983; Kimbell 2006). These differences suggest the potential over prediction of the risks associated with H_2S.

Furthermore, it is important to recognize that the chronic inhalation limit was developed for the protection of the general public, including sensitive or susceptible individuals such as infants and young children, the elderly and individuals with compromised health. Use of such a limit to evaluate the potential risks to worker health is recognized as conservative. For illustrative purposes, the maximum annual average air concentration of the worker locations of 2.1 µg/m³ was compared with the ACGIH TLV-TWA of 1,400 µg/m³ designed to protect workers repeatedly exposed 5 hours per day, 40 hours per week for the duration of their employment. Comparison of the maximum annual average air concentration with this TLV-TWA further supports the conclusion that there is a low potential for adverse effects in workers from long-term inhalation of H_2S.

3.2.1.4 Trimethylbenzenes

The chronic RQ values for trimethylbenzenes are predicted to exceed 1.0 at the maximum cabin location (Cabin J) for the 2013 PDC only. The annual average concentrations of trimethylbenzenes predicted at this cabin location are 3.9 µg/m³ for the 2013 Base and 2013 PRM Application cases, and 5.7 µg/m³ for the 2013 PDC. These annual average trimethylbenzene concentrations are associated with the maximum RQ values for the cabin locations of 0.77 for the 2013 Base and 2013 PRM Application cases, and 1.1 for the 2013 PDC. The annual average air concentrations of trimethylbenzenes are below the exposure limit of 5 $\mu g/m^3$ at all other locations assessed in the 2013 HHRA.

Given that the 2013 Base Case annual average air concentrations for the trimethylbenzenes are not predicted to change at this cabin location under the 2013 PRM Application Case, the incremental change in predicted air concentrations as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with long-term exposure to the trimethylbenzenes at Cabin J. For the 2013 PDC, RQ values were predicted to increase at the maximum cabin location as a result of other planned future developments in the region.

The chronic inhalation exposure limit for trimethylbenzenes is based on a PPRTV developed by the US EPA of 5 $\mu g/m^3$ for neurotoxicity. In the key study, rats were exposed to 0 (control) to 1,300 mg/m³ 1,2,4-trimethylbenzene or 1,2,3-trimethylbenzene for 6 hours per day, 5 days per week for 3 months. Exposure to 1,2,3-trimethylbenzene at exposure concentrations as low as 130 mg/m³ was associated with changes in sensitivity. For 1,2,4-trimethylbenzene, changes in sensitivity were not observed until 500 mg/m³. Recovery in pain sensitivity was observed following a 2-week recovery period after the last exposure. The US EPA conducted benchmark dose modelling based upon the pain sensitivity data for 1,2,3-trimethylbenzene, calculated a human equivalent concentration, and applied a cumulative uncertainty factor of 3,000 to account for differences in sensitivity between rats and humans,

differences in sensitivity within the human population, subchronic exposure duration, and database uncertainties. Although a human effect concentration could not be identified, comparison of the maximum predicted annual average air concentration of 5.7 µg/m³ for the trimethylbenzenes (associated with the RQ of 1.1) with the concentration at which adverse health effects have been observed in rats (130 mg/m³), suggests a potential 22,800-fold margin of safety.

In addition, the US EPA has developed a PPRTV of 7 µg/m³ for 1,2,4-trimethylbenzene for haematological and respiratory lesions in rats. Rats were exposed to concentration of 0 (control) to 1,230 mg/m³ of 1,2,4-trimethylbenzene for 6 hours per day, 5 days per week for 3 months. The US EPA determined that the study NOAEL was 123 mg/m³ based on the incidence of haematological and respiratory lesions at and above 492 mg/m³. The US EPA adjusted the NAOEL for continuous exposure, calculated a human equivalent concentration, and applied a cumulative uncertainty factor of 3,000 to account for differences in sensitivity between rats and humans, differences in sensitivity within the human population, subchronic exposure duration, and database uncertainties. This value was not selected for use in the 2013 HHRA, as the PPRTV for 1,2,3-trimethylbenzene is slightly lower (i.e., more conservative), and based upon benchmark dose modelling. None of the predicted air concentrations for trimethylbenzenes exceed the PPTRV of 7 µg/m³ for 1,2,4-trimethylbenzene (CCME 2008).

The US EPA develops the PPRTVs specifically for their Superfund (contaminated lands) program and acknowledges that they have not undergone the multi-program review and consensus required for the eventual development of a regulatory-endorsed toxicity value. As such, there is considerable uncertainty associated with the trimethylbenzene exposure limit used in the 2013 HHRA and the risks should be interpreted accordingly. At this time, there are no chronic regulatory limits for the protection of public health available for trimethylbenzenes.

Based on the above information, it was concluded that adverse health effects as a result of long-term exposure to trimethylbenzenes are not expected.

3.2.1.5 Eye Irritants

The chronic RQ values for the eye irritants are predicted to exceed 1.0 for all of the receptor groups. At the maximum cabin location (Cabin J), the RQ values are predicted to be 2.4 for the 2013 Base and 2013 PRM Application cases, and 2.5 for the 2013 PDC. The chronic RQ values exceed 1.0 at Cabin I, K and L as well. At the maximum of the community locations, the RQ values are predicted to be 2.1 for the 2013 Base and 2013 PRM Application cases (Fort McKay), and 2.5 for the 2013 PDC (Fort McMurray). At the maximum worker location, the RQ values are predicted to be 2.5 for the 2013 Base and 2013 PRM Application cases (Oil Sands Lodge), and 3.9 for the 2013 PDC (Jackpine Mine Camp). Risk quotients are also predicted to be above 1.0 at Pierre River Camp and PTI Camp. Risk quotients for the eye irritants are less than 1.0 for all remaining locations assessed in the 2013 HHRA.

Given that the 2013 Base Case RQ values for the eye irritants mixture are not predicted to change at these locations under the 2013 PRM Application Case, the incremental change in predicted RQ values as a result of PRM emissions are essentially negligible, and the PRM will have very little to no impact on the 2013 Base Case health risks associated with long-term exposure to the eye irritants.

As stated previously, it was assumed that there could potentially be an additive interaction among the eye irritants. The RQ values for the individual eye irritants were therefore summed to derive the RQ values for the eye irritants mixture. The constituents of the chronic eye irritants mixture are the aliphatic aldehyde group, formaldehyde and xylenes. Of these, the aliphatic aldehyde group was predicted to exceed its chronic inhalation exposure limits. It follows that the principal contributor to the eye irritant risks is the aliphatic aldehyde group (86% to 96%). The second largest contributor is formaldehyde. Together the aliphatic aldehyde group and formaldehyde contribute between 90% and 100% of the eye irritant risks. Because aliphatic aldehyde group and formaldehyde are the principal contributors to the eye irritants risks, the interpretation of the predicted risks focuses on these two compounds.

The degree of conservatism incorporated in the chronic inhalation RQ values for the aliphatic aldehyde group has been previously discussed (see Section 3.2.1.1).

For formaldehyde, the chronic inhalation exposure limit of 11 µg/m³ is based on the incidence of eye, nasal and respiratory irritation in exposed workers (TCEQ 2008). In the key occupational study, workers were exposed to a mean formaldehyde concentration of 260 µg/m³ for an average duration of 10 years. Exposed workers were compared with a control group of non-occupationally exposed workers who on average, were exposed to 90 µg/m³. Both groups of workers included individuals with hypersensitivity to formaldehyde in cutaneous tests. Eye irritation, and immune-mediated discomfort and irritation of the nasal passages and respiratory tract were observed in the exposed group but not in the control group. On this basis, the study LOAEL was identified as 260 µg/m³ and the NOAEL as 90 µg/m³. Three other human studies were examined with similar LOAEL and NOAEL values. The TCEQ adjusted the NOAEL of 90 µg/m³ for continuous exposure, and applied an uncertainty factor of 3 to account for differences in sensitivity within the human population. Comparison of the maximum predicted annual air concentration of 1.6 µg/m³ for the formaldehyde with the lowest concentration at which eye irritation has been observed in humans (260 µg/m³), suggests a potential 160-fold margin of safety. Similar conservatism is incorporated in the prediction of the chronic RQ values for each of the individual eye irritants. As such, summation of the RQ values for the three constituents of the eye irritants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the combined risks.

Based on the above information, the weight of evidence indicates a low potential for adverse health effects as a result of combined exposure to the components of the eye irritants mixture.

3.2.1.6 Nasal Irritants

The chronic RQ values for the nasal irritants are predicted to exceed 1.0 for all of the receptor groups. At the maximum cabin location (Cabin J), the RQ values are predicted to be 2.9, 3.0 and 3.1 for the 2013 Base Case, 2013 PRM Application Case, and 2013 PDC, respectively. The chronic RQ values also exceed 1.0 at Cabin G, H, I, K and L. At the maximum of the community locations, the RQ values are predicted to be 2.6 for the 2013 Base and 2013 PRM Application cases (Fort McKay), and 2.9 for the 2013 PDC (Fort McMurray). At the maximum worker location (Jackpine Mine Camp), the RQ values are predicted to be 3.0 for the 2013 Base and 2013 PRM Application cases, and 4.8 for the 2013 PDC. Risk quotients are also

predicted to be above 1.0 at Pierre River Camp and PTI Camp. Risk quotients for the nasal irritants are less than 1.0 for all remaining locations assessed in the 2013 HHRA.

With the exception of the maximum of the cabin locations (Cabin J), the differences between the nasal irritant risks for the 2013 Base Case and 2013 PRM Application Case are negligible, indicating that the PRM will not materially increase the nasal irritant risks in the region.

The nasal irritants mixture is comprised of acetaldehyde, acrolein, aliphatic aldehyde group, aliphatic C_2-C_4 group, dichlorobenzenes, formaldehyde, H_2S, naphthalene and substituted naphthalenes, nickel, propylene oxide, and xylenes. The principal contributors to the nasal irritant risks for the Aboriginal group (cabin and community locations) and worker group are acrolein (10% to 15%), the aliphatic aldehyde group (51% to 81%) and H_2S (0.4% to 36%). Because acrolein, the aliphatic aldehyde group and H_2S are the principal contributors to the nasal irritants risks, the interpretation of the predicted risks for the mixture focuses on these three compounds.

Of the 11 constituents of the mixture, the aliphatic aldehyde group and H_2S (Pierre River Mine Camp only) were the only constituents predicted to exceed their chronic inhalation exposure limits. The degree of conservatism incorporated in the chronic inhalation RQ values for the aliphatic aldehyde group and H_2S has been previously discussed (see Section 3.2.1.1 and Section 3.2.1.3, respectively).

The chronic exposure limit of 0.35 µg/m³ for acrolein was developed by the California OEHHA (2008) based on a subchronic inhalation study in rats. The California OEHHA identified a NOAEL of 460 µg/m³ based on the epithelial lesions in the upper airways of exposed rats at concentrations greater than 1,380 µg/m³. The segment of the airway most affected by acrolein exposure was found to be the nasal cavity, where both respiratory and olfactory epithelial lesions were observed in association with a dose-response relationship. The maximum predicted annual air concentration of 0.24 µg/m³ (Jackpine Mine Camp) is about 1,900-times lower than the NOAEL of 460 µg/m³ from the study used by the California OEHHA. Because of the adjustments applied by the OEHHA (2008) in the derivation of the exposure limit, there is a considerable margin of safety between the exposure limit and the actual concentration at which effects have been observed.

As previously discussed, recent evidence suggests that rodents might be more susceptible to the occurrence of nasal lesions than humans as a result of higher doses reaching the critical target site or tissue in rodents (Harkema et al. 2006; Reznik 1990; Dorman et al. 1999; Reznik and Stinson 1983; Kimbell 2006). These differences suggest the potential over prediction of the risks associated with acrolein, the aliphatic aldehyde group, H_2S, and the nasal irritants as a whole.

Similar conservatism is incorporated in the prediction of the chronic RQ values for each of the individual nasal irritants. As such, summation of the RQ values for the 11 constituents of the nasal irritants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the combined risks.

For the reasons stated, the weight of evidence indicates a low potential for adverse nasal effects to occur as a result of the PRM emissions.

3.2.1.7 Renal Toxicants

The chronic RQ values for the renal (kidney) toxicants are predicted to exceed 1.0 for the maximum of the cabin locations only. At the maximum cabin location (Cabin J), the RQ values are predicted to be 2.1 for the 2013 Base and 2013 PRM Application cases, and 3.1 for the 2013 PDC. Risk quotients for the renal toxicants are less than 1.0 for all remaining locations assessed in the 2013 HHRA. There is no change predicted between the renal toxicants risks for the 2013 Base Case and 2013 PRM Application Case, indicating that the PRM will not materially increase the risks associated with long-term exposure to the renal toxicants at this location.

The renal toxicants mixture is comprised of the aliphatic C_2-C_4 group, aromatic C_9-C_{16} group, cadmium, cumene and ethylbenzene. At Cabin J, the aromatic C_9-C_{16} group (77% to 78%) and ethylbenzene (14% to 15%) contribute 91% and 93% to the overall kidney risks. The degree of conservatism incorporated in the chronic inhalation RQ values for the aromatic C_9-C_{16} group has been previously discussed (see Section 3.2.1.2).

The chronic exposure limit for ethylbenzene was developed by the ATSDR (2013, 2010) for increased severity of nephropathy. In the key study, rats and mice were exposed to 0 (control) to 3,300 mg/m³ ethylbenzene via inhalation 6 hours per day, 5 days per week for 103 to 104 weeks. The severity of nephropathy was minimal to mild at the lowest exposure concentration of 330 mg/m³. On this basis, the ATSDR (2013, 2010) identified a LOAEL of 330 mg/m³; however, the TCEQ (2010b) and OEHHA (2000) determined this exposure concentration as the NOAEL for increased severity of nephropathy stating that clinical findings and survival were unaffected by treatment, and the severity of nephropathy was similar to the control group. The ATSDR adjusted the LOAEL for calculating a human equivalent concentration, and applied a cumulative uncertainty factor of 300 to account for use of an effect concentration, differences in sensitivity between rats and humans, and differences in sensitivity within the human population. The chronic exposure limit developed by the ATSDR was used in the chronic inhalation assessment of ethylbenzene, instead of the TCEQ and OEHHA limits based on the same key study, as it incorporates dosimetry modelling to partially account for the uncertainty associated with extrapolation from rats to humans, and is the most conservative (i.e., lowest) of the limits. For illustrative purposes, the maximum annual ethylbenzene concentration of 117 µg/m³ was compared with the LOAEL of 330,000 µg/m³. This comparison suggests that there is a considerable margin of safety between the maximum annual ethylbenzene concentration and the actual concentration at which effects have been observed.

Similar conservatism is incorporated in the prediction of the chronic RQ values for each of the individual renal toxicants. As such, summation of the RQ values for the five constituents of the renal toxicants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the combined risks.

3.2.1.8 Hepatotoxicants

Like the kidney toxicants, the chronic RQ values for the hepatotoxicants (liver) are predicted to exceed 1.0 for the maximum of the cabin locations only. At the maximum cabin location (Cabin J), the RQ values

are predicted to be 1.6 for the 2013 Base and 2013 PRM Application cases, and 2.4 for the 2013 PDC. Risk quotients for the hepatotoxicants are less than 1.0 for all other locations assessed in the 2013 HHRA. There is no change predicted between the hepatotoxicants risks for the 2013 Base Case and 2013 PRM Application Case, indicating that the PRM will not materially increase the risks associated with long-term exposure to the hepatotoxicants at this location.

The two constituents of the liver toxicant mixture are the aromatic C_9-C_{16} group and selenium. The aromatic C_9-C_{16} group contributes approximately 100% to the overall risks. As such, interpretation of the liver toxicant risks reflects the discussion of the aromatic C_9-C_{16} group health risks, and the weight of evidence indicates a low potential for adverse health effects as a result of combined exposure to the components of the hepatotoxicants mixture.

3.2.1.9 Neurotoxicants

The chronic RQ values for the neurotoxicants are predicted to exceed 1.0 for the maximum of the cabin locations only. At the maximum cabin location (Cabin J), the RQ values are predicted to be 1.3 for the 2013 Base and 2013 PRM Application cases, and 2.0 for the 2013 PDC. Risk quotients for the neurotoxicants are less than 1.0 for all remaining locations assessed in the 2013 HHRA. There is no change predicted between the neurotoxicants risks for the 2013 Base Case and 2013 PRM Application Case, indicating that the PRM will not materially increase the risks associated with long-term exposure to the neurotoxicants at this location.

The constituents of the neurotoxicants mixture are acetone, aliphatic C_5-C_8 group, aliphatic C_9-C_{16} group, aluminum, CS_2 group, hexane, manganese, mercury, selenium, toluene, trimethylbenzenes and xylenes. None of these compounds were predicted to exceed their chronic inhalation exposure limits. At Cabin J, the principal contributors to the neurotoxicants risks are the aliphatic C_9-C_{16} group (24% to 25%) and trimethylbenzenes (58% to 60%). Together these compounds represent 84% of the risks at Cabin J. Because aliphatic C_9-C_{16} group and trimethylbenzenes are the principal contributors to the risks, the interpretation of the predicted neurotoxicants risks focuses on these two compounds.

The degree of conservatism incorporated in the chronic inhalation RQ values for the trimethylbenzenes has been previously discussed (see Section 3.2.1.4).

The chronic inhalation exposure limit for the aliphatic C_9-C_{16} group is the RfC of 200 µg/m³ developed by MA DEP based on a subchronic rat inhalation study (MA DEP 2003). In the key study, rats were exposed to de-aromatized white spirit vapour 6 hours per day, 5 days per week for 6 months. A LOAEL of 2,600 mg/m³ was determined based on the incidence of neurobehavioural effects (MA DEP 2003). The LOAEL was adjusted for continuous exposure, and an uncertainty factor of 3,000 was applied to account for differences in sensitivity between rats and humans, differences in sensitivity within the human population, subchronic exposure duration, and use of an effect concentration. Although human effect concentrations could not be identified, MADEP (2003) states in its review of the toxicological data that significant neuropsychological disorders have been observed in workers occupationally exposed to white petroleum spirits. This suggests that the endpoint from the rodent study that formed the basis of the RfC is relevant to humans. Comparison of the maximum predicted annual average air concentration

of 99 µg/m³ for the aliphatic C_9-C_{16} group (associated with the RQ value of 0.50 for Cabin J) with the concentration at which neurological effects have been observed in rats, suggests a potential 26,000-fold margin of safety.

Furthermore, the CCME (2008) recommends an RfC of 1,000 µg/m³. In the key study, mice and rats were continuously exposed to JP-8 vapours of up to 1,000,000 µg/m³ for 90 days. No effects were observed at the highest concentration. A cumulative uncertainty factor of 1,000 was applied to the NOAEL. This limit was not selected for use in the chronic inhalation assessment due to the presence of the more conservative (and defensible) MA DEP limit. There is a 10-fold margin of safety between the maximum predicted annual average air concentration for the aliphatic C_9-C_{16} group of 99 µg/m³ and the CCME limit.

Similar conservatism is incorporated in the prediction of the chronic RQ values for each of the individual neurotoxicants. As such, summation of the RQ values for the 12 constituents of the neurotoxicants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the combined risks.

3.2.2 Carcinogens

Inhalation health risks for the COPC, expressed as ILCR values, are presented in Table 3-17 to Table 3-19 for the Aboriginal group (cabin and community locations) and worker group. Note that the risks for the Aboriginal group are “teased out” for the cabin locations and community locations in Table 3-17 and Table 3-18, respectively. Cancer risks are presented for PRM alone (i.e., 2013 PRM Application Case minus 2013 Base Case) and planned future emission sources (i.e., 2013 PDC minus 2013 Base Case).

As shown, the predicted ILCR values associated with the PRM alone and the planned future emission sources are less than 1 in 100,000 for the cabin and industrial camp locations, indicating that the ILCR from the PRM and planned future emission sources are deemed to be essentially negligible. For the community locations, the ILCR values for benzene and the leukemogens exceed 1 in 100,000 for the planned future emission sources only.

Table 3-17 Chronic Inhalation Incremental Lifetime Cancer Risks – Aboriginal Group (Cabin Locations)

Chemical of Potential Concern		Incremental Lifetime Cancer Risks (per 100,000)[1]	
		Pierre River Mine	Future
Organics	1,3-Butadiene	5.3E-03	5.4E-03
	Acetaldehyde	1.2E-02	1.3E-02
	Benzene	4.9E-02	1.3E-01
	Benzo(a)pyrene group (Approach 1)	5.4E-02	1.3E-01
	Benzo(a)pyrene group (Approach 2)	2.8E-04	6.0E-04
	Formaldehyde	1.5E-01	1.5E-01
	Propylene oxide	3.0E-05	6.4E-05
Metals	Arsenic	1.3E-03	1.7E-02
	Beryllium	3.1E-05	4.0E-04
	Cadmium	5.6E-02	1.2E-01
	Chromium VI	4.3E-02	2.8E-01
	Nickel	2.8E-03	3.6E-02
Mixtures[2]	Nasal carcinogens	1.6E-01	1.7E-01
	Lung carcinogens	1.6E-01	5.4E-01
	Leukemogens	5.1E-02	1.3E-01

Notes:
[1] An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2] Individual constituents of the chemical mixtures are identified in Table 2-11. Note that addition of the individual ILCR values provided in the above table for a mixture's chemical constituents might not equate to the ILCR provided for the mixture because the ILCR values in the table represent the maximum ILCR for each receptor group, regardless of the location at which it occurred.

Table 3-18 Chronic Inhalation Incremental Lifetime Cancer Risks – Aboriginal Group (Community Locations)

Chemical of Potential Concern		Incremental Lifetime Cancer Risks (per 100,000)[1]	
		Pierre River Mine	Future
Organics	1,3-Butadiene	3.4E-04	2.6E-02
	Acetaldehyde	8.1E-04	6.2E-02
	Benzene	2.6E-03	**1.8E+00**
	Benzo(a)pyrene group (Approach 1)	4.8E-03	5.4E-02
	Benzo(a)pyrene group (Approach 2)	2.3E-05	2.4E-04
	Formaldehyde	9.7E-03	7.2E-01
	Propylene oxide	6.5E-06	3.5E-05
Metals	Arsenic	2.1E-04	3.7E-03
	Beryllium	5.1E-06	8.8E-05
	Cadmium	3.9E-03	2.7E-02
	Chromium VI	4.2E-03	6.4E-02
	Nickel	4.7E-04	8.0E-03
Mixtures[2]	Nasal carcinogens	1.0E-02	7.9E-01
	Lung carcinogens	1.3E-02	1.6E-01
	Leukemogens	2.9E-03	**1.8E+00**

Notes:

[1] Values in **bold** indicate that the ILCR is greater than 1.0. An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

[2] Individual constituents of the chemical mixtures are identified in Table2-11. Note that addition of the individual ILCR values provided in the above table for a mixture's chemical constituents might not equate to the ILCR provided for the mixture because the ILCR values in the table represent the maximum ILCR for each receptor group, regardless of the location at which it occurred.

Table 3-19 Chronic Inhalation Incremental Lifetime Cancer Risks – Worker Group

Chemical of Potential Concern		Incremental Lifetime Cancer Risks (per 100,000)[1]	
		Pierre River Mine	Future
Organics	1,3-Butadiene	1.2E-02	2.8E-02
	Acetaldehyde	2.8E-02	6.6E-02
	Benzene	6.2E-02	1.5E-01
	Benzo(a)pyrene group (Approach 1)	1.3E-01	3.5E-01
	Benzo(a)pyrene group (Approach 2)	6.6E-04	1.8E-03
	Formaldehyde	3.4E-01	7.6E-01
	Propylene oxide	1.2E-04	1.6E-04
Metals	Arsenic	4.7E-03	1.1E-02
	Beryllium	1.1E-04	2.6E-04
	Cadmium	1.6E-01	4.6E-01
	Chromium VI	1.4E-01	2.4E-01
	Nickel	1.0E-02	2.4E-02
Mixtures[2]	Nasal carcinogens	3.7E-01	8.3E-01
	Lung carcinogens	4.4E-01	9.7E-01
	Leukemogens	7.4E-02	1.8E-01

Notes:
[1] An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2] Individual constituents of the chemical mixtures are identified in Table 2-11. Note that addition of the individual ILCR values provided in the above table for a mixture's chemical constituents might not equate to the ILCR provided for the mixture because the ILCR values in the table represent the maximum ILCR for each receptor group, regardless of the location at which it occurred.

3.2.2.1 Benzene

The chronic ILCR values for benzene are predicted to exceed 1 in 100,000 for the maximum of the community locations only. At the maximum community location, the ILCR values are predicted to be 0.0026 in 100,000 for the PRM alone (Poplar Point), and 1.8 in 100,000 for the planned future emission sources (Fort McMurray). At Fort McMurray, the ILCR for the PRM alone is 0.00051 in 100,000. Incremental lifetime cancer risks for benzene are less than 1 in 100,000 for all remaining locations assessed in the 2013 HHRA.

As shown in Table 3-18, the maximum ILCR for the PRM alone is predicted to be below 1 in 100,000, indicating that the incremental cancer risk from the PRM is deemed to be "essentially negligible" (Health Canada 2010a).

The ILCR of 1.8 in 100,000 predicted for the planned future emission sources can be attributed to the projected increase in the Fort McMurray population and the associated increase in benzene emissions. The primary sources of benzene in Fort McMurray are gas stations, vehicle traffic and home heating. Emission factors from reputable published sources as well as assumptions on the number of vehicles and homes were used to estimate the emissions from these sources. The 2013 Base Case Fort McMurray population of 76,797 results in 0.095 tonnes/day of benzene, while the 2013 PDC used a forecasted population for the year 2030 of 166,834 resulting in 0.206 tonnes/day benzene. Emissions from

communities are difficult to quantify accurately; therefore, there is a high level of uncertainty associated with these predictions.

The chronic inhalation exposure limit of 1.3 µg/m³ used in the carcinogenic assessment of benzene was obtained from the US EPA (2013, 2000). Health Canada also provides a risk specific concentration (RsC) of 3 µg/m³ for benzene based on the same study used by the US EPA. This limit was not used in the carcinogenic assessment of benzene due to the presence of the lower (i.e., more conservative) limit developed by the US EPA. The maximum predicted annual benzene concentration of 2.3 µg/m³ for the planned future emission sources (i.e., 2013 PDC minus 2013 Base Case), which is associated with the ILCR of 1.8 for Fort McMurray, is less than the Health Canada RsC of 3 µg/m³ for benzene (Health Canada 2010b).

Overall, the incremental cancer risk from the PRM is deemed to be "essentially negligible" (Health Canada 2010a) and, despite the predicted exceedance for Fort McMurray as a result of the projected increase in the Fort McMurray population, the weight-of-evidence suggests that there is low potential for an increase in a person's lifetime risk of developing cancer as a result of long-term exposure to benzene in the region.

3.2.2.2 Leukemogens

Like benzene, the ILCR values for the leukemogens are predicted to exceed 1 in 100,000 for the maximum of the community locations only. At the maximum community location, the ILCR values are predicted to be 0.0029 in 100,000 for the PRM alone (Poplar Point), and 1.8 in 100,000 for the planned future emission sources (Fort McMurray). At Fort McMurray, the ILCR for the PRM alone is 0.00058 in 100,000. Incremental lifetime cancer risks for the leukemogens are less than 1 in 100,000 for all remaining locations assessed in the 2013 HHRA.

The two constituents of the leukemogens mixture are the 1,3-butadiene and benzene. Benzene contributes approximately 100% to the overall risks. As such, interpretation of the leukemogen risks reflects the discussion of the benzene health risks presented in Section 3.2.2.1. Overall, the incremental cancer risk from the PRM is deemed to be "essentially negligible" (Health Canada 2010a) and, despite the predicted exceedance for Fort McMurray as a result of the projected increase in the Fort McMurray population, the weight-of-evidence suggests that there is low potential for an increase in a person's lifetime risk of developing leukemia in the region.

3.3 Chronic Multiple Pathway Assessment

As in the chronic inhalation assessment, health risks were estimated for the Aboriginal group (cabin and community locations) exposed via secondary exposure pathways over their entire lifespan of 80 years (Health Canada 2010a). In the case of the workers, it was assumed that secondary pathway exposures would be limited to the 60 years of their adult life. The potential health risks to a person active along the PRM fenceline was not evaluated on a chronic basis as it is intended to reflect exposure to a transient person who will not be in the area for prolonged periods of time. As such, secondary exposure pathways are not considered relevant to the PRM fenceline.

The multiple pathway assessment focused on those COPC emitted into the air with the potential to persist or accumulate in the environment, and those COPC released directly to water. As in the chronic inhalation assessment, the results of the multiple pathway assessment are presented separately for non-carcinogens and carcinogens in recognition of the different approaches used in calculating and interpreting risk estimates.

3.3.1 Non-Carcinogens

For the Aboriginal group, the RQ values are provided for the most sensitive life stage (Table 3-20). The most sensitive life stage is defined as the life stage with the greatest exposure per unit body weight per day. On this basis, young children were typically identified as the most sensitive on a per unit body weight basis. In the case of the workers (Table 3-21), it was assumed that the camps and other worksites would be occupied by adult employees only. As such, the RQ values for the workers are specific to the adult life stage only.

With the exception of the aromatic C_9-C_{16} group, manganese and methyl mercury for the Aboriginal group, all RQ values for the COPC are less than 1.0. The Aboriginal group also has RQ values greater than 1.0 for kidney toxicants, liver toxicants, neurotoxicants, and reproductive and developmental toxicants. The potential implications of these exceedances are discussed below. No exceedances are predicted for the worker group, indicating that the health risks are low at these locations.

This demonstrates that, for most COPC, predicted exposures are less than their health-based exposure limits and are not predicted to result in health-related impacts; therefore, potential chronic health risks for these COPC are considered low and adverse health effects are not predicted.

Table 3-20 Chronic Multiple Pathway Risk Quotients – Aboriginal Group (Cabin and Community Locations)

Chemicals of Potential Concern		Risk Quotients[1,2]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Organics	Acenaphthenes / acenaphthylenes	7.5E-07	3.6E-05	3.8E-05
	Acetone	2.8E-05	4.7E-05	4.8E-05
	Acrolein	1.4E-02	2.3E-02	2.4E-02
	Aliphatic aldehyde group	5.3E-01	8.9E-01	9.0E-01
	Aliphatic C_9-C_{16} group	2.3E-01	2.3E-01	3.7E-01
	Aliphatic C_{17}-C_{34} group	7.1E-04	7.1E-04	1.4E-03
	Anthracene / phenanthrenes and substituted	1.7E-03	1.8E-03	1.8E-03
	Aromatic C_9-C_{16} group	**1.6E+00**	**1.6E+00**	**2.4E+00**
	Aromatic C_{17}-C_{34} group	7.1E-04	7.1E-04	1.4E-03
	Biphenyl	1.7E-06	1.7E-06	4.1E-06
	Fluorenes / fluoranthenes and substituted	2.1E-05	5.6E-04	5.9E-04
	Formaldehyde	7.2E-04	1.2E-03	1.2E-03
	Methyl ethyl ketone group	2.0E-04	2.6E-04	2.6E-04
	Naphthalene and substituted naphthalenes	1.2E-03	1.3E-03	1.3E-03
	Phenol	1.2E-03	1.5E-03	1.5E-03
	Pyrenes and substituted pyrenes	4.9E-03	7.0E-03	7.3E-03
Metals	Aluminum	6.0E-01	6.2E-01	6.1E-01
	Antimony	4.2E-01	4.3E-01	4.5E-01
	Arsenic	4.1E-01	4.2E-01	4.3E-01
	Barium	2.2E-01	2.4E-01	2.4E-01
	Beryllium	1.5E-02	1.5E-02	1.6E-02
	Boron	3.1E-01	3.1E-01	3.3E-01
	Cadmium	4.6E-01	5.3E-01	6.1E-01
	Chromium	3.3E-03	3.6E-03	3.5E-03
	Chromium VI	9.3E-02	1.1E-01	1.0E-01
	Cobalt	5.9E-01	5.9E-01	6.2E-01
	Copper	1.5E-01	1.5E-01	1.6E-01
	Manganese	**1.8E+00**	**1.8E+00**	**1.9E+00**
	Methyl mercury	**6.7E+00**	**6.7E+00**	**6.7E+00**
	Mercury	1.8E-01	1.9E-01	1.9E-01
	Molybdenum	1.1E-01	1.1E-01	2.7E-01
	Nickel	9.0E-02	9.1E-02	9.4E-02
	Selenium	3.8E-01	3.8E-01	3.9E-01
	Silver	4.1E-03	4.2E-03	5.2E-03
	Strontium	1.5E-01	1.5E-01	1.5E-01
	Uranium	5.4E-01	5.4E-01	5.6E-01
	Vanadium	6.3E-01	6.5E-01	7.3E-01
	Zinc	8.3E-01	8.3E-01	8.3E-01

Table 3-20 Chronic Multiple Pathway Risk Quotients – Aboriginal Group (Cabin and Community Locations) (continued)

Chemicals of Potential Concern		Risk Quotients[1,2]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Mixtures[3]	Cardiovascular toxicants	8.0E-01	8.3E-01	8.6E-01
	Gastrointestinal toxicants	6.4E-01	1.0E+00	1.0E+00
	Renal toxicants	**4.6E+00**	**4.7E+00**	**6.0E+00**
	Hepatotoxicants	**3.4E+00**	**3.4E+00**	**4.4E+00**
	Neurotoxicants	**1.1E+01**	**1.1E+01**	**1.2E+01**
	Reproductive / developmental toxicants	**8.5E+00**	**8.5E+00**	**8.7E+00**

Notes:

[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

[2] Chronic inhalation RQ values were added to the RQ values associated with the secondary pathways of exposure for the aromatic C_9-C_{16} group, methyl ethyl ketone group, aluminum, barium, cadmium, manganese, selenium and silver because the chronic inhalation and chronic oral exposure limits for these COPC were based on the same toxicological endpoint.

[3] Note that addition of the individual constituents' RQ values might not equate to the RQ provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each receptor group, regardless of the location at which exposure occurred.

Table 3-21 Chronic Multiple Pathway Risk Quotients – Worker Group

Chemicals of Potential Concern[1]		Risk Quotients[2,3]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Organics	Acenaphthenes / acenaphthylenes	2.5E-11	2.5E-11	6.1E-11
	Acetone	1.5E-10	1.5E-10	2.4E-10
	Acrolein	3.2E-09	3.2E-09	5.0E-09
	Aliphatic aldehyde group	6.3E-08	6.3E-08	9.9E-08
	Aliphatic C_9-C_{16} group	1.7E-07	1.7E-07	1.8E-07
	Aliphatic C_{17}-C_{34} group	5.0E-09	1.3E-08	1.4E-08
	Anthracene / phenanthrenes and substituted	3.4E-06	3.4E-06	3.4E-06
	Aromatic C_9-C_{16} group	1.4E-01	1.4E-01	2.0E-01
	Aromatic C_{17}-C_{34} group	8.4E-10	8.5E-10	2.1E-09
	Biphenyl	1.0E-12	1.0E-12	1.9E-12
	Fluorenes / fluoranthenes and substituted	8.7E-09	8.8E-09	2.1E-08
	Formaldehyde	2.3E-12	2.3E-12	3.7E-12
	Methyl ethyl ketone group	1.2E-04	1.2E-04	1.8E-04
	Naphthalene and substituted naphthalenes	2.7E-06	2.7E-06	2.8E-06
	Phenol	4.6E-13	4.6E-13	4.9E-13
	Pyrenes and substituted pyrenes	2.2E-06	2.2E-06	2.9E-06
Metals	Aluminum	1.7E-01	1.7E-01	1.7E-01
	Antimony	7.5E-03	7.6E-03	7.6E-03
	Arsenic	2.4E-02	2.4E-02	2.4E-02
	Barium	3.0E-03	3.1E-03	3.1E-03
	Beryllium	6.3E-04	6.4E-04	6.4E-04
	Boron	1.0E-04	1.0E-04	1.0E-04
	Cadmium	1.8E-01	1.8E-01	2.2E-01
	Chromium	1.2E-05	1.2E-05	1.2E-05
	Chromium VI	1.5E-03	1.5E-03	1.5E-03
	Cobalt	1.4E-02	1.4E-02	1.4E-02
	Copper	2.5E-04	2.5E-04	2.5E-04
	Manganese	6.3E-02	6.3E-02	6.3E-02
	Mercury	3.0E-03	3.4E-03	3.5E-03
	Molybdenum	2.3E-04	2.4E-04	2.4E-04
	Nickel	1.0E-03	1.1E-03	1.1E-03
	Selenium	2.5E-04	2.6E-04	2.6E-04
	Silver	7.3E-04	8.4E-04	9.9E-04
	Strontium	2.9E-04	2.9E-04	2.9E-04
	Uranium	5.1E-03	5.1E-03	5.1E-03
	Vanadium	2.3E-02	2.3E-02	2.3E-02
	Zinc	3.8E-04	3.9E-04	3.9E-04

Table 3-21 Chronic Multiple Pathway Risk Quotients – Worker Group (continued)

Chemicals of Potential Concern[1]		Risk Quotients[2,3]		
		2013 Base Case	2013 PRM Application Case	2013 PDC
Mixtures[4]	Cardiovascular toxicants	2.1E-03	2.1E-03	2.1E-03
	Gastrointestinal toxicants	1.7E-02	1.7E-02	1.7E-02
	Renal toxicants	3.2E-01	3.2E-01	4.8E-01
	Hepatotoxicants	1.4E-01	1.4E-01	2.0E-01
	Neurotoxicants	6.7E-01	6.7E-01	7.4E-01
	Reproductive / developmental toxicants	7.4E-03	7.4E-03	1.2E-02

Notes:

[1] Values are not presented for methyl mercury as workers were assumed to obtain all food and water from the camps, which would in turn obtain all food and water from off-site, commercial sources.

[2] Chronic inhalation RQ values were added to the RQ values associated with the secondary pathways of exposure for the aromatic C_9-C_{16} group, methyl ethyl ketone group, aluminum, barium, cadmium, manganese, selenium and silver because the chronic inhalation and chronic oral exposure limits for these COPC were based on the same toxicological endpoint.

[3] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

[4] For individual constituents of the chemical mixtures, see Table 2-11. Note that addition of the individual constituents' RQ values might not equate to the RQ provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each receptor group, regardless of the location at which exposure occurred.

3.3.1.1 Aromatic C_9-C_{16} Group

The chronic RQ values for the aromatic C_9-C_{16} group are predicted to exceed 1.0 for the Aboriginal group only. As shown in Table 3-20, RQ values of 1.6 are predicted for the Aboriginal group under the 2013 Base and 2013 PRM Application cases, and 2.4 under the 2013 PDC. Given that the RQ values are not predicted to change between the 2013 Base Case and the 2013 PRM Application Case, PRM is predicted to have negligible influence on the risks estimated for the existing and approved activities under the 2013 Base Case.

In the case of the aromatic C_9-C_{16} group, the chronic inhalation and chronic oral exposure limits are based on the same toxicological endpoints – kidney and liver effects. To ensure that the risks associated with long-term exposure to the aromatic C_9-C_{16} group were not understated, the inhalation RQ values were added to the RQ values for the secondary pathways of exposure. The inhalation RQ values contribute approximately 100% to the overall risks. For the Aboriginal group, the maximum RQ values associated with the secondary pathways of exposure alone (i.e., inhalation of dust; ingestion of soil, water, local country and natural foods, local fish, and local game; and dermal contact with soil, and with water while swimming) are predicted to be 0.000097, 0.00011 and 0.00016 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively.

As such, interpretation of the multiple pathway health risks for the aromatic C_9-C_{16} group reflects the discussion of the inhalation health risks presented in Section 3.2.1.2. For the reasons stated, the weight of evidence indicates a low potential for adverse health effects as a result of long-term exposure to the aromatic C_9-C_{16} group.

3.3.1.2 Manganese

Manganese RQ values greater than 1.0 are only predicted for the Aboriginal group. The manganese RQ values for the different life stages are presented in Table 3-22.

Table 3-22 Chronic Multiple Pathway Risk Quotients for Manganese – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	4.1E-01	4.0E-01	5.0E-01
Toddler (7 months to 4 years)	**1.8E+00**	**1.8E+00**	**1.9E+00**
Child (5 to 11 years)	**1.3E+00**	**1.3E+00**	**1.3E+00**
Adolescent (12 to 19 years)	9.7E-01	9.6E-01	1.0E+00
Adult (20 to 80 years)	9.4E-01	9.3E-01	1.0E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

Given that the RQ values are not predicted to increase between the 2013 Base Case and the 2013 PRM Application Case, PRM is predicted to have negligible influence on the risks estimated for the existing and approved activities under the 2013 Base Case. Similarly, minimal changes are predicted in the RQ

values as a result of planned future developments. This indicates that the risks predicted under the 2013 PRM Application Case and 2013 PDC are attributable to the existing conditions captured under the 2013 Base Case, and the PRM and other planned future developments are not predicted to appreciably increase the risk of adverse health effects associated with long-term exposure to manganese in the region.

Manganese is commonly present in the environment and is an essential element involved in the formation of bone and in various aspects of metabolism (IOM 2001). Dietary sources are the primary route of human exposure to manganese, with people who consume a high amount of plant-based foods and legumes having potentially higher intake than other individuals (ATSDR 2008; IOM 2001; US EPA 1996).

In the current assessment, the primary exposure pathways contributing to the RQ values above 1.0 for the Aboriginal toddler and child are presented in Table 3-23. The contribution of these pathways is similar across the 2013 Base Case, 2013 PRM Application Case and 2013 PDC for these life stages.

Table 3-23 Break-Down of Exposure Pathways Contributing to the Predicted Manganese Risks

Primary Contributing Exposure Pathways	Percentage Break-Down	
	Toddler	Child
Above-ground plant consumption	57%	57%
Below-ground plant (root vegetables) consumption	18%	19%
Berry consumption	8%	12%
Labrador tea consumption	5%	3%
Cattail root consumption	1%	1%
Drinking water consumption	8%	7%
Fish consumption	<1%	<1%
Wild game consumption	1%	1%
Incidental soil ingestion	2%	<1%

As shown in Table 3-23, most of the manganese exposure (87% to 91%) relates to the ingestion of above-ground plants, below-ground plants and berries. These observations are consistent with those of the ATSDR, the Institute of Medicine (IOM) and the US EPA in that those individuals who consume larger amounts of plant-based foods are exposed to higher levels of manganese.

In the 2013 HHRA, the predicted intake levels for manganese are above the estimated daily dietary intakes of manganese for typical Canadian toddlers, children and adults (Health Canada 2011). This is due, in part, to the inclusion of drinking water and soil exposure pathways in the multiple pathway assessment of the 2013 HHRA (Table 3-24).

Table 3-24 Comparison of 2013 HHRA and Typical Canadian Daily Intakes of Manganese

Population	Intake Basis	Average Daily Intake (mg/day)[1]		
		Toddler	Child	Adult
Typical Canadian population (Health Canada 2011)	Dietary items alone	1.5 (1.0 to 2.1)	2.9 (2.6 to 3.5)	3.9 (2.4 to 5.2)
2013 HHRA Aboriginal Group	Dietary items alone	3.2	4.8	7.2
	Total intake	3.6	5.3	8.1

Notes:
[1] Values present in parentheses represent the range of estimated daily dietary intakes of manganese for typical Canadian toddlers (7 months to 4 years), child (5 to 11 years), and adults (20 to 65+ years) (Health Canada 2011).

The RQ values for manganese are based, in part, on the chronic oral exposure limit of 140 µg/kg bw/day recommended by the US EPA (1996). This exposure limit is based on a NOAEL of 10 mg/day (or 0.14 mg/kg bw/day for a 70 kg adult) derived from several population-based studies, each of which evaluated the relationship between manganese exposure and central nervous system effects in humans. The same NOAEL was identified by Health Canada (2010b) and the WHO (2004) in their respective reviews of the toxicological effects associated with long-term exposure to manganese. In the current assessment, the estimated daily intake of manganese for the toddler, child and adult in the 2013 PDC are predicted to be 3.6 mg/day, 5.3 mg/day and 8.1 mg/day, respectively. These intakes remain well below the recognized NOAEL of 10 mg/day (Health Canada 2010b; US EPA 1996; WHO 2004).

Although the weight of evidence suggests that exposure below 10 mg/day is unlikely to be associated with adverse effects (IOM 2001; Santamaria and Sulsky 2010; Andersen et al. 2010), the manganese exposure levels at which adverse effects are expected in humans has not been clearly defined to date. The WHO (2004) noted in its toxicological review that manganese is not considered very toxic to humans given the existence of homeostatic mechanisms, and that the incidence of adverse health effects at the upper range of dietary intake is negligible. Health Canada estimates the average daily intake of manganese for all Canadians (7 months to 65+ years) to be between 1 and 5 mg/day based on Canadian food consumption data in combination with the manganese content of the various food items (Health Canada 2011). As such, it would seem that the predicted daily intake of manganese for the toddlers of 3.6 mg/day (associated with the maximum RQ values of 1.9) is consistent with the average rates of exposure for the Canadian population.

3.3.1.3 Methyl Mercury

Risk quotients greater than 1.0 for methyl mercury are only predicted for the Aboriginal group. The methyl mercury RQ values for the different life stages are presented in Table 3-25.

Table 3-25 Chronic Multiple Pathway Risk Quotients for Methyl Mercury – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	1.9E-05	4.0E-05	4.4E-05
Toddler (7 months to 4 years)	**6.7E+00**	**6.7E+00**	**6.7E+00**
Child (5 to 11 years)	**5.5E+00**	**5.5E+00**	**5.6E+00**
Adolescent (12 to 19 years)	**3.7E+00**	**3.7E+00**	**3.7E+00**
Adult (20 to 80 years)	**3.1E+00**	**3.1E+00**	**3.1E+00**

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

The maximum predicted RQ for methyl mercury is 6.7, with the risks remaining unchanged for the three assessment cases. Given that the RQ values above 1.0 are not predicted to change between the 2013 Base Case and the 2013 PRM Application Case, PRM is predicted to have negligible influence on the risks estimated for the existing and approved activities under the 2013 Base Case. Similarly, planned future developments are not predicted to appreciably increase the risk of adverse health effects associated with long-term exposure to methyl mercury in the region. This indicates that the risks predicted under the 2013 PRM Application Case and 2013 PDC are attributable to the existing conditions captured under the 2013 Base Case.

Methyl mercury risks were described in EIA, Volume 3, Section 5.3.3.3, pages 5-132 to 5-135. The maximum methyl mercury RQ predicted for the Aboriginal group in the 2013 HHRA (6.7) is comparable to the maximum RQ presented in the EIA HHRA (8.3). The variance between the risks is due to a combination of reduced fish consumption rates in the 2013 HHRA and changes to the predicted water quality.

Methyl mercury is the form of mercury that is of greatest concern with respect to accumulation in biological organisms, and subsequent consumption by people (Health Canada 2007). Food intake is the primary route of exposure to mercury compounds in humans, with fish and seafood being the most significant contributors to human exposure (ATSDR 1999). Mercury can cycle between its various forms and does not necessarily permanently exist as one form or another. Microbial activity in the environment can convert inorganic mercury to methyl mercury and vice versa (Health Canada 2007). In the 2013 HHRA, it was assumed that methyl mercury concentrations were equivalent to 100% of total mercury concentrations in fish.

For the Aboriginal group, the maximum RQ value was predicted for the toddler life stage, where 100% of the estimated daily intake of methyl mercury is attributable to local fish consumption. The methyl mercury concentration (i.e., 90th percentile) in fish used in the 2013 HHRA is 0.55 mg/kg wet weight in the 2013 Base Case, 2013 PRM Application Case and 2013 PDC. This concentration is above the subsistence fish consumption guideline of 0.2 mg/kg recommended by Health Canada (2007).

In 2009, Alberta Health and Wellness conducted a health risk assessment of mercury in fish collected as part of regional aquatics monitoring program (RAMP) in the oil sands (AHW 2009b). As part of its assessment, Alberta Health and Wellness investigated the concentrations of mercury in various fish species collected from the waterbodies of the RAMP area and, in turn, characterized the potential health risks associated with these concentrations. Alberta Health and Wellness discussed the overall benefits of fish consumption and the existing advisory to restrict or limit the consumption of walleye, northern pike and whitefish from certain lakes and rivers in the area (AHW 2009b). The results of the study indicated that concentrations of mercury in fish in the waterbodies of the RAMP area were within the ranges for the same fish species from other waterbodies in Alberta, Canada and the United States. As such, the health risks posed to people eating fish from the oil sands region do not appear to be higher than those posed to individuals who eat fish from other parts of the country.

Table 3-26 presents a summary of measured total mercury concentrations from various locations in Alberta (AHW 2009c,d), as well as information regarding mercury concentrations in fish sold by retailers in Canada (Health Canada 2007). As shown in Table 3-26, the methyl mercury concentration of 0.55 mg/kg estimated in the 2013 HHRA falls within the range of values measured in fish collected from these other locations, including local supermarkets. These comparisons suggest that the fish mercury concentrations used in the 2013 HHRA do not necessarily indicate an unusual risk level. The conclusions regarding the methyl mercury risks for the 2013 HHRA are consistent with those described in the EIA HHRA.

Table 3-26 Mercury Concentrations in Alberta Fish

Location	Total Mercury Concentration (mg/kg wet weight)
Twin Valley Reservoir, Alberta	0.22 to 0.68
Little Bow River, Alberta	0.1 to 0.59
Willow Creek, Alberta	0.08 to 0.49
Pine Coulee Reservoir, Alberta	0.13 to 0.79
Lake La Nonne, Alberta	0.56 to 0.63
Lake Ste Anne, Alberta	0.13 to 0.14
Canadian Retail Fish	0.02 to 1.82
2013 HHRA	0.55[(1)]

Notes:
[(1)] Total mercury assumed to be 100% methyl mercury (AHW 2009c,d; Health Canada 2007).

At present, there is a consumption advisory on walleye caught from the Athabasca River, downstream of Fort McMurray (Government of Alberta 2012). The recommended restrictions with respect to daily or weekly consumption patterns are based solely on public health considerations relating to exposure to individual contaminants, with limited consideration given to the counterbalancing benefits of fish consumption or alternative risks of other protein sources that may be consumed in place of fish (AHW 2009b). The fish consumption advisory is related to methyl mercury concentrations and should be considered relevant to the 2013 HHRA. The Athabasca River advisory suggests that in the PRM area, women of child-bearing age, toddlers, children and adults should restrict their consumption of walleye that weigh more than 2 lbs (Government of Alberta 2012). According to Alberta's sportfishing regulations, a toddler (1 to 4 years) should not eat more than 5 grams of fish per day, a child (5 to 11

years) should not eat more than 10 grams of fish per day, pregnant women or women of child-bearing age should not eat more than 21 grams of fish per day, and other adults should restrict their consumption of walleye to no more than 86 grams per day (Government of Alberta 2012). These ingestion rates for fish consumed from the Athabasca River are considerably less than those used in the 2013 HHRA for the toddler, child and pregnant woman or woman of child-bearing age of the Aboriginal group (20 g/day for toddlers, 33 g/day for children, and 40 g/day for adolescents and adults). If the potentially vulnerable groups adhere to the fish consumption advisory, wherein toddlers eat less than 5 grams of fish per day, children eat less than 10 grams of fish per day and pregnant women eat less than 21 grams per day, the health risks associated with methyl mercury would be lower than those described for the Aboriginal group in the 2013 HHRA.

3.3.1.4 Renal Toxicants

For renal toxicants, RQ values greater than 1.0 are only predicted for the Aboriginal group. The renal toxicant RQ values for the different life stages are presented in Table 3-27.

Table 3-27 Chronic Multiple Pathway Risk Quotients for Renal Toxicants – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	2.7E+00	2.7E+00	3.8E+00
Toddler (7 months to 4 years)	4.6E+00	4.7E+00	6.0E+00
Child (5 to 11 years)	3.8E+00	3.9E+00	5.1E+00
Adolescent (12 to 19 years)	3.5E+00	3.5E+00	4.7E+00
Adult (20 to 80 years)	3.6E+00	3.7E+00	4.9E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

The maximum predicted RQ values for the renal toxicants are 4.6, 4.7 and 6.0 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively. The differences between the RQ values for the renal toxicants under the 2013 Base Case and 2013 PRM Application Case are minor, indicating that the PRM will not materially increase the overall kidney toxicant risks in the region.

For the renal toxicants, the maximum inhalation RQ values for the mixture were added to the maximum mixture RQ values associated with the secondary pathways of exposure. As shown in Table 3-28, the inhalation RQ values contribute between 46% and 81% to the overall risks. The degree of conservatism incorporated in the chronic inhalation RQ values for the renal toxicants mixture has been previously discussed (see Section 3.2.1.7). As such, interpretation of the multiple pathway health risks for the renal toxicants mixture focuses on the discussion of the health risks associated with the secondary pathways of exposure.

Table 3-28 Chronic Inhalation and Secondary Pathway Risk Quotients for Renal Toxicants – Aboriginal Group

Life Stages[1]	Inhalation Risk Quotients[2,3,4]			Secondary Pathway Risk Quotients[2,4]		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	**2.1E+00**	**2.1E+00**	**3.1E+00**	5.9E-01	6.3E-01	7.2E-01
Toddler (7 months to 4 years)				**2.5E+00**	**2.6E+00**	**2.9E+00**
Child (5 to 11 years)				**1.7E+00**	**1.8E+00**	**2.0E+00**
Adolescent (12 to 19 years)				**1.4E+00**	**1.4E+00**	**1.6E+00**
Adult (20 to 80 years)				**1.5E+00**	**1.6E+00**	**1.8E+00**

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Inhalation RQ values are not life stage specific.
[4] Values in **bold** indicate an RQ greater than 1.0.

The renal toxicants mixture consists of 14 compounds or groups of compounds: acetone, aliphatic C_9-C_{16} group, aluminum, aromatic C_9-C_{16} group, aromatic C_{17}-C_{34} group, barium, biphenyls, cadmium, fluorenes/fluoranthenes, formaldehyde, mercury, pyrenes and substituted pyrenes, and uranium. The primary contributors to the risks are aluminum (23%), antimony (16%), cadmium (14%) and uranium (21%). When evaluated on their own, none of these compounds or groups presents exceedances in the multiple pathway assessment.

For aluminum, the primary exposure pathways for the toddler in the 2013 PRM Application Case are moose ingestion (28%), cattail ingestion (18%), soil ingestion (18%), drinking water (12%) and above-ground plant ingestion (9%). The maximum predicted EDI for aluminum is 88 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (143 μg/kg bw/day) for aluminum represents the most recent Tolerable Daily Intake (TDI) derived by the World Health Organization, based on several animal and epidemiological studies. In these studies, a range of LOAELs for reproductive, neurological effects, liver and kidney effects of 50 to 75 mg/kg bw/day were identified. The TDI is based on the lowest LOAEL within this range (50 mg/kg bw/day). The maximum predicted EDI for the toddler (88 μg/kg bw/day) is well below both the exposure limit and the LOAEL.

The actual health risks for aluminum are likely overstated, based on the following:

- The degree of conservatism incorporated into the exposure limit; and
- The potential for absorption of aluminum in the gastrointestinal tract of humans. The absorption of aluminum is variable between the types of aluminum, with some forms having been observed to have less than 1% absorption (WHO and FAO 2007). In the 2013 HHRA, 100% absorption was assumed.

For antimony, the primary exposure pathways for the toddler in the 2013 PRM Application Case are moose ingestion (33%), cattail ingestion (13%), drinking water (30%), fish ingestion (9%), and above-ground plant ingestion (9%). The maximum predicted EDI for antimony is 0.091 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (0.2 μg/kg bw/day) for antimony represents the tolerable daily intake (TDI) derived by Health Canada in association with the Canadian Drinking Water Guideline (Health Canada 1999). The basis of this TDI is a NOAEL of 0.5 mg/L for hematuria and cirrhosis of the liver, and increased relative kidney weights. This NOAEL was converted to an average antimony intake of 0.06 mg/kg bw/day. The maximum predicted EDI for the toddler (0.091 μg/kg bw/day) is well below both the exposure limit and the NOAEL. As a result, the actual health risks for antimony are likely overstated.

For cadmium, the primary exposure pathways for the toddler in the 2013 PRM Application Case are moose ingestion (40%), snowshoe hare ingestion (20%), fish ingestion (20%), above-ground plant ingestion (8%), ruffed grouse ingestion (4%), and cattail ingestion (5%). The maximum predicted EDIs for the toddler in the Aboriginal group based on food and water consumption are 0.0058 μg/kg bw/day and 0.43 μg/kg bw/day, respectively.

The exposure limits used in the 2013 HHRA for food consumption (1 μg/kg bw/day) and water consumptions (0.5 μg/kg bw/day) for cadmium were derived by the US EPA (1994) based on human studies involving chronic exposure via food intake. The basis of these limits is a NOAEL of 0.01 mg/kg bw/day for significant proteinuria. A NOAEL of 0.005 mg/kg bw/day was calculated by the US EPA for exposures via drinking water. The maximum predicted EDIs for the toddler are well below both the exposure limits and the NOAELs. As a result, the actual health risks for cadmium are likely overstated.

For uranium, the primary exposure pathways for the toddler in the 2013 PRM Application Case are moose ingestion (63%), fish ingestion (27%) and drinking water (3%). The maximum predicted EDI for uranium is 0.33 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (0.6 μg/kg bw/day) for uranium represents the TDI derived by Health Canada in association with the Canadian Drinking Water Guideline (Health Canada 2001). The basis of this TDI is a LOAEL of 0.96 mg/L uranyl nitrate hexahydrate, which is equivalent to 0.09 mg/kg bw/day (females) and 0.06 mg/kg bw/day (males). The maximum predicted EDI for the toddler (0.33 μg/kg bw/day) is well below both the exposure limit and the LOAEL. As a result, the actual health risks for uranium are likely overstated.

While these COPC shared the same general toxicological endpoint of renal effects, closer examination of the specific effects and mechanisms is necessary to evaluate the potential for additivity. The adverse renal effects cited by WHO and FAO (2007) in support of the aluminum exposure limit include mild histopathological changes in the kidney. The toxicological basis of the antimony exposure limit is increase in relative kidney weights. For cadmium, the basis of the exposure limit includes cadmium

accumulation in the renal cortex and proteinuria. The basis of the uranium exposure limit is renal lesions, including nuclear vesiculation, cytoplasmic vacuolation, tubular dilation, glomerular adhesions, abnormalities in proximal tubule epithelia, and cytoplasmic degranulation. As these endpoints are different, it is reasonable to question the actual additivity of these endpoints.

Similar conservatism is incorporated in the prediction of the chronic inhalation RQ values for each of the individual renal toxicants. As such, summation of the RQ values for the five constituents of the inhalation renal toxicants mixture and the RQ values for the 14 constituents of the multiple pathway renal toxicants mixture likely compounds the conservatism incorporated in each of the individual assessments and overstates the actual combined risks.

3.3.1.5 Hepatotoxicants

Risk quotients greater than 1.0 are predicted for hepatotoxicants for the Aboriginal group only. The hepatotoxicant RQ values for the different life stages are presented in Table 3-29.

Table 3-29 Chronic Multiple Pathway Risk Quotients for Hepatotoxicants – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	2.0E+00	2.0E+00	2.9E+00
Toddler (7 months to 4 years)	3.4E+00	3.4E+00	4.4E+00
Child (5 to 11 years)	2.8E+00	2.8E+00	3.8E+00
Adolescent (12 to 19 years)	2.6E+00	2.6E+00	3.5E+00
Adult (20 to 80 years)	2.7E+00	2.7E+00	3.6E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

The maximum predicted RQ values for the hepatotoxicants are 3.4 for the 2013 Base and 2013 PRM Application cases, and 4.4 for the 2013 PDC. There is no change in the RQ values for the hepatotoxicants under the 2013 Base Case and 2013 PRM Application Case, indicating that the PRM will not materially increase the overall risks of adverse liver effects for residents or workers in the area.

Like the renal toxicants, the maximum inhalation RQ values for the hepatotoxicants mixture were added to the maximum mixture RQ values associated with the secondary pathways of exposure. As shown in Table 3-30, the inhalation RQ values contribute between 47% and 83% to the overall risks. The degree of conservatism incorporated in the chronic inhalation RQ values for the hepatotoxicants mixture has been previously discussed (see Section 3.2.1.8). As such, interpretation of the multiple pathway health risks for the hepatotoxicants focuses on the discussion of the health risks associated with the secondary pathways of exposure.

Table 3-30 Chronic Inhalation and Secondary Pathway Risk Quotients for Hepatotoxicants – Aboriginal Group

Life Stages[1]	Inhalation Risk Quotients[2,3,4]			Secondary Pathway Risk Quotients[2,4]		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	**1.6E+00**	**1.6E+00**	**2.4E+00**	4.0E-01	4.1E-01	4.9E-01
Toddler (7 months to 4 years)				**1.8E+00**	**1.8E+00**	**2.0E+00**
Child (5 to 11 years)				**1.2E+00**	**1.2E+00**	**1.4E+00**
Adolescent (12 to 19 years)				9.7E-01	9.8E-01	**1.1E+00**
Adult (20 to 80 years)				**1.1E+00**	**1.1E+00**	**1.2E+00**

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Inhalation RQ values are not life stage specific.
[4] Values in **bold** indicate an RQ greater than 1.0.

The hepatotoxicants mixture consists of nine compounds or groups of compounds: acenaphthenes / acenaphthylenes, aliphatic C_9-C_{16} group, aliphatic C_{17}-C_{34} group, aluminum, antimony, aromatic C_9-C_{16} group, copper, fluorenes/fluoranthenes and selenium. The primary contributors to the risks are the aliphatic C_9-C_{16} group (13%), aluminum (34%), antimony (24%), and selenium (21%). None of these compounds or groups presents exceedances on their own.

For the aliphatic C_9-C_{16} group, the primary pathway of exposure for the toddler in the 2013 PRM Application Case is moose ingestion (100%). The maximum predicted estimated daily intake (EDI) for the aliphatic C_9-C_{16} group is 38 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (100 μg/kg bw/day) for the aliphatic C_9-C_{16} group is based on two animal studies (TPHCWG 1997). From these studies, a dietary NOAEL of 100 mg/kg bw/day for adverse histopathological effects on the liver was identified. The TPHCWG applied an uncertainty factor of 1,000 to the NOAEL to account for differences in sensitivity between rats and humans, differences in sensitivity within the human population, and subchronic exposure duration. The maximum predicted EDI for the toddler (38 μg/kg bw/day) is well below both the exposure limit and the NOAEL.

The actual health risks for the aliphatic C_9-C_{16} group are likely overstated, for the following reasons:

- The assumptions regarding local moose consumption applied in the 2013 HHRA, where it was assumed that people would be regularly consuming moose at the assumed rates; and
- The degree of conservatism incorporated into the exposure limit for the aliphatic C_9-C_{16} group.

For aluminum, the primary pathways exposure for the toddler in the 2013 PRM Application Case are soil ingestion (18%), moose ingestion (28%), cattail ingestion (18%), drinking water (12%) and above-ground plant ingestion (9%). The predicted estimated daily intake (EDI) for aluminum is 88 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (143 μg/kg bw/day) for aluminum represents the most recent TDI derived by the WHO, based on several animal and epidemiological studies. In these studies, a range of LOAELs for reproductive, neurological effects, liver and kidney effects of 50 to 75 mg/kg bw/day were identified. The TDI is based on the lowest LOAEL within this range (50 mg/kg bw/day). The EDI for the toddler (88 μg/kg bw/day) is well below both the exposure limit and the LOAEL.

The actual health risks for aluminum are likely overstated, based on the following:

- The degree of conservatism incorporated into the exposure limit; and
- The potential for absorption of aluminum in the gastrointestinal tract of humans. The absorption of aluminum is variable between the types of aluminum, with some forms having been observed to have less than 1% absorption (WHO and FAO 2007). In the 2013 HHRA, 100% absorption was assumed.

For antimony, the primary exposure pathways for the toddler in the 2013 PRM Application Case are moose ingestion (33%), cattail ingestion (13%), drinking water (30%) fish ingestion (9%), and above-ground plant ingestion (9%). The maximum predicted EDI for antimony is 0.091 μg/kg bw/day for the toddler in the Aboriginal group.

The exposure limit used in the 2013 HHRA (0.2 μg/kg bw/day) for antimony represents the TDI derived by Health Canada in association with the Canadian Drinking Water Guideline (Health Canada 1999). The basis of this TDI is a NOAEL of 0.5 mg/L for hematuria and cirrhosis of the liver, and increased relative kidney weights. The maximum predicted EDI for the toddler (0.091 μg/kg bw/day) is well below both the exposure limit and the NOAEL. As a result, the actual health risks for the antimony are likely overstated.

For selenium, the primary exposure pathways for the toddler life stage in the 2013 PRM Application Case are fish ingestion (58%) and snowshoe hare ingestion (35%). The predicted EDI for selenium is 1.9 μg/kg bw/day for the Aboriginal toddler. The exposure limit of 5 μg/kg bw/day is based on a NOAEL of 0.015 mg/kg/day observed in a large epidemiological study that included low, medium and high exposure areas in China. The predicted selenium EDI for the toddler is well below the NOAEL of 0.015 mg/kg body weight per day (15 μg/kg bw/day).

The actual health risks for selenium are likely overstated, for the following reasons:

- The assumptions regarding local fish and snowshoe hare consumption applied in the 2013 HHRA, where it was assumed that people would be regularly consuming these local foods at the assumed rates; and
- The degree of conservatism incorporated into the exposure limit for selenium.

While the aliphatic C_9-C_{16} group, aluminum, antimony and selenium are cited as having hepatic endpoints, the nature of these endpoints must be considered to determine whether or not the mechanisms and effects are additive. The hepatotoxicant effects of aliphatic C_9-C_{16} group and aluminum include mild histopathological changes in the liver in various species, over acute and subchronic study durations. For the selenium exposure limit, the hepatic effects associated with clinical selenosis (the

basis of the US EPA reference dose) were cited as including subclinical blood substances symptomatic of liver dysfunction, specifically the prolongation of clotting time and serum glutathione titres. However, the key study on which the US EPA reference dose is based (Yang et al. 1989) notes that in the high selenium intake areas studied, liver damage or disease has not been reported. Histological and subclinical endpoints were considered for antimony as well. The antimony exposure limit included mild histopathological changes and clinical chemical changes related to the liver as its basis. These effects are generally subclinical, meaning that they may not be related to actual, measurable adverse health effects. While there is some potential that these subclinical effects might be additive, because of the variety of endpoints, it is also possible that the effects are less than additive.

Based on the above information, the weight of evidence suggests that there is low potential for adverse health effects associated with long-term exposure to the liver toxicants in the region.

3.3.1.6 Neurotoxicants

Exceedances are only predicted for the Aboriginal group. The neurotoxicants RQ values for the different life stages are presented in Table 3-31. There is no difference between the maximum neurotoxicants RQ values predicted for the 2013 Base Case and 2013 PRM Application Case, indicating that the PRM will not materially increase the overall neurological risks in the region.

Table 3-31 Chronic Multiple Pathway Risk Quotients for Neurotoxicants – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	1.9E+00	1.9E+00	2.7E+00
Toddler (7 months to 4 years)	1.1E+01	1.1E+01	1.2E+01
Child (5 to 11 years)	8.8E+00	8.8E+00	9.6E+00
Adolescent (12 to 19 years)	6.5E+00	6.5E+00	7.3E+00
Adult (20 to 80 years)	5.9E+00	5.9E+00	6.7E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

Like the renal and hepatotoxicants before, the maximum inhalation RQ values for the neurotoxicants mixture were added to the maximum mixture RQ values associated with the secondary pathways of exposure. As shown in Table 3-32, the inhalation RQ values contribute between 12% and 78% to the overall risks. The degree of conservatism incorporated in the chronic inhalation RQ values for the neurotoxicants mixture has been previously discussed (see Section 3.2.1.9). As such, interpretation of the multiple pathway health risks for the neurotoxicants focuses on the discussion of the health risks associated with the secondary pathways of exposure.

Table 3-32 Chronic Inhalation and Secondary Pathway Risk Quotients for Neurotoxicants – Aboriginal Group

Life Stages[1]	Inhalation Risk Quotients[2,3,4]			Secondary Pathway Risk Quotients[2,4]		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	**1.3E+00[4]**	**1.3E+00**	**2.0E+00**	5.7E-01	5.6E-01	6.7E-01
Toddler (7 months to 4 years)				**9.5E+00**	**9.5E+00**	**9.6E+00**
Child (5 to 11 years)				**7.5E+00**	**7.5E+00**	**7.6E+00**
Adolescent (12 to 19 years)				**5.2E+00**	**5.2E+00**	**5.3E+00**
Adult (20 to 80 years)				**4.6E+00**	**4.6E+00**	**4.7E+00**

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Inhalation RQ values are not life stage specific.
[4] Values in **bold** indicate an RQ greater than 1.0.

The neurotoxicants mixture consists of aluminum, lead, manganese, methyl mercury and selenium. The primary contributors to the risks are methyl mercury (71%) and manganese (19%).

The methyl mercury risks are 100% attributable to local fish consumption, while the manganese risks are primarily from plant food intakes in the model. Together, these COPC account for about 91% of the mixture risk for the Aboriginal group. As discussed in Sections 3.3.1.2 and 3.3.1.3, the RQ values for both manganese and methyl mercury are likely overstated due to the conservative assumptions incorporated into the 2013 HHRA.

Manganese is an essential element involved in the formation of bone and in various aspects of metabolism (IOM 2001). The World Health Organization (WHO 2004) notes that manganese is not considered very toxic to humans given the existence of homeostatic mechanisms, and that the incidence of adverse health effects at the upper range of dietary intake is negligible.

The estimated manganese risks are based on a no-observable-adverse-effect-level (NOAEL) of 10 mg/day derived from several population-based studies, each of which evaluated the relationship between manganese exposure and central nervous system effects in humans. In the current assessment, the daily intakes of manganese for the toddler, child and adult in the 2013 PDC are predicted to be 3.6 mg/day, 5.3 mg/day and 8.1 mg/day, respectively. These intakes remain well below the recognized NOAEL of 10 mg/day (Health Canada 2010b; US EPA 1996; WHO 2004). As well, the predicted daily intake of manganese for the toddlers (associated with the maximum RQ value) is consistent with the average rates of exposure for the Canadian population.

With respect to methyl mercury, the maximum predicted RQ is 6.7, with the risks remaining unchanged for the three assessment cases. Given that the RQ values above 1.0 are not predicted to change between the 2013 Base Case and the 2013 PRM Application Case, PRM is predicted to have negligible influence on the risks estimated for the existing and approved activities under the 2013 Base Case. This

indicates that the risks predicted under the 2013 PRM Application Case are due to the existing conditions captured under the 2013 Base Case.

The results of a 2009 Alberta Health and Wellness study indicated that concentrations of mercury in fish in the waterbodies of the PRM area were within the ranges for the same fish species from other waterbodies in Alberta, Canada and the United States (AHW 2009b). As such, the health risks posed to people eating fish from the oil sands region do not appear to be higher than those posed to individuals who eat fish from other parts of the country.

Overall, the weight of evidence suggests that there is low potential for adverse health effects associated with long-term exposure to the neurotoxicants in the region.

3.3.1.7 Reproductive and Developmental Toxicants

Exceedances are only predicted for the Aboriginal group. The reproductive and developmental toxicants RQ values for the different life stages are presented in Table 3-33. The differences between the reproductive and developmental toxicants for the 2013 Base Case and 2013 PRM Application Case are negligible, indicating that the PRM will not materially increase the overall reproductive and developmental risks in the region.

Table 3-33 Chronic Multiple Pathway Risk Quotients for Reproductive and Developmental Toxicants – Aboriginal Group

Life Stages[1]	Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	2.7E-01	2.9E-01	3.2E-01
Toddler (7 months to 4 years)	8.5E+00	8.5E+00	8.7E+00
Child (5 to 11 years)	6.7E+00	6.8E+00	6.9E+00
Adolescent (12 to 19 years)	4.6E+00	4.7E+00	4.8E+00
Adult (20 to 80 years)	4.1E+00	4.2E+00	4.2E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

For the reproductive and developmental toxicants, the maximum inhalation RQ values for the mixture were added to the maximum mixture RQ values associated with the secondary pathways of exposure. As shown in Table 3-34, the contribution from the inhalation RQ values to the overall risks is negligible. As such, interpretation of the multiple pathway health risks for the reproductive and developmental toxicants focuses on the discussion of the health risks associated with the secondary pathways of exposure.

Table 3-34 Chronic Inhalation and Secondary Pathway Risk Quotients for Reproductive and Developmental Toxicants – Aboriginal Group

Life Stages[1]	Inhalation Risk Quotients[2,3]			Secondary Pathway Risk Quotients[2]		
	2013 Base Case	2013 PRM Application Case	2013 PDC	2013 Base Case	2013 PRM Application Case	2013 PDC
Infant (0 to 6 months)	1.2E-02	1.3E-02	1.5E-02	2.6E-01	2.7E-01	3.1E-01
Toddler (7 months to 4 years)				8.5E+00	8.5E+00	8.6E+00
Child (5 to 11 years)				6.7E+00	6.8E+00	6.9E+00
Adolescent (12 to 19 years)				4.6E+00	4.7E+00	4.7E+00
Adult (20 to 80 years)				4.1E+00	4.1E+00	4.2E+00

Notes:
[1] The five receptor life stages that were included in the 2013 HHRA are consistent with Health Canada guidance (Health Canada 2010a).
[2] With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[3] Inhalation RQ values are not life stage specific.

The reproductive and developmental toxicants mixture consists of acetone, aluminum, boron, methyl mercury, methyl ethyl ketone group, nickel, phenol, strontium and vanadium. Methyl mercury is the primary contributor to the risks (79%).

The methyl mercury risks for the Aboriginal group are likely overstated because of the conservative assumptions incorporated into the 2013 HHRA. All methyl mercury risks are attributable to the assumed consumption of local fish. As discussed in Sections 3.3.1.3, the RQ values for methyl mercury are likely overstated due to the conservative assumptions incorporated into the 2013 HHRA.

While the components of the mixture were grouped together on the basis that they may cause reproductive and development effects, consideration should be given to the various types of reproductive or developmental effects, and whether or not the risks are truly additive or not.

Aluminum and methyl mercury are potential neurotoxins, and are classified as reproductive and developmental toxicants on the basis that fetuses and young children are most susceptible. The other reproductive and developmental toxicants, while representing minor contributions on their own, together contribute over 20% to the mixture risks. Acetone was classified as a reproductive and developmental toxicant based on depressed sperm motility, caudal weight, epididymal weight, and increased incidence of abnormal sperm, as opposed to the neurological effect for methyl mercury. The RQ values for boron and the methyl ethyl ketone group are based on exposure limits for decreased fetal body weights. Phenol risks are based on decreases in food consumption and body weight in maternal rats and decreases in fetal body weights due to loss of bone tissue formation. The strontium risks are based on bone effects in fetuses, while the nickel RQ values are based on the incidence of post-implantation lethality. The endpoint of the vanadium exposure limit included reduced body weight, tail length and relative organ weight of the liver, spleen and kidneys in offspring. Thus, the assumption that the potential risks of reproductive and developmental toxicity are additive is likely overly conservative, given the variety of endpoints and mechanisms by which such effects may occur for the constituents of this mixture.

The PRM is predicted to have negligible impact on the potential health risks associated with the long-term exposure to the reproductive and developmental toxicants. Despite the predicted exceedances for the Aboriginal group, the weight of evidence suggests that there is low potential for adverse health effects associated with long-term exposure to the reproductive and developmental toxicants in the region.

3.3.2 Carcinogens

For the Aboriginal group (cabin and community locations), carcinogenic risks are estimated for people exposed via secondary exposure pathways over their entire lifespan of 80 years (Health Canada 2010a). In the case of the workers, it was assumed that secondary pathway exposures would be limited to the 60 years of their adult life (i.e., 20 to 80 years of age).

The multiple pathway incremental lifetime cancer risks are presented in Table 3-35 (Aboriginal group) and Table 3-36 (worker group). Cancer risks are presented for the PRM (i.e., 2013 PRM Application Case minus 2013 Base Case) and future emission sources (i.e., 2013 PDC minus 2013 Base Case).

As shown, the predicted incremental lifetime cancer risks associated with the PRM alone and the planned future emission sources are all less than 1 in 100,000, indicating that the incremental lifetime cancer risk from the PRM and future emission sources are deemed to be essentially negligible.

Table 3-35 Chronic Multiple Pathway Incremental Lifetime Cancer Risks – Aboriginal Group (Cabin and Community Locations)

Chemical of Potential Concern[1]		Incremental Lifetime Cancer Risks (per 100,000)[2]	
		Pierre River Mine	Future
Organics	Benzo(a)pyrene group	1.0E-02	8.8E-02
	Propylene oxide	1.0E-02	2.1E-02
Metals	Arsenic	1.1E-01	4.3E-01

Notes:
[1] Chronic inhalation ILCR values were added to the ILCR values associated with the secondary pathways of exposure for arsenic because the inhalation and oral exposure limits for arsenic are based on a similar toxicological endpoint.
[2] An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

Table 3-36 Chronic Multiple Pathway Incremental Lifetime Cancer Risks – Worker Group

Chemical of Potential Concern[1]		Incremental Lifetime Cancer Risks (per 100,000)[2]	
		Pierre River Mine	Future
Organics	Benzo(a)pyrene group	1.0E-02	8.8E-02
	Propylene oxide	1.0E-02	2.1E-02
Metals	Arsenic	1.1E-01	4.3E-01

Notes:
[1] Chronic inhalation ILCR values were added to the ILCR values associated with the secondary pathways of exposure for arsenic because the inhalation and oral exposure limits for arsenic are based on a similar toxicological endpoint.
[2] An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

3.4 Pit Lake Assessment

Public access to the PRM pit lakes will be managed until considered safe. Consideration was not given to the potential for direct human exposure to the water in the pit lakes given this risk management action. However, ecological exposure to water in the pit lakes was assumed for the prediction of game meat concentrations in the 2013 HHRA, as it is possible that wild game commonly consumed by the area residents would have access to the pit lakes over time.

For the non-carcinogenic COPC, health risks were predicted using all assumptions and predicted media concentrations for the 2013 HHRA of the 2013 PDC, with the exception of game meat concentrations that took into account pit lake water exposures. Incremental cancer risks attributable to the pit lakes were based on assumptions and predicted media concentrations for the 2013 PDC incremental case, with the exception of game meat concentrations, which were based on pit lake water quality. Risks are only presented for the Aboriginal group in the pit lake assessment, as this group represents the likeliest exposure scenario.

3.4.1 Non-Carcinogens

The predicted chronic multiple pathway RQ values for the pit lake scenario are presented in Table 3-37. Overall, the RQ values are essentially identical or similar to the predicted risks in the 2013 PDC. As such, interpretation of the pit lake results is indistinguishable from the discussion presented in Section 3.3 for the chronic multiple pathway assessment.

Table 3-37 Chronic Multiple Pathway Risk Quotients for the Pit Lake Scenario – Aboriginal Group (Cabin and Community Locations)

Chemical of Potential Concern		Risk Quotient[(1,2)]
		Pit Lake Scenario
Organics	Acenaphthenes / acenaphthylenes	2.1E-05
	Acetone	4.8E-05
	Acrolein	2.4E-02
	Aliphatic aldehyde group	9.0E-01
	Aliphatic C_9-C_{16} group	3.7E-01
	Aliphatic C_{17}-C_{34} group	1.4E-03
	Anthracene / phenanthrenes and substituted	1.8E-03
	Aromatic C_9-C_{16} group	1.6E-04
	Aromatic C_{17}-C_{34} group	3.3E-05
	Biphenyl	6.4E-06
	Fluorenes / fluoranthenes and substituted	2.7E-04
	Formaldehyde	1.2E-03
	Methyl ethyl ketone group	2.6E-04
	Naphthalene and substituted naphthalenes	1.3E-03
	Phenol	1.5E-03
	Pyrenes and substituted pyrenes	7.1E-03

Table 3-37 Chronic Multiple Pathway Risk Quotients for the Pit Lake Scenario – Aboriginal Group (Cabin and Community Locations) (continued)

Chemical of Potential Concern		Risk Quotient[1,2]
		Pit Lake Scenario
Metals	Aluminum	7.7E-01
	Antimony	4.6E-01
	Arsenic	4.3E-01
	Barium	2.4E-01
	Beryllium	1.6E-02
	Boron	3.3E-01
	Cadmium	7.0E-01
	Chromium	4.3E-03
	Chromium VI	1.0E-01
	Cobalt	6.2E-01
	Copper	1.6E-01
	Manganese	**1.9E+00**
	Methyl mercury	**6.7E+00**
	Mercury	2.2E-01
	Molybdenum	2.8E-01
	Nickel	9.4E-02
	Selenium	3.9E-01
	Silver	7.4E-03
	Strontium	1.5E-01
	Uranium	5.6E-01
	Vanadium	9.4E-01
	Zinc	8.7E-01
Mixtures[3]	Cardiovascular toxicants	8.6E-01
	Gastrointestinal toxicants	**1.0E+00**
	Renal toxicants	**6.3E+00**
	Hepatotoxicants	**4.6E+00**
	Neurotoxicants	**1.2E+01**
	Reproductive / developmental toxicants	**9.0E+00**

Notes:
[1] Values in **bold** indicate an RQ greater than 1.0. With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.
[2] Chronic inhalation RQ values were added to the multiple pathway RQ values for the aromatic C_9-C_{16} group, methyl ethyl ketone group, aluminum, barium, cadmium, manganese, selenium and silver because the chronic inhalation and chronic oral exposure limits for these COPC were based on the same toxicological endpoint.
[3] For individual constituents of the chemical mixtures, see Table 2-11. Note that addition of the individual constituents' RQ values might not equate to the RQ provided for the mixture because all of the RQ values in this table represent the maximum RQ values for each receptor group, regardless of the location at which exposure occurred.

3.4.2 Carcinogens

The carcinogenic assessment for the pit lakes focused on incremental exposures associated with the predicted water quality in these lakes, primarily because of wildlife exposure to the pit lake water, given that human access will be restricted. Table 3-38 presents the incremental lifetime cancer risks for the three carcinogenic COPC assessed in the multiple pathway assessment. For the three carcinogenic COPC, incremental lifetime cancer risks are less than 1.0, indicating that incremental lifetime cancer risks are below the acceptable level of 1 in 100,000 risk or considered essentially negligible.

Table 3-38 Chronic Multiple Pathway Incremental Lifetime Cancer Risks for the Pit Lake Scenario – Aboriginal Group (Cabin and Community Locations)

Chemical of Potential Concern[1]		Incremental Lifetime Cancer Risks (per 100,000)[2]
		Pit Lake Scenario
Organics	Benzo(a)pyrene group	1.1E-01
	Propylene oxide	1.0E-02
Metals	Arsenic	3.4E-01

Notes:
[1] Chronic inhalation ILCR values were added to the ILCR values associated with the secondary pathways of exposure for arsenic because the inhalation and oral exposure limits for arsenic are based on a similar toxicological endpoint.
[2] An ILCR equal to or less than 1.0 signifies an incremental lifetime cancer risk that is below the benchmark ILCR of 1 in 100,000 (i.e., within the generally accepted limit deemed to be protective of public health). With scientific notation, any value expressed to the negative power (i.e., E-x) shows that predicted exposures were less than the exposure limit; whereas, a value expressed to the positive power (i.e., E+x) shows exposure estimates exceeded the exposure limit.

3.5 Lead

Health Canada (2013) along with a number of other agencies (e.g., US EPA 2006, Cal EPA 2009, WHO 2009, JECFA 2011, ACCLPP 2012) no longer support the premise that lead is a threshold toxicant. On this basis, Health Canada has concluded that the interim exposure limit of 1.85 µg/kg bw/day (Health Canada 2009) is no longer valid. Due to the uncertainty in identifying a level without an adverse effect, there is currently no exposure limit available to evaluate risks from inhalation or oral lead exposures. In light of the uncertainty regarding regulatory guidance and the lack of an inhalation or oral exposure limit, an alternative method of evaluation was used to assess the potential risks from inhalation and oral exposures to lead.

As suggested by Wilson and Richardson (2013), toxicokinetic approaches or physiologically-based pharmacokinetic (PBPK) models can be used to predict the blood lead level (BLL) of children due to lead exposures from multiple exposure pathways (i.e., air, soil, water and food). The PBPK models provide a scientifically sound means to predict the target tissue dose of chemicals in humans who are exposed to environmental levels (ATSDR 2007). In the 2013 HHRA, the Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children (IEUBKwinv1.1 build 11; US EPA 2010c) was used to predict the incremental changes in BLL from multiple exposure pathways associated with the PRM. The focus of the IEUBK model is on infants and children (i.e., 0 to 84 months of age) who have been identified as susceptible subpopulations, and on neurodevelopmental effects since the critical health effect is considered protective for other adverse health effects of lead across the entire population (Health Canada 2013).

Effects associated with BLLs below 10 µg/dL (down to 1 to 2 µg/dL) include neurodevelopmental, neurodegenerative, cardiovascular, renal, and reproductive effects. The evidence of an association between health effects with BLLs in the lower range of exposure is strongest for neurodevelopmental effects in children, most commonly assessed as a reduction of intelligence quotient (IQ) and attention-related behaviours (CalEPA 2009, JECFA 2011, Health Canada 2013). Current evidence suggests that a 1 to 2 µg/dL BLL may be associated with 1 IQ decrement on a population basis (Cal EPA 2009, Health Canada 2013). The study by Lanphear et al. (2005) has been established as the critical study for the characterization of adverse effects of lead on children's IQ score. The Lanphear et al. (2005) study

analyzed data from seven longitudinal studies, but excluded potentially important covariates from the pooled analysis, such as socioeconomic status, nutritional status, and paternal IQ. In addition, since the Lanphear et al. (2005) study was conducted, BLLs have declined considerably; therefore, there is uncertainty regarding the extrapolation of the dose-response curve to the current Canadian population (Health Canada 2013). In the case of IQ, a 1 point decrement is an extremely subtle effect that could never be reliably measured, detected, or attributed on an individual basis (Wilson and Richardson 2013). Finally, there is uncertainty associated with adverse health effects observed at BLLs as low as 1 to 2 µg/dL (Health Canada 2013).

The 2013 HHRA conservatively used the 1 to 2 µg/dL BLL increase as a point of reference for comparison of predicted PRM-related BLL in children. Similarly, the OEHHA uses a threshold blood concentration with a source-specific "benchmark change" of 1 µg/dL (Cal EPA 2007). The change in BLL is intended to be used as a *de minimus* increase in BLL resulting from exposure to environmental lead (Cal EPA 2007). The "benchmark change" of 1 µg/dL is based on the dose–response modelling conducted by California OEHHA (Cal EPA 2007) and EFSA (2010), where each incremental increase in BLL of 1 µg/dL is associated with approximately 1 point deficit in IQ.

Background lead levels in Alberta and Canada are available as an alternative point of reference. Lead concentrations in blood serum of pregnant Albertan women were largely below quantification limits (< 0.2 µg/L), except for a few samples that ranged from 0.2 to 1.0 µg/L (AHW 2008). The blood serum levels measured in Alberta women cannot be directly compared with the 1 to 2 µg/dL BLL (i.e., whole blood) because serum is known to contain only a small fraction (1%) of total lead.

Canadian levels of lead in blood and urine were measured in all participants aged 6 to 79 years in the Canadian Health Measures Survey (CHMS) between 2007 and 2009. The BLL for children aged 6 to 11 years are presented as µg/dL in whole blood in Table 3-39 (Health Canada 2013). The data provide reference ranges for blood levels of lead in the Canadian population. Since the CMHS (Health Canada 2013) failed to collect data from 1 to 6 year old children, the sensitive population group for lead exposure, standard practice is to use data from the US population National Health and Nutrition Examination Survey (NHANES) as a surrogate (Table 3-39).

Table 3-39 Arithmetic Mean, Geometric Mean and 95th Percentile Blood Lead Concentrations (µg/dL) for the Canadian Population

Sex	Age	Geometric Mean (95% Confidence Interval)	95th Percentile (95% Confidence Interval)
Male and Female[(1)]	1-5	1.51 (1.37 to 1.66)	4.10 (3.40 to 5.19)
Male and Female[(2)]	1-5	1.33 (1.20 to 1.39)	3.83 (3.39 to 4.40)
Male and Female[(3)]	6-11	0.90 (0.81 to 0.99)	1.95 (1.65 to 2.26)
Female[(3)]	20-39	1.12 (1.01 to 1.21)	3.12 (2.75 to 3.49)

Notes:
[(1)] Based on 2007-2008 NHANES dataset (Health Canada 2013).
[(2)] Based on 2007-2010 NHANES dataset (CDC 2012, 2013).
[(3)] Based on 2007-2009 Canadian Health Measures Survey Health Canada (2013).

Finally, predicted background lead exposure in First Nations people is available as an alternative point of reference. Based on data collected as part of the First Nations, Food, Nutrition and Environment Study (FNFNES), average daily intake of lead from food and tap water for BC First Nations people living on reserve was estimated to be 0.23 µg/kg body weight per day (Chan et al. 2011). This estimated exposure was converted to a daily intake of 3.8 and 7.6 µg/day for the toddler and child, respectively, which was based on a body weight of 16.5 and 32.9 kg for the toddler and child (Health Canada 2010a). It is important to note that consumption of some game meat may be associated with an increased risk of lead exposure due to lead shot contamination (Chan et al. 2011).

The input values that were used in the IEUBK model to predict BLL in children for the Aboriginal Group are presented in (Table 3–40). All remaining input values in the IEUBK model were unchanged or remained at default values.

Table 3-40 Input Parameter Values Used in the IEUBK Model to Predict Pierre River Mine-Related Blood Lead for Children in the Aboriginal Group

Parameter	Age Group	Aboriginal Group Pierre River Mine	Reference
Air concentration [µg/m^3]	All ages	2.2E-05	Predicted
Dietary exposure [µg/day][(1)]	Toddler[(2)]	2.1E-01	Predicted
	Child[(3)]	3.1E-01	Predicted
Soil concentration [mg/kg]	All ages	4.7E-03	Predicted
Drinking water concentration [µg/L]	All ages	2.3E-02	Predicted
Dermal exposure[(4)]	All ages	0.0E+00	Health Canada 2013
Mother's blood lead concentration at childbirth [µg Pb/dL]	Adult	1.1E+00	Table 3-39

Notes:
(1) Sum the following dietary items: plant, berries, Labrador tea, root, cattail, fish, moose, ruffed grouse, and snowshoe hare.
(2) Toddler dietary exposure assumed constant for the following age groups in the IEUBK model: 1-2, 2-3, and 3-4.
(3) Child dietary exposure assumed constant for the following age groups in the IEUBK model: 4-5, 5-6, and 6-7.
(4) The dermal route is not considered to be a significant route of exposure to lead (Health Canada 2013).

Based on the IEUBK model, the predicted geometric mean BLL in children is 0.064 µg/dL for the Aboriginal group (Table 3-41). Increased risk of adverse effects from lead exposures in air, soil, water and diet due to the PRM are not expected based on the following:

- Predicted BLL are two orders in magnitude lower than background levels in children presented in Table 3-39; and
- The probability of exceeding the point of reference value of 1 to 2 µg/dL is 0% for the Aboriginal group (Table 3–41).

Table 3-41 Predicted PRM-Related IEUBK Lead Concentrations for Children in the Aboriginal Group

Parameter	Aboriginal Group
Predicted geometric mean BLL in children aged 0 to 84 months	0.064 µg Pb/dL
Probability of blood lead concentrations above cut-off value of 1 µg/dL	0%
Probability of blood lead concentrations above cut-off value of 2 µg/dL	0%

3.6 Naphthenic Acids

Because a health-based exposure limit is currently not available for naphthenic acids, it remains difficult to quantify the likelihood of adverse human health effects.

The likeliest route of exposure to naphthenic acids is through direct contact with water to which they are released, either through the ingestion pathway or dermal pathway. Based on Raoult's Law, the total vapour pressure of naphthenic acids is expected to be exceedingly low (API 2003). This indicates that volatilization will not be an important fate process. As such, exposure is not expected to occur through inhalation. Calculating the multimedia distribution for a range of molecular weight and ring structures of naphthenic acids found in oil sands extracts revealed that, following a release to surface water, the principle distribution of these constituents over time would be to sediment (Rogers et al. 2002). In their recent review of the health effects of naphthenic acids, Kindzierski et al. (2012) stated that the "properties of aged OSPW-derived [naphthenic acids] (i.e., low octanol water partition values and apparent rapid depuration) offer no meaningful scientific evidence to support the fish ingestion pathway as being important for potential human exposure to these compounds".

Therefore, research conducted to date suggests that the most plausible route of exposure is directly related to water, as opposed to secondary (or indirect) pathways such as fish ingestion. However, because there are no drinking water quality guidelines or other health-based guidelines available for naphthenic acids, health risks can only be assessed on a qualitative basis at this time.

Concentrations of naphthenic acids in watercourses and water bodies were predicted using the models and methods presented in the EIA, Volume 4B, Appendix 4-2. These prediction methods have been followed in this submission, except that assumptions regarding naphthenic acid degradation and speciation have been updated to align with the End Pit Lake Guidance Document (CEMA 2012). In the Athabasca River, the maximum predicted naphthenic acid concentrations are 0.00036 mg/L for the 2013 Base Case, and 0.00038 mg/L for the 2013 PRM Application Case and 2013 PDC. The maximum predicted concentrations for naphthenic acids in the PRM small streams are 0.58 mg/L, 0.62 mg/L and 1.6 mg/L for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC, respectively.

Ambient levels of naphthenic acids in the Athabasca Oil Sands watersheds range from non-detectable to 2 µg/L (Headley and McMartin 2004; WRS 2003). This suggests that the maximum predicted concentrations remain within the typical range of background levels (<2 mg/L) in the region.

4.0 Conclusions

Consistent with the EIA HHRA, for the 2013 HHRA, consideration was given to PRM-related emissions or releases predicted to result in changes to environmental quality. These included possible PRM emissions to air and releases to water. The 2013 HHRA is based on the JRP request for a re-evaluation of the effects associated with the PRM alone (i.e., without inclusion of the JME), along with an assessment of the 2013 PDC.

Overall, emissions from the PRM alone, and in combination with emissions from other sources, are not expected to result in adverse health effects in the area. The changes between the 2013 Base Case and 2013 PRM Application Case risks are generally small, suggesting that the PRM is not expected to contribute appreciably to health risks in the region. Similarly, the changes between the 2013 Base Case and 2013 PDC risks are generally small. Cumulative environmental risks associated with the additional projects and activities planned for the region are not expected to result in adverse health effects.

Based on the re-analysis, the 2013 PDC and the exclusion of the JME from the 2013 PRM Application Case do not alter the assessment results or the conclusions originally presented in the EIA HHRA.

The conclusions of the acute inhalation assessment, chronic inhalation assessment and multiple pathway assessment are described below.

4.1 Acute Inhalation Health Risks

Consistent with the EIA HHRA, acute health risks associated with PRM and cumulative air emissions were characterized by comparing predicted peak short-term air concentrations with health-based exposure limits.

The majority of the predicted acute inhalation risks, when expressed as risk quotients, do not exceed 1.0 with the exceptions of acrolein, aliphatic aldehyde group, aromatic C_9-C_{16} group, and SO_2 in the 2013 Base Case, 2013 PRM Application Case and 2013 PDC. For these COPC, existing and 2013 Base Case emission sources appeared to be associated with the most risk. An analysis of the magnitude of the predicted air concentrations as well as the frequencies with which exceedances may occur, revealed a considerable degree of conservatism in the assessment. Overall, the anticipated health risks associated with short-term exposures to the COPC are expected to be low. The eye, nasal and respiratory irritant mixtures are also associated with exceedances because of the predicted exceedances for some of the constituent COPC. However, because of the conservative nature of the 2013 HHRA, the overall health risks associated with these acute mixtures are expected to be low.

The differences between the 2013 Base Case and 2013 PRM Application Case risks are negligible to low, indicating that the PRM will not materially increase the acute inhalation risks at the nearby cabins, communities or industrial camp sites.

The overall conclusions of the current acute inhalation assessment are consistent with those presented in the EIA HHRA.

4.2 Chronic Inhalation Health Risks

Adopting the same approach that was employed in the EIA HHRA, chronic health risks associated with the air emissions from the PRM were characterized by comparing predicted maximum long-term air concentrations with health-based guidelines.

Overall, the majority of the predicted long-term air concentrations of the COPC are predicted to be below their health-based guidelines. The exceptions are associated with chronic exposures to the

aliphatic aldehyde group, aromatic C_9-C_{16} group, H_2S, and trimethylbenzenes. Because of the conservatism incorporated into the chronic inhalation assessment, the predicted risks are anticipated to overstate the actual risks. The predicted exceedances for the mixtures are due primarily to the conservatism built into the individual COPC risk quotients.

The PRM emissions are not expected to have a material impact on the potential health risks associated with the long-term inhalation of the individual COPC or mixtures. Despite some of the predicted exceedances, the weight of evidence indicates that there is low potential for adverse health effects associated with long-term inhalation of emissions from the PRM alone or when considered in combination with emissions from other sources in the region.

The inhalation cancer risks are predicted to be low for the PRM. For the carcinogens, predicted risk estimates, expressed as incremental lifetime cancer risks, associated with the PRM and planned future emission sources in the area are all less than 1 in 100,000, indicating that the incremental cancer risk from the PRM and planned future developments are deemed to be negligible. The exception being the incremental lifetime cancer risk for benzene associated with the planned future emission sources only.

The incremental lifetime cancer risk for benzene for the PRM alone is predicted to be below 1 in 100,000, indicating that the incremental cancer risk from the PRM is deemed to be essentially negligible. The incremental increase in potential cancer risk for the planned future emission sources can be attributed to the projected increase in the Fort McMurray population and the associated increase in benzene emissions. As emissions from communities are difficult to accurately quantify, there is considerable uncertainty associated with these predictions for the future community emission sources.

The overall conclusions of the current chronic inhalation assessment are consistent with those presented in the EIA HHRA.

4.3 Chronic Multiple Pathway Health Risks

Like the EIA HHRA, health risks associated with multiple pathways of exposure were calculated by comparing exposure estimates with health-based exposure limits.

In most cases, the risk quotients for the non-carcinogenic COPC do not exceed 1.0. The exceptions include: manganese, methyl mercury, liver effects, kidney effects, neurological effects, and reproductive and developmental effects. For all of the chemical mixtures, the degree of conservatism incorporated into the assessment for each of the individual COPC in the mixture, as well as the assumption that the predicted adverse effects are additive, have likely resulted in the overstatement of the actual risks to these mixtures.

The differences between the 2013 Base Case and 2013 PRM Application Case risks are generally negligible, indicating that the PRM will not materially increase the multiple pathway risks at the nearby cabins, communities or industrial camp sites. Despite the predicted exceedances for some of the COPC and mixtures for the Aboriginal group, the weight of evidence indicates that there is low potential for

adverse health effects associated with long-term multiple pathway exposure to the individual COPC or mixtures in the region.

All predicted incremental cancer risks for COPC with carcinogenic oral exposure limits are less than 1 in 100,000. As such, the contribution from the PRM on potential cancer risks in the area in relation to human exposure via multiple pathways (food, water, soil, etc.) is anticipated to be negligible.

A pit lake scenario was evaluated to characterize the potential health risks associated with exposure to water from these lakes. The exceedances predicted for the pit lake scenario are comparable to the 2013 PDC for the Aboriginal group. In all instances, the conservative assumptions applied in the 2013 HHRA, specifically the exposure and toxicity assessments, likely resulted in the predicted risks being overstated.

The overall conclusions of the current chronic multiple pathway assessment are consistent with those presented in the EIA HHRA.

5.0 References

ACB (Alberta Cancer Board). 2009. *Cancer Incidence in Fort Chipewyan, Alberta 1995-2006*. Alberta Cancer Board, Division of Population Health and Information Surveillance.

ACCLPP (Advisory Committee for Childhood Lead Poisoning Prevention). 2012. *Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention*. Centers for Disease Control and Prevention. Available at: http://www.cdc.gov/nceh/lead/ACCLPP/Final_Document_030712.pdf

ACGIH (American Conference of Governmental Industrial Hygienists). 1992. *Naphthalene*. CAS: 91-20-3. Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. ACGIH®, Cincinnati. OH.

ACGIH. 2013. *TLVs® and BEIs® Based on the Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices*. ISBN: 978-1-607260-59-2. ACGIH®, Cincinnati. OH.

AENV (Alberta Environment). 2009. *Alberta Tier 1 Soil and Groundwater Remediation Guidelines*. Edmonton, AB. February 2009.

AHW (Alberta Health and Wellness). 1997. *Swan Hills Special Waste Treatment Centre Human Health Impact Assessment*. Health Surveillance Branch, Alberta Health and Wellness. October 1997.

AHW. 2000. *The Alberta Oil Sands Community Exposure and Health Effects Assessment Program*. Technical Report. Prepared by a Consortium of Government, University and Corporate Partners. Alberta Health Surveillance, Edmonton, AB.

AHW. 2008. *Alberta Biomonitoring Program: Chemicals in Serum of Pregnant Women in Alberta*. Edmonton: Alberta Health and Wellness. Surveillance And Environmental Health, Public Health Division. Available at: http://www.health.alberta.ca/documents/Chemical-Biomonitoring-2008.pdf

AHW. 2009a. *Human Health Risk Assessment – Mercury in Fish*. The Regional Aquatics Monitoring Program (RAMP). October 2009. ISBN 978-0-7785-8245-8.

AHW. 2009b. *Human Health Risk Assessment. Mercury in Fish in Central Alberta*. Surveillance and Environmental Health, Alberta Health and Wellness. Edmonton, Alberta. March 2009. ISBN 978-0-7785-7427-9. Cited from: Health Canada. 1999. Lesser Slave Lake Health Study. Unpublished. Medical Service Branch, Health Canada.

AHW. 2009c. *Human Health Risk Assessment*. *Mercury in Fish in Central Alberta*. Lac La Nonne and Lac Ste. Anne. March 2009. ISBN: 978-0-7785-7428-6

AHW. 2009d. *Human Health Risk Assessment*. *Mercury in Fish*. Pine Coulee and Twin Valley Water Management Projects, Southern Alberta. October 2009. ISBN: 978-0-7785-8242-7

AHW. 2011. *Guidance on Human Health Risk Assessment for Environmental Impact Assessment in Alberta*. August 2011.

AHW. 2012. *Interactive Health Data Application (IDHA)*. Available at: http://www.ahw.gov.ab.ca/IHDA_Retrieval/.

Andersen, M.E., Dorman, D.C., Clewell, H.J. III, Taylor, M.D. and Nong, A. 2010. *Multi-Dose Route, Multi-Species Pharmacokinetic Models for Manganese and their Use in Risk Assessment*. J Toxicol Environ Health Part A. 73: 217-234.

API (American Petroleum Institute). 2003. *Robust summary of information on reclaimed substances: Naphthenic Acid*. Report No. 201-14906B

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. *Toxicological Profile for Mercury*. US Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA. March 1999.

ATSDR. 2007. *Toxicological Profile for Lead*. US Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA. August 2007.

ATSDR. 2008. *Draft Toxicological Profile for Manganese*. US Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA. September 2008.

ATSDR. 2010. *Toxicological Profile for Ethylbenzene*. US Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA. November 2010.

ATSDR. 2013. *Minimal Risk Levels (MRLs) for Hazardous Substances*. US Department of Health and Human Services, Public Health Service. Atlanta, GA. February 2013. Available at: http://www.atsdr.cdc.gov/mrls/mrllist.asp

BCS (Bureau of Chemical Safety). 2004. *Fish Consumption: Review and Recommendation of Current Intake Figures for Canadian Consumers*. Available at: http://www.hc-sc.gc.ca/fn-an/pubs/mercur/merc_fish_poisson-eng.php

Boethling RS, Fenner K, Howard P, Klecka G, Madsen T, Snape JR, and Whelan MJ. 2009. *Environmental persisitence of organic pollutants: guidance for development and review of POP risk profile*s. Integr Environ Assess Manag 5(4)pp 539-556

Cal EPA (California Environmental Protection Agency). 2007. *Development of Health Criteria for School Risk Assessment Pursuant to Health and Safety Code Section 901(g): Child Specific Benchmark Change in Blood Lead Concentration for School Site Risk Assessment*. Integrated Risk Assessment Branch Office of Environmental Health Hazard Assessment California Environmental Protection Agency. Final Report. April 2007.

Cal EPA. 2009. *Revised California Human Health Screening Level for Lead (Review Draft)*. Office of Environmental Health Hazard Assessment, Sacramento, CA, USA. Available at http://www.oehha.ca.gov/risk/pdf/LeadCHHSL51809.pdf

Calabrese, E.J. 1991. *Multiple Chemical Interactions. Toxicology and Environmental Health Series*. Chelsea, MI: Lewis Publishers Inc.

CCME (Canadian Council of Ministers of the Environment). 2000. *Canada-Wide Standards for Particulate Matter (PM) and Ozone*. Canadian Council of Ministers of the Environment. June 5-6, 2000.

CCME. 2008. *Canada-Wide Standards for Petroleum Hydrocarbons (PHC) in Soil: Scientific Rationale*. Supporting Technical Document. January, 2008. ISBN 978-1-896997-77-3.

CCS (Canadian Cancer Society). 2013.*Canadian Cancer Statistics 2013*. Available at: http://www.cancer.ca/~/media/cancer.ca/CW/cancer%20information/cancer%20101/Canadian%20cancer%20statistics/canadian-cancer-statistics-2013-EN.pdf

CDC (Centre for Disease Control). 2012. *Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention.* Report of the Advisory Committee on Childhood Lead Poisoning Prevention of the Centers for Disease Control and Prevention. January 4, 2012

CDC. 2013. *Fourth National Report on Human Exposure to Environmental Chemicals Updated Tables, September 2012*. Updated February 2013.

CEMA (Cumulative Environmental Management Association). 2012. *End Pit Lake Guidance Document*. D. Wylynko and J. Hyrnyshyn (Eds.). September 2012. Fort McMurray, Ab.

Chan, L, O Receveur, D Sharp, H Schwartz, A Ing, and C Tikhonov. 2011. *First Nations Food, Nutrition and Environment Study (FNFNES): Results from British Columbia (2008/2009*). Prince George: University of Northern British Columbia, 2011.

CPHI (Canadian Population Health Initiative). 2006. *How Healthy are Rural Canadians? An Assessment of their Health Status and Health Determinants*. ISBN 13: 978-1-55392-881-2.

Darley, E., Middleton, J. and Garber, M. 1960*. Plant damage and eye irritation from ozone-hydrocarbon reactions*. Journal of Agriculture and Food Chemistry 8(6): 483-484.

Dorman, D.C., Brenneman, K.A. and Struve, M.F. 1999. *Experimental investigations into the neurotoxicity and nasal toxicity of hydrogen sulfide in rats*. Environmental Epidemiology and Toxicology 1:249-255.

EFSA (European Food Safety Authority). 2010. *Scientific opinion on lead in food*. EFSA Journal, 8(4): 1570. 147 pp. Available at: www.efsa.europa.eu/fr/scdocs/doc/1570.pdf

Environment Canada. 2003. *Guidance Manual for the Categorization of Organic and Inorganic Substances on Canada's Domestic Substances List*. Determining persistence, bioaccumulation

potential, and inherent toxicity to non-human organisms. Existing Substances Branch, Environment Canada. June 2003.

Environment Canada. 2007. *Use of Environmental Fate and Effects Criteria to Determine the Alternate Threshold of a Substance for the National Pollutant Release Inventory*. September 2007. Available at: https://www.ec.gc.ca/inrp-npri/default.asp?lang=en&n=889870FD-1

Fengxiang, X.H., Su, Y., Sridhar, B.B.M. and Monts, D.L. 2004. *Distribution, transformation and bioavialability of trivalent and hexavalent chromium in contaminated soil*. Plant and Soil 265:243-252.

Government of Canada. 1994. *Priority Substances List Assessment Report*. Chromium and its Compounds. ISBN 0-662-22047-1 Cat. No. En40-215/40E. Environment Canada, Health Canada, Canadian Environmental Protection Act.

Government of Alberta. 2012. *Fish Consumption Ad*visory. Available at: http://mywildalberta.com/Fishing/SafetyProcedures/FishConsumptionAdvisory.aspx

Harkema JR, Carey SA, Wagner JG. 2006. *The Nose Revisited: A Brief Review of the Comparative Structure, Function, and Toxicologic Pathology of the Nasal Epithe*lium. Toxicol Pathol 34: 252-269.

Headley J.V. and McMartin D.W. 2004. *A review of the occurance and fate of naphthenic acids in aquatic environments*. Jour. Environ. Sci. Health A Tox. Hazard Subst. Environ. Eng. 38(8); pp 1989-2010.

Health and Welfare Canada. 1991. *Report on Present patterns and trends in infant feeding in Canada*. Ottawa: Health and Welfare Canada. 1991.

Health Canada. 1994a. *Human Health Risk Assessment for Priority Substances*. Canadian Environmental Protection Act. Minister of Supply and Services Canada, 1994. Canada Communication Group. Ottawa, ON.

Health Canada. 1999. *Guidelines for Canadian Drinking Water Quality: Supporting Documentation, Antimony*. Canadian Drinking Water Quality Guidelines. May 1997 (edited August 1999).

Health Canada. 2001. *Uranium: Guidelines for Canadian Drinking Water Quality – Supporting Documentation.* Last edited January 2001.

Health Canada. 2004. *Federal Contaminated Site Risk Assessment in Canada*. Part I and II. Environmental Health Assessment Services Safe Environments Program. Ottawa, Ontario. September 2004. ISBN 0 662-38244-7 and ISBN 0-662-38245-5.

Health Canada. 2007. *Health Canada's revised assessment of mercury in fish enhances protection while reflecting advice in Canada's Food Guide*. Available at: http://www.hc-sc.gc.ca/ahc-asc/media/advisories-avis/2007/2007_31_e.html

Health Canada. 2009. *Memorandum: Interim CSD Guidance on a TRV for Lead (Pb) and Interpretation of Pb Bioaccessibility Data for Federal Contaminated Site Human Health Risk Assessment in Canada (December 2009)*.

Health Canada. 2010a. *Federal contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA)*. Version 2.0. Contaminated Sites Division, Safe Environments Programme, Ottawa, On.

Health Canada. 2010b. *Federal Contaminated Site Risk Assessment in Canada, Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors, Version 2.0.* Contaminated Sites Division, Safe Environments Programme, Ottawa, ON.

Health Canada. 2011. *Dietary Intakes of Contaminants & Other Chemicals for Different Age-Sex Groups of Canadians*. Average dietary intakes (µg/kg bw/day) of trace elements for Canadians in different age/sex groups for Total Diet Study in 2007. Available at: http://www.hc-sc.gc.ca/fn-an/surveill/total-diet/intake-apport/index-eng.php

Health Canada. 2013. *Final Human Health State of the Science Report on Lead*. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/dhhssrl-rpecscepsh/index-eng.php February 2013.

IOM (Institute of Medicine). 2001. *Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc*. Institute of Medicine, Food and Nutrition Board. Washington DC. National Academy Press. P. 10-1 to 10-22.

JECFA (Joint Expert Committee on Food Additives). 2011. *WHO Food Additive Series: 64 Safety Evaluation of Certain Food Additives and Contaminants*. Prepared by the seventy third meeting of JECFA, Joint FAO/WHO Expert Committee on Food Additives, ISBN 978 924 166064 8. Geneva, Switzerland

Kimbell, JS. 2006. *Nasal Dosimetry of Inhaled Gases and Particles: Where do Inhaled Agents go in the Nose?* Toxicol Pathol 34: 270-273.

Kindzierski WB, Chelme-Ayala P, El-Din MG. 2010. *Wood Buffalo Environmental Association Ambient Air quality Data Summary and Trend Analysis.* Report Summary. Department of Public Health Sciences. School of Public Health, University of Alberta. April 2010.

Kindzierski, W., J. Jin and M. Gamal El-Din. 2012. *Review of Health Effects of Naphthenic Acids: Data Gaps and Implications for Understanding Human Health Risk*. Oil Sands Research and Information Network, University of Alberta, School of Energy and the Environment, Edmonton, Alberta. OSRIN Report No. TR-20. 43 pp.

Kurek J., Kirk J.L., Muir D.C., Wang X., Evans M.S. and Smol J.P. 2013. *Legacy of a half century of Athabasca oil sands development recorded by lake ecosystems.* Proceedings of the National Academy of Sciences 110(5): 1761-1766.

Lanphear, B.P., Hornung, R., Khoury, J., Yolton, K., Baghurst, P., Bellinger, D.C., Canfield, R.L., Dietrich, K.N., Bornschein, R., Greene, T., Rothenberg, S.J, Needleman, H.L, Schnaas, L., Wasserman, G., Graziano, J. and Roberts, R. 2005. *Low-level environmental lead exposure and children's intellectual function: an international pooled analysis*. Environ. Health Perspect., 113: 894–899.

MA DEP (Massachusetts Department of Environmental Protection). 2003. *Updated petroleum hydrocarbon fraction toxicity values for the VPH / EPH / APH methodology final*. Boston, MA: Massachusetts Department of Environmental Protection.

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.

Mackay, D., Shiu, W. and Ma, K. 1993. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals: Volumes III*. Lewis Publishers. Chelsea, Michigan.

Market Facts of Canada. 1991. *Research Report: National Seafood Consumption Study*. Conducted for: Health and Welfare Canada. #C388/JdeB.

O'Connor and Richardson (O'Connor Associates Environmental Inc. and G. Mark Richardson). 1997. *Compendium of Canadian Human Exposure Factors for Risk Assessment*. Ottawa, Ontario.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. *Determination of Noncancer Chronic Reference Exposure Levels*. Chronic Toxicity Summary Ethylbenzene. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=208

OEHHA. 2008. *Acrolein Reference Exposure Levels*. TSD for Non-Cancer RELs: Appendix D1. December 2008.

Percy, K. (ed). 2012. *Alberta Oil Sands: Energy, Industry and the Environme*nt. Elsevier. ISBN: 978-0-08-097760-7.

Reznik G, Stinson SF (eds). 1983. *Nasal Tumours in Animals and Man*. Volumes 1 and 2. CRC Press, Boca Raton.

Reznik GK. 1990. *Comparative Anatomy, Physiology and Function of the Upper Respiratory Tract*. Environ Health Perspect 85: 171-176.

Rogers, V.V., Wickstrom, M., Liber,K. and MacKinnon, M.D. 2002. *Acute and subchronic mammalian toxicity of naphthenic acids from oil sands tailings*. Toxicological Sciences 66:347-355.

RSCEP (Royal Society of Canada Expert Panel). 2010. *Environmental and health impacts of Canada's oil sands industry*. Report. The Royal Society of Canada. The Academies of Arts, Humanities and Sciences of Canada. December 2010.

Santamaria, A.B. and Sulsky, S.I. 2010. *Risk Assessment of an Essential Element: Manganese*. J Toxicol Environ Health Part A 73: 128-155.

SRC (Syracuse Research Corporation). 2011. *Interactive PhysProp Database Demo*. SRC Inc., 2011. Available at: http://www.syrres.com/what-we-do/databaseforms.aspx?id=386

Tanaka, P.A., Yeung, D.L. and Anderson, G.H. 1987. *Infant feeding practices: 1984-1985 versus 1977-197*8. Can. Med. Assoc. J. 136, 940-944.

TCEQ (Texas Commission on Environmental Quality). 2008. *Formaldehyde (CAS Registry Number: 50-00-0). Development Support Document, Final, August 7, 2008*. Prepared by: Joseph T. Haney, Jr., Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality. Available at: http://www.tceq.state.tx.us/assets/public/implementation/tox/dsd/final/formaldehyde_50-00-0_final.pdf

TCEQ. 2009. *Methacrolein (CAS Registry Number: 78-85-3*). Development Support Document, Final, October 2009. Prepared by: Jessica L. Corarrubia and Roberta L Grant, Toxicology Division, Chief Engineer's Office. Texas Commission on Environmental Quality. Available at: http://www.tceq.com/assets/public/implementation/tox/dsd/final/october09/methacrolein_78-85-3.pdf

TCEQ. 2010a. *Acrolein. CAS Registry Number: 107-02-8*. Final Development Support Document. November 2010. Available at: http://tceq.com/assets/public/implementation/tox/dsd/final/nov10/acrolein.pdf

TCEQ. 2010b. *Ethylbenzene (CAS Registry Number: 100-41-4).* Developmental Support Document, Final, November 19, 2010. Prepared by: Manuel Reyna. Toxicology Division, Chief Engineer's Office, Texas Commission on Environmental Quality.

TPHCWG (Total Petroleum Hydrocarbon Criteria Working Group). 1997. *Development of fraction specific reference doses (RfDs) and reference concentrations (RfCs) for total petroleum hydrocarbons (TPH).* TPHCWG Series. Volume 4. Amherst Scientific Publishers. Amherst, MA. ISBN 1-884-940-13-7

US EPA (United States Environmental Protection Agency). 1994. *IRIS Summary of Cadmium (7440-43-9). Reference Dose for Chronic Oral Exposure (RfD)*. Available at: http://www.epa.gov/ncea/iris/subst/0141.htm#reforal

US EPA. 1996. IRIS Summary of Manganese (CASRN 7439-96-5). *Reference Dose for Chronic Oral Exposure (RfD)*. Available at: http://www.epa.gov/ncea/iris/subst/0373.htm#reforal.

US EPA. 2000. *IRIS Summary of Benzene (CASRN 71-43-2). Carcinogenicity Assessment for Lifetime Exposure.* Available at: http://www.epa.gov/ncea/iris/subst/0276.htm#carc.

US EPA. 2002. *A Review of the Reference Dose and Reference Concentration Process: Risk Assessment Forum.* Washington, DC: Risk Assessment Forum, United States Environmental Protection Agency. EPA/630/P-02/002F. December 2002.

US EPA. 2003. *Attachment 1-3 Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs) Evaluation of Dermal Contact and Inhalation Exposure Pathways for the Purpose of Setting Eco-SSL*s. OSWER Directive 92857-55. November 2003.

U.S. EPA. 2005. 2005 *National Emissions Inventory Data and Documentation*. Available at: http://www.epa.gov/ttnchie1/net/2005inventory.html.

US EPA. 2006. *National Ambient Air Quality Standards: Air Quality Criteria for Lead*. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=158823

US EPA. 2008. *Acute Exposure Guideline Levels for Selected Airborne Chemicals Volume 6*. The National Academies Press, Washington, DC. For Crotonaldehyde AEGLs pp 123. Available at: http://www.epa.gov/oppt/aegl/pubs/crotonaldehyde_final_volume6_2007.pdf

US EPA. 2009. *Propionaldehyde (CAS Reg. No. 123-38-6) Interim Acute Exposure Guideline Levels (AEGLs) for NAS/COT Subcommittee for AEGLS*. Available at: http://www.epa.gov/oppt/aegl/pubs/propionaldehyde_interim_dec_2008.pdf

US EPA. 2010a. *40 CFR Parts 50 and 58. Primary National Ambient Air Quality Standards for Nitrogen Dioxide*. Final Rule.

US EPA. 2010b. *Code of the Federal Register. Environmental Protection Agency. 40 CFR Parts 50, 53 and 58. Primary National Ambient Air Quality Standard for Sulfur Dioxide: Final Ru*le.

US EPA. 2010c*. Integrated Exposure Uptake Biokinetic Model for Lead in Children, Windows® version (IEUBKwin v1.1 build 11).* February 2010 32 bit version.

US EPA. 2011. *Estimation Programs Interface Suite™ for Microsoft® Windows, v 4.10*. United States Environmental Protection Agency, Washington, DC, USA.

US EPA. 2013. *Integrated Risk Information System (IRIS) database on line search. A Z List of Substances*. Available at: http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showSubstanceList&list_type=alpha&view

WBEA (Wood Buffalo Environmental Association). 2007. *Human Exposure Monitoring Program. Part I – Methods Report, Part II – 2005 Monitoring Year Resu*lts. February 2007.

WBEA. 2013. *Media Release: Published book synthesizes years of air monitoring results in the Athabasca Oil Sands Region – January 31, 2013*. Available at: http://www.wbea.org/news-room/whats-new-

archive/251-media-release-published-book-synthesizes-years-of-air-monitoring-results-in-the-athabasca-oil-sands-region-january-31-2013

Weber-Tschopp. A., Fischer, T., Gierer, R. and Grandjean, E. 1977. *Experimentally induced irritating effects of acrolein on men*. Int. Arch Occup Environ Health 40(2):117-130.

Wein, EE. 1989. *Nutrient Intakes and Use of Country Foods by Native Canadians Near Wood Buffalo National Park.* Thesis presented to the Faculty of Graduate Studies, University of Guelph. February 1989.

Wein, EE, JH Sabry and FT Evers. 1989. *Food health beliefs and preferences of Northern Native Canadians*. Ecology of Food and Nutrition 23:177-188.

Wein, EE, JH Sabry and FT Evers. 1991. *Food Consumption Patterns and Use of Country Foods by Native Canadians near Wood Buffalo National Park, Canada*. Arctic 44(3):196-205.

WHO and FAO (Food and Agriculture Organization of the United Nations). 2007. *Evaluation of Certain Food Additives and Contaminants*. Sixty-seventh report of the Joint FAO WHO Expert Committee on Food Additives. WHO Technical Report Series 940. Geneva. Available at: http://whqlibdoc.who.int/trs/WHO_TRS_940_eng.pdf

WHO (World Health Organization). 2000. *Air Quality Guidelines for Europe, Second Editi*on. World Health Organization, Regional Office for Europe, Copenhagen.

WHO. 2004. *Manganese in Drinking-water*. Background document for development of WHO Guidelines for Drinking-water Quality.

WHO. 2009. *Blood Lead Levels in Children*. Factsheet 4.5, European Environment and Health Information System. Available at: http://www.euro.who.int/__data/assets/pdf_file/0003/97050/4.5. Levels of lead inchildrens blood EDITING_layouted.pdf

Wilson, R. and Richardson, G.M. 2013. *Lead (Pb) is now a non-threshold substance: How does this affect soil quality guidelines?* Human and Ecological Risk Assessment: An International Journal, Doi:10.1080/10807039.2013.771534.

WRS (Western Resource Solutions). 2003. *Development of Reach Specific Water Quality Objectives for Variables of Concern in the Lower Athabasca River: Identification of Variables of Concern and Assessment of the Adequacy of Available Guidelines.* Final report Prepared for: Cumulative Environmental Management Association Wood Buffalo Region. July 2003. ACB. 2005.

Yang, G., Yin, S., Zhou, R., et al. 1989. *Studies of safe maximal daily dietary selenium intake in a seleniferous area in China*. II. Relation between Se- intake and the manifestation of clinical signs and certain biochemical alterations in blood and urine. Journal of Trace Elements and Electrolytes in Health and Disease. 3(2): 123-130. Cited in: US EPA 1991.

SCREENING LEVEL
WILDLIFE HEALTH RISK ASSESSMENT for the SHELL PIERRE RIVER MINE UPDATE

[ATTACHMENT A to Appendix 3.3 - HUMAN HEALTH RISK ASSESSMENT FOR THE SHELL PIERRE RIVER MINE UPDATE]

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

1.0 INTRODUCTION

The primary objective of the Screening Level Wildlife Risk Assessment (SLWRA) is to describe the nature and significance of potential adverse effects to terrestrial wildlife that may be associated with chemical emissions from the Shell Canada Pierre River Mine (PRM). A population-level effect can be described as a decline or change in abundance or distribution of a wildlife population over time, such that natural recruitment is unable to re-establish the population to its original level (Suter II et al. 2000).

The Environmental Impact Assessment (EIA) of Shell's Jackpine Mine Expansion (JME) and PRM was submitted to the Alberta Energy Resources Conservation Board (now Alberta Energy Regulator) and Alberta Environment and Sustainable Resource Development (AESRD) in December 2007. The Wildlife Health Risk Assessment (WHRA) for the JME and PRM was presented in Volume 3, Section 5.4 of the EIA. The SLWRA presented herein provides additional information to the EIA WHRA, specifically as it relates to the potential effects associated with PRM (i.e., Joint Review Panel Supplemental Information Request # 5). For the purposes of this attachment, the original WHRA is referred to as the EIA WHRA and this assessment is referred to as the 2013 SLWRA.

Like the EIA WHRA, the 2013 SLWRA examines the short-term (acute) and long-term (chronic) health risks to wildlife that may be attributable to the PRM, combined with existing or approved developments, as well as with other proposed or planned regional developments. The 2013 SLWRA evaluates potential risks to wildlife associated with Chemicals of Potential Concern (COPCs) emitted from PRM. To assess potential risks to terrestrial wildlife, predicted chemical exposures are compared with inhalation Toxicity Reference Values (TRVs) and soil and surface water quality guidelines intended to be protective of the health of terrestrial wildlife.

Acute exposures generally extend over a period ranging from hours to days (i.e., 30 days or less). In contrast, chronic exposures occur continuously over extended periods ranging from months to years (i.e., 31 days or longer, throughout an animal's lifetime). As such, the temporal scope for the 2013 SLWRA extends from acute exposures of one hour in duration up to chronic exposures equivalent to an animal's lifetime.

Potential health risks to wildlife were assessed for the following three assessment cases:

- 2013 Base Case: includes existing environmental conditions and approved projects or activities.
- 2013 PRM Application Case: includes the 2013 Base Case with PRM added.
- 2013 Planned Development Case (PDC): includes the 2013 PRM Application Case plus other planned projects or activities reasonably expected to occur.

1.1 Issues and Assessment Criteria

In order to focus the scope of the 2013 SLWRA, specific assessment and measurement endpoints were identified. An assessment endpoint is defined as "the characteristic of the ecological system that is the focus of the risk assessment" and that needs to be protected. A measurement endpoint is defined as "the effect on an ecological component that can be measured and described in some quantitative fashion" (CCME 1996; Gaudet et al. 1994).

For the purpose of the 2013 SLWRA, the assessment endpoint was identified as potential effects on wildlife populations. The associated measurement endpoints included the following:

- Ratios, expressed as Hazard Quotients (HQ values), between maximum predicted chemical concentrations in air and corresponding inhalation TRVs.

- Comparison between predicted chemical concentrations in soil and their corresponding soil quality guidelines (SQGs).
- Comparison between predicted chemical concentrations in surface water and corresponding surface water quality guidelines (SWQGs).

The inhalation TRVs, SQGs, and SWQGs identified for the 2013 SLWRA are intended to be protective of wildlife populations. This means that if the predicted air concentrations, soil concentrations or surface water concentrations are less than the TRVs, SQGs, or SWQGs, respectively, impacts to wildlife health are not expected.

2.0 METHODS

The current assessment is a SLWRA conducted according to principles provided by Environment Canada and the Canadian Council of Ministers of the Environment (CCME) (CCME 1996; Gaudet et al. 1994).

The three tiers in an Ecological Risk Assessment include:

- Screening-Level Ecological Risk Assessment (SLERA) (i.e., like the 2013 SLWRA);
- Preliminary Quantitative Risk Assessment (PQRA); and,
- Detailed Quantitative Risk Assessment (DQRA).

In contrast to the EIA WHRA, the 2013 SLWRA is a screening level ecological risk assessment. The scope for the 2013 SLWRA in the initial tier employs conservative assumptions and readily available data. Using conservative assumptions regarding both chemical exposure and chemical toxicity to wildlife receptors provides a high degree of conservatism into the assessment. If the findings of the 2013 SLWRA were to be markedly different than those described in the EIA WHRA, the scope of the current assessment would be expanded to the detailed quantitative risk assessment originally presented in the EIA. However, further study was considered unnecessary if the SLWRA did not identify an impact to the terrestrial wildlife as a result of the PRM emissions.

The four steps of the 2013 SLWRA are shown in Figure 2-1 and include:

- **Problem Formulation or Planning Stage**: identification of the COPCs associated with the PRM emissions predicted for the scenarios of concern (e.g., dependent on assessment case, exposure averaging times), characterization of wildlife receptors potentially “at risk”, and determination of the relevant exposure pathways.
- **Exposure Assessment**: quantification of the potential amount or dose of each COPC received by wildlife receptors through all relevant exposure pathways.
- **Toxicity or Hazard Assessment**: determination of toxicity reference values for chemicals of concern based on levels of exposure associated with minimal impact to wildlife populations following exposure for a prescribed period (i.e., acute or chronic exposure).
- **Risk Characterization**: comparison of estimated exposures (determined in the exposure assessment) with maximum safe dose levels (established in the toxicity assessment) to identify potential health risks for the different assessment cases. This includes discussion of sources of uncertainty and how any uncertainty was addressed in the risk assessment.

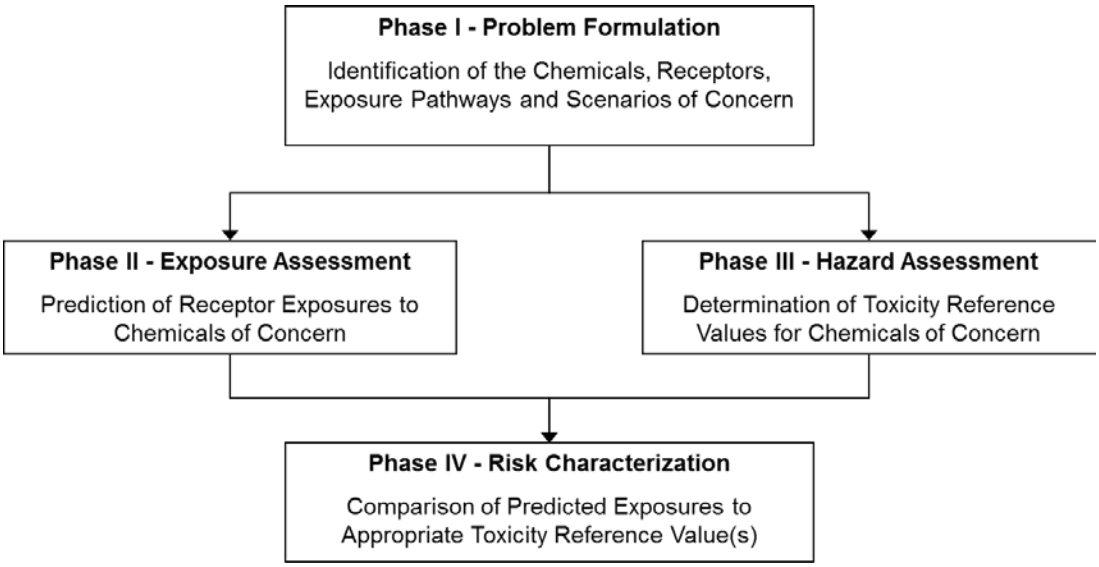


Figure 2-1 Ecological Risk Assessment Methodology

2.1 Problem Formulation

The purpose of the problem formulation, which includes the following steps, is to further focus the 2013 SLWRA:

- Identification of COPCs;
- Characterization of terrestrial wildlife receptors;
- Selection of relevant environmental media; and
- Identification of relevant exposure pathways.

2.1.1 Identification of Chemicals of Potential Concern

The identification of COPCs began with the review of an inventory of chemicals that could be released or emitted from the PRM, and to which wildlife might be exposed. Consideration was given to both air and water emissions from the PRM. The COPCs assessed in the 2013 SLWRA also took into consideration the availability of sufficient toxicological information to assess potential health risks.

In summary, the COPCs for the PRM were identified through:

- Development of an inventory of chemicals that could be emitted or released by the PRM;
- Determination of whether or not sufficient toxicological information was available to assess potential health risks (i.e., through use of available regulatory exposure limits or guidelines);
- Selection of chemical surrogates or assignment to appropriate chemical groups for the compounds for which no suitable exposure limits were available.

Table 2-1 lists the COPCs assessed in the 2013 SLWRA.

Table 2-1 Summary of Chemicals of Potential Concern for the Pierre River Mine Project

Chemical Category	Chemical
Criteria Air Contaminants (CACs)	Carbon monoxide (CO)
	Nitrogen dioxide (NO_2)
	Particulate matter ($PM_{2.5}$)
	Sulphur dioxide (SO_2)
Organic Compounds	1,3-Butadiene
	Acetaldehyde
	Acetone
	Acrolein
	Aliphatic aldehydes
	Aliphatic C_2-C_4
	Aliphatic C_5-C_8
	Aliphatic C_9-C_{16}
	Aliphatic C_{17}-C_{34}
	Ammonia[1]
	Aromatic C_9-C_{16}
	Aromatic C_{17}-C_{34}
	Benzene
	Cyclohexane
	Dichlorobenzene
	Ethylbenzene
	Formaldehyde
	Hexane
	Isopropylbenzene (cumene)
	Methyl ethyl ketone group[2]
	Phenol[1]
	Propylene oxide
	Toluene
	Trimethylbenzenes
	Xylenes
Polycyclic Aromatic Hydrocarbons (PAHs)	Carcinogenic PAHs[3]
	Naphthalene
	Pyrene
Metals and Minerals	Aluminum
	Antimony
	Arsenic
	Barium
	Beryllium
	Boron[1]
	Cadmium
	Chromium
	Chromium VI
	Cobalt
	Copper
	Lead
	Lithium[1]
	Manganese
	Mercury
	Methyl mercury[4]
	Molybdenum
	Nickel
	Selenium
	Silver

Table 2-1 Summary of Chemicals of Potential Concern for the Pierre River Mine Project (continued)

Chemical Category	Chemical
Metals and Minerals (continued)	Strontium[2]
	Thallium[1]
	Uranium
	Vanadium
	Zinc
Sulphur Compounds	CS_2 group[5]
	H_2S group[6]

Notes:
(1) Chemical not emitted into air or soil, so only assessed in the water quality assessment.
(2) Includes methyl ethyl ketone, 3-buten-2-one, camphor, and valencane.
(3) Includes carcinogenic PAH group 1, carcinogenic PAH group 2, and carcinogenic PAH group 3.
(4) Although the Project will not emit methyl mercury directly to the environment, it might release inorganic mercury into surface water. Bio-transformation of inorganic mercury to methylated organic species in water bodies can occur in sediment and in the water column. Methylation is the key step in the entrance of mercury into the food chain (U.S. EPA 1997). On this basis, methyl mercury, in addition to mercury, was identified as a COPC in the 2013 SLWRA.
(5) Includes carbon disulphide (CS_2) and carbonyl sulphide.
(6) Includes hydrogen sulphide (H_2S) and mercaptans.

Chemicals in the initial emissions inventory were retained as COPCs in the 2013 SLWRA if TRVs or guidelines were available. For example, particulate matter ($PM_{2.5}$) was not assessed in the 2013 SLWRA due to lack of available toxicity data for avian and mammalian wildlife.

To assess the potential risks to wildlife associated with non-inhalation pathways, it was necessary to identify those chemicals emitted by the PRM that, although emitted into air, could potentially be deposited onto the ground surfaces and possibly persist or accumulate in sufficient quantities for wildlife exposure via soil or surface water.

To identify persistent COPCs that could deposit and accumulate in soils, consideration was given to the physical-chemical properties of the chemicals that influence their fate and persistence in the environment, and subsequently their potential occurrence in the secondary pathways of exposure (e.g., plants). Inorganic COPC (i.e., metals) were automatically included in the soil and surface water assessment.

The purpose of the physical-chemical screening was to assess the potential health risks associated with exposure via deposition of persistent chemicals to the local environment. As part of the physical-chemical screening, organic chemicals from the emissions inventory were evaluated based on the chemical's volatility and potential for accumulation and persistence in the terrestrial environment. The physical-chemical screening process was described in Section 2.3.2 of the human health risk assessment (HHRA) Update (Table 2-8).

2.1.2 Wildlife Receptor Identification

Wildlife species that frequent the area, including resident and migratory populations, could potentially be exposed to chemicals emitted from PRM. For this 2013 SLWRA, potential risks to wildlife were not assessed for individual species, but instead, consideration was given to wildlife species more generically as part of the avian group (bird species) or as part of the mammalian group (representing all small and large terrestrial animals).

2.1.3 Environmental Media Identification

In order to assess potential risks to wildlife through multiple exposure pathways, potential changes in soil quality as a result of atmospheric deposition from air emissions were estimated in the 2013 SLWRA. Predicted concentrations of COPCs deposited onto soil were screened against soil quality guidelines for the protection of wildlife mammalian and avian species. In addition, potential changes in surface water quality were based on the Surface Water Quality Assessment (Appendix 2, Section 2.0). Predicted concentrations of COPCs in surface water are screened against water quality guidelines for the protection of terrestrial wildlife.

2.1.4 Identification of Exposure Pathways

Avian and mammalian species could be exposed to the emissions from the PRM both directly, through inhalation, and indirectly through the ingestion of environmental media such as soil, vegetation, water and other animals (i.e., prey).

Maximum predicted air concentrations are used for assessing acute and chronic inhalation exposures to wildlife receptors in the PRM area. Although inhalation is generally considered to be a minor wildlife pathway as oral pathways predominate for uptake of chemicals (Environment Canada 1994; Suter II et al. 2000; US EPA OSW 2005), the inhalation pathway is included in the 2013 SLWRA for the following reasons:

- The chemical emissions from PRM will be directly emitted into the atmosphere.
- Some of the emitted COPCs will be volatile, so the inhalation pathway could be an important exposure pathway for those chemicals.

For the inhalation assessment, maximum predicted chemical concentrations were compared with the corresponding available inhalation mammalian and avian TRVs. It is assumed that if predicted COPC concentrations in air were below the TRVs, air emissions associated with PRM would not pose a threat to wildlife populations.

For the multiple pathway assessment, ingestion is assumed to be the primary exposure pathway for the non-volatile and inorganic COPCs that have the potential to accumulate in the terrestrial environment. After chemicals are deposited onto soils, they can become incorporated into the upper profile of the soils and may be taken up by vegetation, they may remain deposited on vegetation, or may be sequestered into soils and soil dwelling organisms (i.e., potentially accumulating in wildlife foods). Wildlife receptors could potentially be exposed to COPC through direct contact with environmental media. The consideration of oral exposures also included surface water consumption by wildlife for COPC that may potentially be deposited onto surface waters.

For the 2013 SLWRA, predicted soil concentrations were compared to the following:

- ESRD SQGs (ESRD 2010);
- United States Environmental Protection Agency Ecological Soil Screening Levels (Eco-SSLs) (US EPA 2010); and
- Ontario Ministry of the Environment standards for metals and PAHs protective of ecological receptors (OMOE 2011).

It was assumed that if predicted COPC concentrations in soil met the ESRD SQGs or Eco-SSLs or OME standards, corresponding wildlife food chain concentrations (i.e., soil and food) would not pose a risk to

local wildlife populations. Dermal exposure was not considered in the SLWRA, as it is generally insignificant relative to exposure received through food, water, and soil ingestion (Suter II et al. 2000; US EPA OSW 2005).

For the 2013 SLWRA, predicted surface water concentrations were compared to ESRD (ESRD 2010) SWQGs, to be protective of wildlife water consumption. It was assumed that if predicted COPC concentrations in surface water met the ESRD SWQGs, corresponding wildlife exposures would not pose a risk to local wildlife populations.

2.2 Exposure Assessment

Determination of potential exposures via inhalation and ingestion of COPCs relied on predictive exposure modelling (e.g., air quality and water quality). In addition, soil data were collected from the local study area in order to characterize the existing COPC concentrations in soil.

2.2.1 Maximum Predicted Air and Soil Concentrations

For the inhalation assessment, maximum predicted 1-hour, 24-hour, and annual ground-level air concentrations of COPCs were used. Predicted chemical group air concentrations were estimated by summing the maximum predicted air concentrations for each of the constituent COPCs included in the chemical group.

The concentrations of COPCs in soil were predicted for the three assessment cases (2013 Base Case, 2013 PRM Application Case, and 2013 PDC) based on predicted modelled air concentrations. The soil concentrations were predicted using models that estimated the movement of the COPCs emitted by PRM onto soil. In addition, soil concentrations were based on the measured soil data if available. Soil concentrations were based on the 95 UCLM (95% upper confidence limit on the mean) or maximum values depending on the percentage of detected values.

Chemicals identified to be emitted directly to surface water were automatically included in the multiple pathway assessment. Chemicals identified as COPCs in the air emissions inventory were included in the physical-chemical screening in order to determine whether the chemical will be carried forward into the multiple pathway exposure assessment. Table 2-2 presents those chemicals included in the multiple pathway assessment identified either from the water emissions inventory, or from the physical-chemical screening of the air emissions.

Table 2-2 Chemicals Selected for Inclusion in the Multiple Pathway Assessment

Chemical Category	Chemical
Organic Compounds	Acetaldehyde
	Acetone
	Acrolein
	Aliphatic aldehydes
	Aliphatic C_9-C_{16} group
	Aliphatic C_{17}-C_{34} group
	Ammonia
	Aromatic C_9-C_{16} group
	Aromatic C_{17}-C_{34} group
	Formaldehyde
	Methyl ethyl ketone group
	Phenol
	Propylene oxide
PAHs	Acenaphthenes/Acenaphthylenes
	Anthracene/phenanthrenes and substituted
	Biphenyl
	Carcinogenic PAH group 1
	Carcinogenic PAH group 2
	Carcinogenic PAH group 3
	Fluorenes/fluoranthenes and substituted fluorenes
	Naphthalene and substituted naphthalenes
	Pyrenes and substituted Pyrenes
Metals and minerals	Aluminum
	Antimony
	Arsenic
	Barium
	Beryllium
	Boron
	Cadmium
	Chromium
	Chromium VI
	Cobalt
	Copper
	Lead
	Lithium
	Manganese
	Mercury
	Methyl mercury
	Molybdenum
	Nickel
	Selenium
	Silver
	Strontium
	Thallium
	Uranium
	Vanadium
	Zinc

2.3 Toxicity Assessment

In the case of the inhalation assessment, both acute and chronic TRVs are identified for the COPCs.

In the soil quality assessment, ESRD SQGs, US EPA Eco-SSLs and OMOE standards are considered protective of local wildlife populations exposed to the COPCs through secondary exposure pathways (e.g., soil ingestion and the food chain).

2.3.1 Identification of Acute and Chronic Inhalation Exposure Limits for Wildlife

The maximum predicted ground-level air concentrations for the COPCs were compared to the TRVs for each of the COPCs predicted in the three assessment cases. If maximum predicted ground-level air concentrations were equal to or lower than the TRVs, it was assumed that all wildlife receptors would be protected from adverse health effects associated with inhalation of the COPCs.

In the case of the inhalation assessment, both acute and chronic exposure durations were assessed if TRVs were identified for each COPC on an acute and chronic basis.

Inhalation Toxicity Reference Values

Much of the information regarding the wildlife toxicity of the COPC was obtained from the medical and scientific literature related to the exposure of laboratory test animals such as mice, rats, and guinea pigs for mammalian species, and poultry for avian species. Virtually no studies have been identified in which actual wildlife species were exposed to COPCs under controlled conditions. The lack of wildlife toxicity data presents three challenges:

- Health effects data gathered from the laboratory animals must be extrapolated to the wildlife species being assessed. This may require the use of uncertainty factors to account for possible differences in physiology and uncertainty in sensitivity to the chemicals. The use of such uncertainty factors is a common practice in risk assessment.
- The study designs involved exposures of the laboratory test animals to a range of levels, often showing no effect at low exposure but adverse effects at higher exposures. The differences between the concentrations tested in the laboratory and those to which wildlife might be exposed must be considered to fully assess the significance of the information. In many cases, the concentrations tested in the laboratory animals were considerably higher than those that wildlife might be exposed to in the environment.
- The bioaccessibility or bioavailability (i.e., chemical form) in which the compound is introduced to the test organism is designed to maximize uptake into the blood stream. Bioaccessibility is maximized in the lab to maximize toxic effects. The uptake of the highly bioaccessible form often results in very elevated exposures compared to uptake in the environment, where chemicals are often much less bioaccessible for a variety of physical and chemical reasons.

In the SLWRA, both acute and chronic inhalation TRVs were identified for the COPCs when sufficient toxicity data were available.

Acute Inhalation Toxicity Reference Values

Very little acute toxicity information for wildlife species is available. In lieu of this information, acute lethal concentrations (LC_{50}) under laboratory conditions were assessed herein. The LC_{50} is the concentration that is associated with lethality in 50% of the test animals. The acute inhalation TRVs were derived based on the lowest LC_{50} value reported in the literature. The LC_{LO} refers to the 'lowest published lethal concentration' (NIOSH 2007). Use of the lowest values reduces the likelihood that potential risks are understated.

Since the lowest value reported for all species was used to derive the acute TRV, no uncertainty factors were applied to account for possible differences in sensitivity between species. All mammalian wildlife receptors were evaluated under one acute TRV identified based on the lowest LC_{50} value for all mammalian laboratory animals. Similarly, all avian wildlife receptors were evaluated under one acute TRV identified based on the lowest LC_{50} value reported for all bird species.

The literature review for acute TRVs consisted of an online search of the following:

- International Programme on Chemical Safety (IPCS).
- National Toxicity Program Chemical Repository (NTP).
- National Library of Medicine's Hazardous Substances Data Bank (HSDB).
- National Library of Medicine's ChemIDplus.
- Agency for Toxic Substances and Disease Registry (ATSDR).

Table 2-3 provides the acute TRVs for each of the COPCs in the 2013 SLWRA.

Table 2-3 Acute Inhalation Toxicity Reference Values used in the Screening Level Wildlife Risk Assessment

Chemical Of Potential Concern	Receptor	Averaging Period	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Criteria Air Contaminants						
Carbon monoxide	Avian	1-hour	1,500	Lethality	An LC50 of 1,334 ppm (1,500 mg/m³) was identified in wild birds.	ChemIDplus 2013
	Mammal	1-hour	2,078	Lethality	An LC50 of 2,078 mg/m³ was identified in rats exposed via inhalation to carbon monoxide for 4 hours.	Ramamoorthy et al. 1995
Nitrogen dioxide	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	56	Lethality	An LC50 of 56 mg/m³ was identified in guinea pigs exposed via inhalation to nitrogen dioxide for 1 hour.	HSDB 2013
Sulphur dioxide	Avian	1-hour	2,600	Lethality	An LC20 of 1,000 ppm (2,600 mg/m³) was identified in white leghorn poultry continuously exposed to sulphur dioxide vapours of 0 to 5,000 ppm for 1 hour.	Fedde and Kuhlmann 1979
	Mammal	1-hour	2,600	Lethality	An LC50 of 2,600 mg/m³ was identified in mice exposed via inhalation to sulphur dioxide for 4 hours.	HSDB 2013
Metals						
Aluminum	Avian	ND	ND	ND	ND	ND
	Mammal	24-hours	5.5	Growth	A LOAEL of 33 mg/m³ was identified in Golden Syrian hamsters exposed to aluminum via inhalation for 4-6 hours/day for 3 days. This value was adjusted for continuous exposure.	ATSDR 2008a
Arsenic	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	100	Lethality	An LCLo of 100 mg/m^3 was identified in cats exposed via inhalation to arsenic trichloride for 1 hour.	NIOSH 1996
Barium	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Beryllium	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	0.15	Lethality	An LC50 of 0.15 mg/m^3 was identified in rats exposed via inhalation to beryllium for 4 hours.	IPCS 2001
Cadmium	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	28.4	Lethality	An LC50 of 28.4 mg/m^3 was identified in rabbits exposed via inhalation to cadmium metal dust for 4 hours.	ATSDR 2008b
Chromium	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	31.5	Lethality	An LC50 of 31.5 mg/m^3 was identified in mice exposed via inhalation to chromium (III) chloride anhydrous for 2 hours.	ChemIDplus 2013

Table 2-3 Acute Inhalation Toxicity Reference Values used in the Screening Level Wildlife Risk Assessment (continued)

Chemical Of Potential Concern	Receptor	Averaging Period	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Chromium VI	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	29	Lethality	An LC_{50} of 29 mg/m³ was identified in rats exposed to potassium dichromate via inhalation for 4 hours.	ATSDR 2008c
Cobalt	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	165	Lethality	An LC50 of 165 mg/m^3 was identified in rats exposed via inhalation to cobalt hydrocarbonyl for 30 minutes.	IPCS 2006
Copper	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	1,303	Lethality	An LC50 greater than 1,303 mg/m^3 was identified in rabbits exposed via inhalation to copper (II) hydroxide (duration unknown).	IPCS 1998
Lead	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Mercury	Avian	ND	ND	ND	ND	ND
	Mammal	24-hour	29	Lethality	An LCLo of 29 mg/m^3 was identified in rabbits exposed via inhalation to mercury for 30 hours.	ChemIDplus 2013
Manganese	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	61	Reproductive	A NOAEL of 61 mg/m³ was identified in mice exposed via inhalation to manganese dioxide for 7 hours per day, 5 days per week for 18 weeks.	ATSDR 2012
Molybdenum	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	5,840	Lethality	An LC50 of 5,840 mg/m^3 was identified in rats exposed via inhalation to molybdenum trioxide for 4 hours.	ChemIDplus 2013
Nickel	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	67	Lethality	An LC50 of 67 mg/m^3 was identified in mice exposed via inhalation to nickel carbonyl for 30 minutes.	HSDB 2013
Selenium	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	33	Lethality	An LCLo of 33 mg/m^3 was identified in rats exposed via inhalation to selenium for 8 hours.	ChemIDplus 2013
Silver	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Strontium	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Vanadium	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	70	Lethality	An LC50 of 70 mg/m^3 was identified in rats exposed via inhalation to vanadium pentoxide fume for 1 hour.	HSDB 2013
Zinc	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	1,975	Lethality	An LC50 of 1,975 mg/m^3 was identified in rats exposed via inhalation to zinc chloride for 10 minutes.	HSDB 2013

Table 2-3 Acute Inhalation Toxicity Reference Values used in the Screening Level Wildlife Risk Assessment (continued)

Chemical Of Potential Concern	Receptor	Averaging Period	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Organic Compounds and Polycyclic Aromatic Hydrocarbons						
1-Pentene	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
1,3 Butadiene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	268,000	Lethality	An LC50 of 268,000mg/m³ was provided for mice exposed via inhalation to 1,3-butadiene for 2 hours.	ATSDR 2009
Acetaldehyde	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	2,700	Lethality	An LC50 of 1,500ppm (2702.5 mg/m³) was identified for mice exposed via inhalation to acetaldehyde for 4 hours.	HSDB 2013
Acetone	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	44,000	Lethality	An LC50 of 44,000 mg/m³ was identified for mice exposed via inhalation to acetone for 4 hours.	ChemIDplus 2013
Acrolein	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	17	Lethality	An LC50 of 17 mg/m³ was identified in rats exposed via inhalation to acrolein for 4 hours.	HSDB 2013
Aliphatic aldehydes	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	200	Lethality	An LC50 of 200 mg/m^3 was identified in rats exposed via inhalation to crotonaldehyde for 2 hours.	HSDB 2013 ChemIDplus 2013
Aliphatic C_2-C_4 group	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	658,000	Lethality	An LC50 of 658 g/m^3 (658,000 mg/m^3) was identified in rats exposed via inhalation to butane for 4 hours.	HSDB 2013 ChemIDPlus 2013
Aliphatic C_5-C_8 group	Avian	1-hour	3,500	Growth	LOAEL of 3,500 mg/m³ in Leghorn hens exposed for 30 days continuously to n-hexane vapours.	TPHCWG 1997
	Mammal	24-hour	2,500	Maternal toxicity	A NOAEL of 10,000 mg/m³ was identified in rats and mice exposed via inhalation to commercial hexane for 6 hours/day on days 6-15 of gestation. Due to endpoint, value was adjusted for continuous exposure.	TPHCWG 1997
Aliphatic C_9-C_{16} group	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Aliphatic C_{17}-C_{34} group	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Aromatic C_9-C_{16} group	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	500	Growth, reproduction	A NOAEL of 500 mg/m³ was identified in mice exposed via inhalation to high flash aromatic naphtha for 6 hours/day on gestational days 6-15.	TPHCWG 1997 MA DEP 2003
Benzene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	15,000	Lethality	An LC50 of 15,000 mg/m³ was identified in mice	IPCS 1993

Table 2-3 Acute Inhalation Toxicity Reference Values used in the Screening Level Wildlife Risk Assessment (continued)

Chemical Of Potential Concern	Receptor	Averaging Period	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
					exposed via inhalation to benzene for 8 hours.	
Carcinogenic PAHs Group 1	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Carcinogenic PAHs Group 2	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Carcinogenic PAHs Group 3	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Cyclohexane	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	70,000	Lethality	An LC50 of 70,000 mg/m³ was identified in mammals (species unspecified) exposed via inhalation to cyclohexane for 1 hour.	ChemIDplus 2013
Dichlorobenzene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	12,000	Lethality	An LC50 of 12,000 mg/m³ was identified in mammals (species unidentified) exposed via inhalation to 1,4-dichlorobenzene (exposure duration unknown).	ChemIDplus 2013
Ethylacetylene	Avian	ND	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND	ND
Ethylbenzene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	17,200	Lethality	An LC50 of 17,200 mg/m³ was identified in rats exposed via inhalation to ethylbenzene for 4 hours.	IPCS 1996
Ethylene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	1,089,877	Lethality	An LC_{LO} of 1,089,877 mg/m³ (950,000 ppm) was identified in mammals (species unspecified) exposed via inhalation to ethylene (exposure duration unknown).	ChemIDplus 2013
Formaldehyde	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	414	Lethality	An LC50 of 414 mg/m³ was identified in mice exposed via inhalation to formaldehyde for 4 hours.	CICAD 2002
Hexane	Avian	1-hour	3,500	Growth	A LOAEL of 3,500 mg/m³ was identified in Leghorn hens exposed continuously to n-hexane vapours for 30 days.	Abou-Donia et al. 1991
	Mammal	1-hour	169,000	Lethality	An LC50 of 48,000 ppm (169,000 mg/m³) was identified in mice and rats exposed via inhalation to hexane for 4 hours.	HSDB 2013
Isopropylbenzene (cumene)	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	9,800	Lethality	An LC50 of 2,000 ppm (9,800 mg/m³) was identified in mice exposed via inhalation to cumene for 7 hours.	WHO 1999 HSDB 2013 ChemIDplus 2013

Table 2-3 Acute Inhalation Toxicity Reference Values used in the Screening Level Wildlife Risk Assessment (continued)

Chemical Of Potential Concern	Receptor	Averaging Period	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Methyl ethyl ketone group	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	23,500	Lethality	An LC50 of 23,500 mg/m^3 was identified in rats exposed to methyl ethyl ketone for 8 hours.	ChemIDPlus 2013
Naphthalene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	340	Lethality	An LC50 of 340 mg/m³ was identified in rats exposed via inhalation to naphthalene for 1 hour. The LC50 for naphthalene is more conservative than the TRV used for the aromatic C_9-C_{16} group, thus the naphthalene group was assessed both individually and as part of the aromatic C_9-C_{16} group.	ChemIDplus 2013
Propylene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	658,000	Lethality	An LC50 of 658 mg/L (658,000 mg/m³) was identified in rats exposed via inhalation to propylene for 4 hours.	HSDB 2013
Propylene Oxide	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	4,100	Lethality	An LC50 of 1,740 ppm (4,100 mg/m^3) was identified in mice exposed via inhalation to propylene oxide for 4 hours.	HSDB 2013
Toluene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	100,000	Lethality	An LC50 of 100,000 mg/m³ was identified in rats exposed via inhalation to toluene for 1 hour.	HSDB 2013
Trimethylbenzene	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	24,000	Lethality	An LC50 of 24,000 mg/m^3 was identified in rats exposed via inhalation to 1,3,5-trimethylbenzene for 4 hours.	ChemIDplus 2013
Xylenes	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	17,000	Lethality	An LC50 of 17,000 mg/m³ was identified in mice exposed via inhalation to xylenes for 6 hours.	HSDB 2013
Sulphur Compounds						
Carbon disulphide	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	690	Lethality	An LC50 of 690 mg/m³ was identified in mice exposed via inhalation to carbon disulphide for 1 hour.	IPCS 2002
Hydrogen sulphide	Avian	ND	ND	ND	ND	ND
	Mammal	1-hour	820	Lethality	An LC50 of 820 mg/m³ was identified in mice exposed via inhalation to hydrogen sulphide for 2 hours.	ATSDR 2006

Notes: ND = No appropriate or relevant data were available.

Chronic Toxicity Reference Values

There is limited standardized guidance on the derivation of chronic wildlife TRVs available in the form of regulatory guidelines, directives, or protocols. In 1998, the British Columbia Ministry of Water, Land and Air Protection (BC MWLAP 1998) recommended an approach for the extrapolation of toxicity data between mammalian species based on an effective concentration (EC_{20}) or concentration that affects 20% of the exposed (i.e., test) organisms. Based on BC MWLAP (1998), the goal of a screening level ecological risk assessment is "not to protect each individual organism from a toxic effect, but rather to protect enough individuals so that a viable population and community of organisms can be maintained." Therefore, consideration of endpoints having an effect on a species' population was more appropriate than endpoints affecting individuals within a population. As such, the BC MWLAP (1998) gave preference to reproductive endpoints, but lethality, growth and developmental effects were considered to be acceptable if these were the only endpoints available for selection of a TRV. According to the BC MWLAP (1998), an uncertainty factor of 10 should be applied to the EC_{20} to account for interspecies differences. If an EC_{20} is not available, then the BC MWLAP (1998) recommends that a concentration curve be generated from the available toxicity data. Otherwise, the use of a Lowest Observed Adverse Effect Level (LOAEL) is recommended without any application of uncertainty factors.

A summary of the BC MWLAP (1998) recommendations for ecological risk assessments follows:

- Use an EC_{20} as a TRV.
- If an EC_{20} is not available or cannot be calculated, use the LOAEL from the most applicable study.
- If the data are from similar species do not use uncertainty factors.
- If the animals are not closely related or if it is unknown whether or not they are likely to have similar physiological responses, apply an uncertainty factor of 10.

The US EPA OSW (1999) provides guidance for deriving chronic TRVs using no-observed-adverse-effect levels (NOAELs) based on population-level effects for chronic exposure, such as development, reproduction and survivorship, whereas the CCME (2006) recommends using a LOAEL and applying an uncertainty factor of 1 to 5, based on professional judgment, for extrapolation between wildlife species.

For the current assessment, EC_{20} values were not available for any of the COPCs. For the chronic inhalation TRVs, reliance was placed on NOAELs as opposed to LOAELs to reduce the likelihood of the underestimation of potential risks to sensitive wildlife species. The lowest reported NOAEL value for all species associated with population-level effects was selected. Due to the similarity in respiratory physiology between different species, no adjustments were required to be made to the NOAEL for the individual wildlife receptors. The lowest NOAEL identified for mammalian laboratory animals was used to evaluate potential risks to all the mammalian wildlife receptors and the lowest NOAEL for birds was used to evaluate potential risks to all the avian wildlife receptors.

For many of the COPCs, a TRV was derived for this assessment from the available toxicological data. If a NOAEL was not available, the lowest LOAEL was used as the TRV with a 10-fold uncertainty factor applied to account for the extrapolation of a LOAEL to derive a NOAEL. The TRVs were based on ecologically population-level relevant endpoints (i.e., growth, reproduction, and survivorship).

The literature review for NOAEL values (or LOAEL values in the event that NOAELs were not available), consisted of an online search of the following:

- Agency for Toxic Substances and Disease Registry (ATSDR).
- American Conference of Governmental Industrial Hygienists (ACGIH).
- California Office of Environmental Health Hazard Assessment (OEHHA).
- Health Canada and Environment Canada (Government of Canada).
- International Programme on Chemical Safety (IPCS).
- National Toxicology Program Chemical Repository (NTP).
- Netherlands National Institute of Public Health and the Environment (RIVM).
- Ontario Ministry of the Environment (OMOE).
- National Library of Medicine's Hazardous Substances Data Bank (HSDB).
- National Library of Medicine's Toxicology Literature Online (TOXLINE).
- World Health Organization (WHO).
- United States Environmental Protection Agency (US EPA).

A summary of the TRVs used to evaluate potential wildlife health risks associated with chronic inhalation exposure to COPCs are provided in Table 2-4.

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Criteria Air Contaminants					
Nitrogen dioxide	Avian	ND	ND	ND	ND
	Mammal	0.025	Developmental effects	A NOAEL of 0.10 mg/m³ was identified in rats exposed to 0, 0.05, 0.10, 1.0 or 10 mg/m³ nitrogen dioxide for 6 hours/day, 7 days/week, through gestation until the offspring were 2 months old. The NOAEL was adjusted to continuous exposure.	Tabacova et al. 1985
Sulphur dioxide	Avian	ND	ND	ND	ND
	Mammal	2.6	Respiratory effects	A NOAEL of 2.6 mg/m³ was identified in guinea pigs exposed continuously to an average sulphur dioxide concentration of 0.34, 2.6 or 15 mg/m³ for 52 weeks.	HSDB 2013
Metals					
Aluminum	Avian	ND	ND	ND	ND
	Mammal	0.11	Growth	A NOAEL of 0.65 mg/m³ was identified in F344 rats exposed to aluminum via inhalation for 6 hours/day, 5 days/week for 12 to 24 months. This value was adjusted for continuous exposure.	ATSDR 2008a
Arsenic	Avian	ND	ND	ND	ND
	Mammal	2	Developmental/ reproductive effects	A NOAEL of 8 mg/m³ was identified in rats exposed to 0.2 to mg/m³ arsenic (as arsenic trioxide) for 6 hours/day from 14 days prior to mating through gestation day 19. The NOAEL was adjusted to continuous exposure.	ATSDR 2007a
Barium	Avian	ND	ND	ND	ND
	Mammal	0.11	Growth effects	A NOAEL of 0.8 mg/m³ was identified in rats exposed to 0.8 or 3.6 mg/m³ barium (as barium carbonate dust) for 4 hours/day, 6 days/week for 4 months. The NOAEL was adjusted to continuous exposure.	WHO 2001a; RIVM 2001; US EPA 1998
Beryllium	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Cadmium	Avian	ND	ND	ND	ND
	Mammal	0.00030	Developmental effects	A LOAEL of 0.02 mg/m³ was identified in rats exposed to cadmium (as cadmium oxide) for 5 hours/day, 5 days/week for 5 months prior to mating, during mating and the first 20 days of gestation. The LOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied for use of a LOAEL.	ATSDR 2008b Government of Canada 1994

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Chromium	Avian	ND	ND	ND	ND
	Mammal	0.092	Reproductive and growth effects	A NOAEL of 0.1 mg/m³ was identified in rats exposed to a 3:2 mixture of chromium(VI) trioxide and chromium(III) oxide for 22 hours/day, 7 days/week for 18 months. The NOAEL was adjusted to continuous exposure.	ATSDR 2008c
Chromium VI	Avian	ND	ND	ND	ND
	Mammal	0.09	Growth	A NOAEL of 0.1 mg/m³ was identified for reduced body weights in male Wistar rats exposed via inhalation to hexavalent chromium for 22 hours/day, 7 days/week for about 72 weeks. The NOAEL was adjusted for continuous exposure.	ATSDR 2008c
Cobalt	Avian	ND	ND	ND	ND
	Mammal	0.0020	Reproductive effects	A LOAEL of 1.14 mg/m^3 was identified in mice exposed to 0, 1.14, 3.80 or 11.38 mg/m^3 cobalt (as cobalt sulphate heptahydrate) for 6 hours per day, 5 days per week for 13 weeks. The LOAEL was adjusted to account to continuous exposure. An uncertainty factor of 10 was applied for use of a LOAEL and an uncertainty factor of 10 was applied for use of a subchronic study. A similar study exposed mice to 0, 0.11, 0.38, 1.14 or 3.8 mg/m^3 for 6 hours/day, 5 days/week for 105 weeks. Growth effects were observed at all exposures. Adjustment of the lowest exposure (i.e., 0.11 mg/m^3) to continuous exposure and application of an uncertainty factor of 10 for lack of a NOAEL also results in a TRV of 0.0020 mg/m^3.	ATSDR 2004 WHO 2006
Copper	Avian	ND	ND	ND	ND
	Mammal	0.25	Respiratory effects	A LOAEL of 2.5 mg/m^3 was identified in rats exposed to 2.5 or 19.6 mg/m^3 copper (as copper chloride) for 4 months. An uncertainty factor of 10 was applied to account for use of a LOAEL.	WHO 1998a
Lead	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Manganese	Avian	ND	ND	ND	ND
	Mammal	0.5	Growth effects	A NOAEL of 0.5 mg/m³ was identified for decreased fetal brain weights exposed to 0, 0.05, 0.5 or 1 mg/m³ of manganese (as manganese sulphate) for 6 hours/day, 7 days/week during breeding, for up to 14 days during the mating period, during gestation and up to post-natal day 18.	OEHHA 2008
Mercury	Avian	ND	ND	ND	ND
	Mammal	0.05	Developmental effects	A LOAEL of 0.05 mg/m^3 was identified in offspring of Sprague Dawley rats exposed to inorganic mercury via inhalation for 1 to 4 hours/day, 7 days/week during post-partum days 11 to 17. Not adjusted for continuous exposure due to the nature of the endpoint.	ATSDR 1999b

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Molybdenum	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Nickel	Avian	ND	ND	ND	ND
	Mammal	0.020	Growth effects	A NOAEL of 0.11 mg/m³ was identified in rats exposed to 0, 0.11 or 0.73 mg/m³ nickel (as nickel subsulphide) for 6 hours/day, 5 days/week for 104 weeks. The NOAEL was adjusted to continuous exposure.	ATSDR 2005b; OEHHA 2000
Selenium	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Silver	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Strontium	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Vanadium	Avian	ND	ND	ND	ND
	Mammal	0.0089	Respiratory effects	A NOAEL of 0.5 mg/m³ was identified in rats and mice exposed to vanadium pentoxide for 6 hours/day, 5 days/week for 13 weeks. The NOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied to account for subchronic exposure.	WHO 2001b
Zinc	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Organic Compounds and Polycyclic Aromatic Hydrocarbons					
Aliphatic aldehydes	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Aliphatic C_2-C_4 group	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Aliphatic C_5-C_8 group	Avian	35	Growth effects	A LOAEL of 3,500 mg/m^3 was identified in Leghorn hens exposed continuously to n-hexane vapours for 30 days. An uncertainty factor of 100 was applied to account for use of a subchronic study and a LOAEL.	Abou-Donia et al. 1991
	Mammal	1,840	Reproductive effects	A NOAEL of 3000 ppm (10,307 mg/m^3) was identified in rats exposed to 0, 900, 3000, or 9000 ppm commercial hexane for 6 hours/day, 5 days/week for 2 generations. The NOAEL was adjusted to continuous exposure.	TPHCWG 1997
Aliphatic C_9-C_{16} group	Avian	ND	ND	ND	ND
	Mammal	35	Growth effects	A NOAEL of 300 ppm (1,970 mg/m^3) was identified in rats exposed via inhalation to dearomatized white spirit vapours for 6 hours/day, 5 days/week for 12 weeks. The NOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied to account for use of a subchronic study.	MA DEP 2003

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Aliphatic C_{17}-C_{34} group	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Aromatic C_9-C_{16} group	Avian	ND	ND	ND	ND
	Mammal	123	Developmental/ reproductive effects	A NOAEL of 100 ppm (491 mg/m^3) was identified in mice exposed to 0, 100, 500 or 1,500 ppm high flash aromatic naphtha for 6 hours/day on gestation days 6-15. NOAEL is based upon the incidence of maternal and fetal effects. The NOAEL was adjusted to continuous exposure.	MA DEP 2003
1-Pentene	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
1,3-Butadiene	Avian	ND	ND	ND	ND
	Mammal	0.25	Reproductive	A LOAEL of 6.25 ppm (14 mg/m³) was identified in female mice exposed to 0, 6.25, 20, 62.5, 200 or 625 ppm of 1,3-butadiene for a duration of 6 hours/day, 5 days/week for 103-weeks. This value was adjusted for continuous exposure, and an uncertainty factor of 10 was applied to account for the use of a LOAEL.	ATSDR 2009
Acetaldehyde	Avian	ND	ND	ND	ND
	Mammal	13	Growth effects	A NOAEL of 400 ppm (720 mg/m³) was identified in rats exposed to 400, 1000, 2200, or 5000 ppm acetaldehyde for 6 hours/day, 5 days/week for 4 weeks. The NOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied for use of a subchronic study.	Government of Canada 2000a
Acetone	Avian	ND	ND	ND	ND
	Mammal	1,300	Developmental and growth effects	A NOAEL of 2,200 ppm (5,200 mg/m³) was identified in pregnant rats and mice. Rats were exposed to 0, 440, 2200, or 11,000 ppm acetone 6 hours/day, 7 days/week for 14 days during days 6-9 of gestation. Mice were exposed 0, 440, 2200, or 11,000 ppm acetone 6 hours/day, 7 days/week for 12 days during days 6-17 of gestation. NOAEL is based upon reduced maternal body weight, uterine weight, and decreased fetal weight for rats and decreased fetal weight for mice. The NOAEL was adjusted to continuous exposure.	ACGIH 1996 ATSDR 1994 WHO 1998b
Acrolein	Avian	ND	ND	ND	ND
	Mammal	0.16	Growth effects	A NOAEL of 0.4 ppm (0.9 mg/m³) was identified in rats exposed to 0, 0.4, 1.4, or 4.9 ppm acrolein for 6 hours/day, 5 days/week for 13 weeks. The NOAEL was adjusted to continuous exposure.	US EPA 2003a

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Benzene	Avian	ND	ND	ND	ND
	Mammal	15	Developmental effects	A LOAEL of 47 ppm (150 mg/m³) was identified in rats exposed to 0, 47, 141, 470 or 939 ppm benzene for 24 hours/day on gestation days 7-14. An uncertainty factor of 10 was applied for use of a LOAEL.	Government of Canada 1993
Carcinogenic PAH group 1	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Carcinogenic PAH group 2	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Carcinogenic PAH group 3	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Cyclohexane	Avian	ND	ND	ND	ND
	Mammal	1,230	Developmental effects	A NOAEL of 2,000 ppm (6,886 mg/m³) was identified in rats exposed to 0, 500, 2000, or 7000 ppm cyclohexane vapours for 6 hours per day, 5 days per week, for 10 weeks prior to mating. Female rats were then exposed daily following breeding and during pregnancy and lactation, with the exception of gestation day 21 to lactation day 4. The NOAEL was adjusted to continuous exposure.	US EPA 2003b
Dichlorobenzene	Avian	ND	ND	ND	ND
	Mammal	7.5	Growth effects	A NOAEL of 50 ppm (300 mg/m³) was identified in rats exposed to 0, 50, 150 or 450 ppm 1,4-dichlorobenzene for 6 hours/day, 7 days/week, for 10 or 11 weeks. The NOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied for use of a subchronic study.	US EPA 1996
Ethylacetylene	Avian	ND	ND	ND	ND
	Mammal	ND	ND	ND	ND
Ethylbenzene	Avian	ND	ND	ND	ND
	Mammal	110	Developmental effects	A NOAEL of 100 ppm (434 mg/m³) was identified in New Zealand white rabbits exposed to 0, 100 or 1,000 ppm ethylbenzene for 6-7 hours per day, 7 days per week on gestation days 1-24. The NOAEL was adjusted to continuous exposure.	US EPA 1991a
Ethylene	Avian	ND	ND	ND	ND
	Mammal	1,434	Developmental/ Reproductive effects	A NOAEL of 5,000 ppm (5,736 mg/m³) was identified in rats head-only exposed to up to 5,000 ppm ethylene for 6 hours/day prior to mating for 2 weeks, during mating and for females until gestational day 20. The NOAEL was adjusted to continuous exposure.	ACGIH 2005

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Formaldehyde	Avian	ND	ND	ND	ND
	Mammal	0.45	Survivorship and growth effects	A NOAEL of 2 ppm (2.5 mg/m³) was identified in rats exposed to 0, 2, 5.6 or 14.3 ppm formaldehyde for 6 hours/day, 5 days/week for 24 months. The NOAEL was adjusted to continuous exposure.	US EPA 1991b
Naphthalene	Avian	ND	ND	ND	ND
	Mammal	9.4	Growth effects	A NOAEL of 10 ppm (52.4 mg/m³) was identified in rats exposed to 0, 10, 30 or 60 ppm naphthalene vapours for 6 hours/day, 5 days/week for 2 years. The NOAEL was adjusted to continuous exposure.	ATSDR 2005a
Isopropylbenzene (cumene)	Avian	ND	ND	ND	ND
	Mammal	121	Reproductive/ developmental effects	A NOAEL of 485 mg/m³ was identified in rats exposed to cumene for 6 hours/day on gestation days 6-15. The NOAEL was adjusted to continuous exposure.	WHO 1999
Methyl ethyl ketone group	Avian	ND	ND	ND	ND
	Mammal	860	Reproduction	A NOAEL of 1000 ppm (2950 mg/m^3) was identified for MEK from a study where pregnant female Swiss mice were exposed for 7-hours/day during gestational days 6-15. Decreased mean fetal body weights were observed at 3000 mg/m^3. The NOAEL was adjusted for continuous exposure.	US EPA 2003c
Hexane	Avian	35	Growth effects	A LOAEL of 3,500 mg/m³ was identified in leghorn hens exposed continuously to n-hexane vapours for 30 days. An uncertainty factor of 100 was applied to account for use of a subchronic study and a LOAEL.	Abou-Donia et al. 1991
	Mammal	580	Developmental effects	A NOAEL of 200 ppm (700 mg/m³) was identified in rats exposed to 0, 200, 1,000 or 5,000 ppm hexane vapours for 20 hours/day on days 6-19 of gestation. The NOAEL was adjusted to continuous exposure.	ATSDR 1999a US EPA 2005b
Propylene	Avian	ND	ND	ND	ND
	Mammal	4,300	Developmental/ reproductive effects	A NOAEL of 10,000 ppm (17,200 mg/m³) was identified in female Wistar rats exposed to 200, 1000, or 10,000 ppm propylene for 6 hours/day from days 6-19 postcoitum (14 exposures). The NOAEL was adjusted to continuous exposure.	ACGIH 2006
Propylene oxide	Avian	ND	ND	ND	ND
	Mammal	43	Growth effects	A NOAEL of 100 ppm (238 mg/m³) was identified in Fischer 344 rats exposed to 0, 30, 100, or 300 ppm propylene oxide for 6 hours/day, 5 days/week for 14 weeks before mating to produce the F1 litters. The NOAEL was adjusted to continuous exposure.	US EPA 1991c

Table 2-4 Chronic Inhalation Toxicity Reference Values used in the Screening Level Wildlife Health Risk Assessment (continued)

Chemical of Potential Concern	Receptor	Toxicity Reference Value [mg/m³]	Endpoint	Rationale	Reference
Toluene	Avian	ND	ND	ND	ND
	Mammal	7.3	Reproductive effects	A LOAEL of 100 ppm (375 mg/m³) was identified in mice exposed to toluene vapours for 6.5 hours/day, 5 days/week for 14 weeks. The LOAEL was adjusted to continuous exposure. An uncertainty factor of 10 was applied for use of a LOAEL.	Government of Canada 1992 ATSDR 2000
Trimethylbenzene	Avian	ND	ND	ND	ND
	Mammal	370		A NOAEL of 300 ppm (1,479 mg/m^3) was identified in rats exposed to 0, 100, 300, 600 or 900 ppm 1,2,4-trimethylbenzene vapour and 0, 100, 300, 600 or 1,200 ppm 1,3,5-trimethylbenzene vapour for 6 hours per day, on days 6-20 of gestation. The NOAEL was adjusted for continuous exposure.	OMOE 2006
Xylenes	Avian	ND	ND	ND	ND
	Mammal	15	Developmental effects	A LOAEL of 150 mg/m³ was identified in rats exposed continuously to xylenes on gestation days 7-14. An uncertainty factor of 10 was applied for use of a LOAEL.	ATSDR 2007b
Sulphur Compounds					
Carbon disulphide	Avian	ND	ND	ND	ND
	Mammal	26	Developmental effects	A NOAEL of 40 ppm (125 mg/m³) was identified in rats and rabbits exposed to 0, 20 or 40 ppm carbon disulphide for 7 hours/day, 5 days/week during pre-gestational and/or gestational periods. The NOAEL was adjusted to continuous exposure.	ATSDR 1996 Government of Canada 2000b US EPA 1995
Hydrogen sulphide	Avian	ND	ND	ND	ND
	Mammal	0.76	Growth effects	A NOAEL of 30.5 ppm (42.5 mg/m³) was identified in rats and mice exposed to 0, 10.1, 30.5 or 80 ppm hydrogen sulphide for 6 hours/day, 5 days/week for 90 days. The NOAEL was adjusted to continuous exposure and for the use of subchronic data.	US EPA 2003d ATSDR 2006

Notes: ND = No appropriate or relevant data were available.

2.3.2 Soil Quality Guidelines, Soil Standards and Ecological Soil Screening Levels for Wildlife

As described in Section 2.1.4, the SLWRA relied on soil quality benchmarks or guidelines from three different regulatory agencies to characterize the potential non-inhalation risks posed to terrestrial wildlife.

The Alberta ESRD SQGs used in the 2013 SLWRA were developed to be protective of wildlife for soil and food ingestion for the most stringent land use designation (i.e., agricultural or natural land use) (ESRD 2010). The soil quality guidelines developed by ESRD (ESRD 2010) were calculated using models consistent with those developed for CCME (2006) protocols.

The OMOE soil standards selected for the 2013 SLWRA were developed to be protective of wildlife for soil and food ingestion and for the most stringent land use (i.e., residential or natural land use) (OMOE 2011). The standards developed by OMOE (OMOE 2011) were calculated using models consistent with those applied in various jurisdictions (i.e., US EPA and CCME) and presented in the relevant scientific literature (Sample and Suter 1994; Sample et al 1996).

An US EPA Eco-SSL refers to the concentration of a contaminant in soil that is considered protective of ecological receptors that come in contact with and/or consume biota that live in or on the soil (US EPA 2005a). The US EPA uses a two-step approach to derive the Eco-SSLs. In the first step, TRVs were developed for a mammalian and avian receptor. In deriving the TRVs for the Eco-SSLs, the US EPA used a 'weight-of-evidence' approach and conducted comprehensive literature reviews of available toxicity data for avian or mammalian species. NOAELs or LOAELs for reproduction, growth or survival were identified, as these endpoints are the most relevant to wildlife species. In developing the mammal and avian TRVs, the US EPA OSW (1999) gave preference to the lowest chronic or subchronic NOAEL, followed by chronic or subchronic LOAEL. If neither was available, then acute median lethality point estimates or single dose toxicity values were used. In the second step of the Eco-SSL approach, the US EPA back-calculated the Eco-SSLs (soil concentrations) for three surrogate mammalian or avian species based on the TRV derived in the first step and a wildlife exposure model. For the SLWRA, the lowest Eco-SSL provided for the three surrogate species was selected.

Table 2-5 summarizes the Soil Quality Guidelines used for the COPCs in the soil quality assessment.

Table 2-5 Soil Quality Guidelines Protective of Wildlife for Chemicals of Potential Concern

Chemical Of Potential Concern	Soil Quality Guidelines for Wildlife (mg/kg)				
	ESRD SQG[1]	OMOE Standards[2]		US EPA Eco-SSL[3]	
	Wildlife	Avian	Mammalian	Avian	Mammalian
Metals and Metalloids					
Aluminum	ND	ND	ND	ND	ND
Antimony	ND	ND	25	ND	0.27
Arsenic	380	333	51	43	46
Barium	ND	672	394	ND	2,000
Beryllium	ND	13	ND	ND	21
Cadmium	3.8	1.9	2.4	0.8	0.36
Chromium	ND	161	3,000	26	34
Chromium VI	ND	ND	914	ND	130
Cobalt	ND	180	239	120	230
Copper	300	3,060	283	28	49
Lead	70	32	1,760	11	56
Manganese	ND	ND	ND	4,300	4,000
Mercury	ND	20	32	ND	ND
Methyl mercury	ND	0.034	0.11	ND	ND
Molybdenum	ND	74	6.9	ND	ND
Nickel	355	5,430	5,010	210	130
Selenium	4.5	5.5	2.4	1.2	0.6
Silver	ND	ND	ND	4.2	14
Strontium	ND	ND	ND	ND	ND
Uranium	33	ND	33	ND	ND
Vanadium	ND	18	108	7.8	280
Zinc	640	337	4,200	46	79
Organic Compounds and Polycyclic Aromatic Hydrocarbons					
Acetaldehyde	ND	ND	ND	ND	ND
Acetone	ND	ND	32	ND	ND
Acrolein	ND	ND	ND	ND	ND
Aliphatic aldehydes	ND	ND	ND	ND	ND
Acenaphthenes/ Acenaphthylenes	21.5	ND	6,630	ND	ND
Aliphatic C_9-C_{16} group	9,800	ND	ND	ND	ND
Aliphatic C_{17}-C_{34} group	16,000	ND	ND	ND	ND
Aromatic C_9-C_{16} group	9,800	ND	ND	ND	ND
Aromatic C_{17}-C_{34} group	16,000	ND	ND	ND	ND
Anthracenes / phenanthrenes	43	ND	38,000	ND	ND
Biphenyl	ND	ND	ND	ND	ND
Carcinogenic PAH Group 1 (Based on benzo(a)pyrene)	0.6	ND	1,600	ND	ND
Carcinogenic PAH Group 2 (Based on benz(a)anthracene)	6.2	ND	ND	ND	ND
Carcinogenic PAH Group 3 (Based on chrysene)	6.2	ND	ND	ND	ND
Fluoranthene / fluorene	15.4	ND	0.69	ND	ND
Naphthalene	8.8	ND	379	ND	ND
Pyrene	7.7	ND	2,700	ND	ND
Formaldehyde	ND	ND	ND	ND	ND

Table 2-5 Soil Quality Guidelines Protective of Wildlife for Chemicals of Potential Concern (continued)

Chemical Of Potential Concern	Soil Quality Guidelines for Wildlife (mg/kg)				
	ESRD SQG[1]	OMOE Standards[2]		US EPA Eco-SSL[3]	
	Wildlife	Avian	Mammalian	Avian	Mammalian
Methyl ethyl ketone group	ND	ND	5,680	ND	ND
Propylene oxide	ND	ND	ND	ND	ND
HMW PAH group	ND	ND	ND	ND	1.1
LMW PAH group	ND	ND	ND	ND	100
F2 Fraction	9,800[4]	ND	ND	ND	ND
F3 Fraction	16,000[5]	ND	ND	ND	ND

Notes:
(1) Source: ESRD 2010.
(2) Source OMOE 2011.
(3) Source: US EPA 2010.
(4) Value is the PHC F2 fraction SQG.
(5) Value is the PHC F3 fraction SQG
ND = No data. No SQG or Eco-SSL available.
HMW PAH = represent 2 and 3 ring PAHs, based on data presented in US EPA (2007). For the purpose of this assessment, the LMW PAH group includes: acenaphthene/acenaphthylene, anthracene/phenanthrene, biphenyl, fluorene/fluoranthene, and naphthalene.
LMW PAH = represent PAHs with 4 or more rings, based on data presented in US EPA (2007). For the purpose of this assessment, the HMW PAH group includes: Carcinogenic PAHs, fluorene/fluoranthene, and pyrenes.
F2 Fraction = the F2 fraction is comprised of C_{11}-C_{16} aromatic and aliphatic PHCs (CCME 2008), and biphenyl.
F3 Fraction = the F3 fraction is comprised of C_{17}-C_{34} aromatics and aliphatic PHCs (CCME 2008), and the carcinogenic PAH groups.

2.3.3 Surface Water Quality Guidelines for Wildlife

The ESRD SWQGs selected for the 2013 SLWRA were developed to be protective of wildlife or livestock consumption of surface water (ESRD 2010). The SWQGs were calculated from published daily threshold exposure doses and ecological exposure parameters provided by ESRD (2010).

The SWQGs used for the selected chemicals assessed in the water quality component of the 2013 SLWRA are summarized in Table 2-6.

Table 2-7 presents a summary of the COPCs that are assessed in the SLWRA for inhalation and multiple pathway exposures.

Table 2-6 Surface Water Quality Guidelines Protective of Wildlife

Chemical Category	Chemicals of Potential Concern	Surface Water Quality Guideline for Wildlife (mg/L)
Metals and Metalloids	Aluminum	5
	Antimony	—
	Arsenic	0.025
	Barium	—
	Beryllium	—
	Boron	5
	Cadmium	0.08
	Chromium	0.05
	Chromium VI	—
	Cobalt	—
	Copper	0.5
	Lead	0.1
	Lithium	—
	Manganese	—
	Mercury	0.003
	Methyl mercury	—
	Molybdenum	—
	Nickel	1
	Selenium	0.05
	Silver	0.05
	Strontium	—
	Thallium	—
	Uranium	0.2
	Vanadium	—
	Zinc	50
Organics and Polycyclic Aromatic Hydrocarbons	Acetaldehyde	—
	Acetone	—
	Acrolein	—
	Aliphatic aldehydes	—
	Acenaphthenes/ Acenaphthylenes	—
	Aliphatic C_9-C_{16} group	42.6
	Aliphatic C_{17}-C_{34} group	69.0
	Aromatic C_9-C_{16} group	42.6
	Aromatic C_{17}-C_{34} group	69.0
	Ammonia	—
	Anthracenes / phenanthrenes	—
	Biphenyl	—
	Carcinogenic PAH Group 1	—
	Carcinogenic PAH Group 2	—
	Carcinogenic PAH Group 3	—
	Fluoranthene / fluorene	—
	Naphthalene	—
	Pyrene	—
	Formaldehyde	—
	Methyl ethyl ketone group	—
	Phenol	0.002
	Propylene oxide	–
	F2 Fraction	42.6
	F3 Fraction	69.0

NOTES:
— Not Available
F2 Fraction = the F2 fraction is comprised of C_{11}-C_{16} aromatic and aliphatic PHCs (CCME 2008), and biphenyl.
F3 Fraction = the F3 fraction is comprised C_{17}-C_{34} aromatics and aliphatic PHCs (CCME 2008), and the carcinogenic PAH groups.

Table 2-7 Summary of Chemicals of Potential Concern Assessed in the Inhalation and Multiple Pathway Assessments

Chemical	Inhalation Assessment		Multiple Pathway Assessment	
	Acute	Chronic	Soil	Surface Water
Criteria Air Contaminants				
CO	CO [A,M]	–	NA	NA
NO_2	NO_2 [M]	NO_2 [M]	NA	NA
$PM_{2.5}$	–	–	NA	NA
SO_2	SO_2 [A,M]	SO_2 [M]	NA	NA
Organic Compounds				
1,3-Butadiene	1,3-Butadiene [M]	1,3-Butadiene [M]	NA	NA
Acetaldehyde	Acetaldehyde [M]	Acetaldehyde [M]	–	–
Acetone	Acetone [M]	Acetone [M]	Acetone	–
Acrolein	Acrolein [M]	Acrolein [M]	–	–
Aliphatic aldehydes	Aliphatic aldehydes [M]	–	–	–
Aliphatic C_2-C_4	Aliphatic C_2-C_4 group [M]	–	NA	NA
Aliphatic C_5-C_8	Aliphatic C_5-C_8 group [A, M]	Aliphatic C_5-C_8 group [A, M]	NA	NA
Aliphatic C_9-C_{16}	–	Aliphatic C_9-C_{16} group [M]	Aliphatic C_9-C_{16} group F2 Fraction	Aliphatic C_9-C_{16} group F2 Fraction
Aliphatic C_{17}-C_{34}	–	–	Aliphatic C_{17}-C_{34} group F3 Fraction	Aliphatic C_{17}-C_{34} group F3 Fraction
Ammonia	NA	NA	NA	–
Aromatic C_9-C_{16}	Aromatic C_9-C_{16} group [M]	Aromatic C_9-C_{16} group [M]	Aromatic C_9-C_{16} group F2 Fraction	Aromatic C_9-C_{16} group F2 Fraction
Aromatic C_{17}-C_{34}	–	–	Aromatic C_{17}-C_{34} group F3 Fraction	Aromatic C_{17}-C_{34} group F3 Fraction
Benzene	Benzene [M]	Benzene [M]	NA	NA
Cyclohexane	Cyclohexane [M] Aliphatic C_5-C_8 group [A,M]	Cyclohexane [M] Aliphatic C_5-C_8 group [A,M]	NA	NA
Dichlorobenzene	Dichlorobenzenes [M]	Dichlorobenzenes [M]	NA	NA
Ethylbenzene	Ethylbenzene [M]	Ethylbenzene [M]	NA	NA
Ethylene	Ethylene [M]	Ethylene [M]	NA	NA
Formaldehyde	Formaldehyde [M]	Formaldehyde [M]	–	–
Hexane	Hexane [A,M] Aliphatic C_5-C_8 group [A,M]	Hexane [A,M] Aliphatic C_5-C_8 group [A,M]	NA	NA
Isopropylbenzene (cumene)	Isopropylbenzene (cumene) [M]	Isopropylbenzene (cumene) [M]	NA	NA
Methyl ethyl ketone group	Methyl ethyl ketone group [M]	Methyl ethyl ketone group [M]	Methyl ethyl ketone group	–
Phenol	NA	NA	NA	Phenol
Propylene	Propylene [M]	Propylene [M]	NA	NA

Table 2-7 Summary of Chemicals of Potential Concern Assessed in the Inhalation and Multiple Pathway Assessments (continued)

Chemical	Inhalation Assessment		Multiple Pathway Assessment	
	Acute	Chronic	Soil	Surface Water
Propylene oxide	Propylene oxide (M)	Propylene oxide (M)	–	–
Toluene	Toluene (M)	Toluene (M)	NA	NA
Trimethylbenzenes	Aromatic C_9-C_{16} group (M)	Aromatic C_9-C_{16} group (M)	NA	NA
Xylenes	Xylenes (M)	Xylenes (M)	NA	NA
Polycyclic Aromatic Hydrocarbons				
Biphenyl	NA	NA	Aromatic C_9-C_{16} group	Aromatic C_9-C_{16} group
Carcinogenic PAH group 1	–	–	Carcinogenic PAH group 1	–
Carcinogenic PAH group 2	–	–	Carcinogenic PAH group 2	–
Carcinogenic PAH group 3	–	–	Carcinogenic PAH group 3	–
Naphthalene	Naphthalene (M) Aromatic C_9-C_{16} group (M)	Naphthalene (M) Aromatic C_9-C_{16} group (M)	Naphthalene LMW PAH group	–
Pyrene	Aromatic C_9-C_{16} group (M)	Aromatic C_9-C_{16} group (M)	Pyrene F2 Fraction HMW PAH group	F2 Fraction
Metals and minerals				
Aluminum	Aluminum(M)	Aluminum(M)	–	Aluminum
Antimony	NA	NA	Antimony	–
Arsenic	Arsenic(M)	Arsenic(M)	Arsenic	Arsenic
Barium	–	Barium(M)	Barium	–
Beryllium	Beryllium(M)	–	Beryllium	–
Boron	NA	NA	NA	Boron
Cadmium	Cadmium(M)	Cadmium(M)	Cadmium	Cadmium
Chromium	Chromium(M)	Chromium(M)	Chromium	Chromium
Chromium VI	Chromium VI(M)	Chromium VI(M)	Chromium VI	–
Cobalt	Cobalt(M)	Cobalt(M)	Cobalt	–
Copper	Copper(M)	Copper(M)	Copper	Copper
Lead	–	–	Lead	Lead
Lithium	NA	NA	NA	–
Manganese	Manganese(M)	Manganese(M)	Manganese	–
Mercury	Mercury(M)	Mercury(M)	Mercury	Mercury
Methyl mercury	NA	NA	Methyl mercury	–
Molybdenum	Molybdenum(M)	–	Molybdenum	–
Nickel	Nickel(M)	Nickel(M)	Nickel	Nickel
Selenium	Selenium(M)	–	Selenium	Selenium
Silver	–	–	Silver	Silver
Strontium	NA	NA	–	–

Table 2-7 Summary of Chemicals of Potential Concern Assessed in the Inhalation and Multiple Pathway Assessments (continued)

Chemical	Inhalation Assessment		Multiple Pathway Assessment	
	Acute	Chronic	Soil	Surface Water
Thallium	NA	NA	NA	–
Uranium	NA	NA	Uranium	Uranium
Vanadium	Vanadium[(M)]	Vanadium[(M)]	Vanadium	–
Zinc	Zinc[(M)]	–	Zinc	Zinc
Sulphur Compounds				
CS_2 group	Carbon disulphide group [(M)]	Carbon disulphide group [(M)]	NA	NA
H_2S group	Hydrogen sulphide [(M)]	Hydrogen sulphide [(M)]	NA	NA

Notes:

[(1)] In the soil and surface water assessments, fluoranthene was assessed in the LMW PAH group and the HMW PAH group because CCME (2008) considers it as a HMW PAH, but US EPA (2007) classifies it as a LMW PAH. Note that fluoranthene is a 4 ringed PAH but only has 3 benzenoid rings.

[(A)] COPC was assessed for the avian receptor based on an available inhalation TRV or soil guideline from a regulatory agency. See Section 2.2 (Exposure Assessment) for further details.

[(M)] COPC was assessed for the mammalian receptor based on an available inhalation TRV or soil guideline from a regulatory agency. See Section 2.2(Exposure Assessment) for further details.

F2 Fraction = the F2 fraction is comprised of C_{11}-C_{16} aromatic and aliphatic PHCs (CCME 2008), and biphenyl.

F3 Fraction = the F3 fraction is comprised of C_{17}-C_{34} aromatics and aliphatic PHCs (CCME 2008), and the carcinogenic PAH groups.

LMW PAHs represent 2 and 3 ring PAHs, based on data presented in US EPA (2007). For the purpose of this assessment, the LMW PAH group includes: acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, naphthalene and phenanthrene.

HMW PAHs represent PAHs with 4 or more rings, based on data presented in US EPA (2007). For the purpose of this assessment, the HMW PAH group includes: the aromatic C_{17}-C_{34} group and pyrene. Note that the aromatic C_{17}-C_{34} group also include: 3-methylcholanthrene, 7,12-dimethylbenz(a)anthracene, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, fluoranthene, and indeno(1,2,3-cd)pyrene.

– Not available. A TRV, SQG or SWQG was not available, and the COPC could not be evaluated further.

NA = Not assessed. In the case for CACs, hydrogen chloride, hydrogen fluoride and hydrogen sulphide, chemicals were strictly related to inhalation exposure and therefore were not included in the soil and surface water assessments. For other chemicals (e.g., benzene and toluene) these chemicals did not screen-on in the physical-chemical screening process, and therefore were not assessed in the soil and surface water assessments.

2.4 Hazard Characterization

This section describes the general approach used for the evaluation of potential hazards that may occur in wildlife in association with exposure to the COPC. Different approaches were used in the SLWRA for the evaluation of potential inhalation and multiple pathway hazards. These approaches are described below.

2.4.1 Inhalation Assessment

The hazard characterization step of the 2013 SLWRA for inhalation exposure involved comparing maximum predicted COPC air concentrations for each of the assessment cases to wildlife inhalation TRVs.

Hazard Quotient (HQ) values were calculated by dividing the predicted chemical concentration in air by the available TRV, as indicated in the following equation:

$$\text{Inhalation Pathway HQ} = \frac{\text{Maximum Predicted Air Concentration (mg/m}^3\text{)}}{\text{TRV (mg/m}^3\text{)}}$$

Interpretation of the predicted HQ values went as follows:

- HQ < 1: estimated maximum exposure is less than the associated TRV, indicating that risks to wildlife are negligible for the COPC.
- HQ >1: estimated maximum exposure is greater than the associated TRV, indicating that potential wildlife health effects may exist.

2.4.2 Multiple Pathway Assessment

For the soil quality assessment of the 2013 SLWRA, maximum predicted soil concentrations were compared against a range of soil quality guidelines (ESRD SQGs, OMOE or EPA Eco-SSLs). Similarly, maximum predicted water concentrations were compared against water quality guidelines intended to be protective of terrestrial wildlife for the water quality assessment of the 2013 SLWRA. Where soil or water concentrations did not exceed guidelines, it was assumed that potential risks to wildlife would be negligible. Where COPC concentrations exceed guidelines, the potential risks to wildlife were interpreted through review of the basis of the guidelines and comparison to measured soil concentrations from reference areas (i.e., outside the oil sands region).

3.0 RESULTS OF THE SCREENING LEVEL WILDLIFE RISK ASSESSMENT

Separate assessments were completed for the acute and chronic exposure estimates. Acute exposure was only evaluated for the inhalation pathway as secondary exposure pathways require deposition of COPCs onto environmental media which would be relevant for chronic exposure only. In the chronic assessment, distinction was made between inhalation and soil ingestion exposures, as previously described.

In recognition of the influence of duration and pathway of exposure, results were segregated into:

- acute inhalation pathway;
- chronic inhalation pathway;
- chronic soil pathway; and

- chronic surface water pathway.

The acute and chronic results are presented in scientific notation as many of the calculated numerical values were well below 1.0. For instance, the acute risk estimate for the mammalian receptor associated with exposure to the 2013 Base Case carbon monoxide air concentration is 1.6E-03, which is equivalent to an HQ of 0.0016 (see Table 3-1). An explanation of the acute and chronic inhalation, as well as the soil and water assessments is provided in the following sections.

3.1 Acute Inhalation Assessment

Acute maximum inhalation risk estimates, expressed as HQ values, are based on an assumed exposure period that lasts from hours to days. The maximum predicted acute inhalation HQ values for all the receptor locations are provided in Table 3-1 for the mammalian and Table 3-2 for the avian wildlife receptors.

Table 3-1 Maximum Acute Inhalation Hazard Quotients for Mammalian Wildlife

Category	Chemicals of Potential Concern	2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	CO	1.6E-03	1.6E-03	1.6E-03
	NO_2	3.3E-03	3.4E-03	3.2E-03
	SO_2	6.3E-05	6.3E-05	6.4E-05
Metals	Aluminum	2.3E-06	2.3E-06	2.4E-06
	Arsenic	3.6E-09	7.7E-09	8.0E-09
	Beryllium	1.9E-07	3.1E-07	3.2E-07
	Cadmium	8.3E-07	8.4E-07	1.0E-06
	Chromium	3.1E-07	3.1E-07	3.1E-07
	Chromium VI	6.1E-08	6.1E-08	6.1E-08
	Cobalt	2.4E-08	2.4E-08	2.7E-08
	Copper	3.0E-09	3.0E-09	5.5E-09
	Manganese	6.5E-08	6.5E-08	9.1E-08
	Mercury	4.7E-09	6.2E-09	9.7E-09
	Molybdenum	4.2E-10	7.3E-10	7.6E-10
	Nickel	2.5E-07	2.5E-07	2.6E-07
	Selenium	1.6E-07	1.6E-07	1.6E-07
	Vanadium	9.9E-08	1.3E-07	1.3E-07
	Zinc	4.5E-08	5.7E-08	6.6E-08
Organics	1,3-Butadiene	1.3E-09	1.3E-09	1.2E-09
	Acetaldehyde	1.7E-05	1.7E-05	1.7E-05
	Acetone	1.4E-03	1.4E-03	1.4E-03
	Acrolein	2.2E-04	2.2E-04	2.1E-04
	Aliphatic aldehyde group	3.4E-04	3.4E-04	3.3E-04
	Aliphatic C_2-C_4 group	3.7E-07	1.4E-06	1.4E-06
	Aliphatic C_5-C_8 group	4.5E-04	1.5E-03	1.6E-03
	Aromatic C_9-C_{16} group	2.2E-03	2.2E-03	6.5E-03
	Benzene	2.1E-06	2.7E-06	5.3E-06
	Cyclohexane	6.8E-06	6.8E-06	2.0E-05
	Dichlorobenzene	8.3E-11	2.1E-10	2.2E-10
	Ethylbenzene	6.4E-05	6.4E-05	1.9E-04
	Ethylene	3.2E-08	3.2E-08	8.3E-08
	Formaldehyde	5.9E-05	5.9E-05	5.7E-05
	Hexane	2.5E-06	2.5E-06	2.6E-06
	Isopropylbenzene (cumene)	1.8E-05	1.8E-05	5.4E-05
	Methyl ethyl ketone group	1.6E-06	1.6E-06	1.6E-06

Table 3-1 Maximum Acute Inhalation Hazard Quotients for Mammalian Wildlife (continued)

Category	Chemicals of Potential Concern	2013 Base Case	2013 PRM Application Case	2013 PDC
Organics (continued)	Naphthalene and substituted naphthalenes	5.2E-07	5.3E-07	5.4E-07
	Propylene	1.4E-08	1.4E-08	3.6E-08
	Propylene oxide	1.5E-09	1.0E-08	1.0E-08
	Toluene	4.0E-06	4.0E-06	1.2E-05
	Trimethylbenzene	2.1E-06	2.1E-06	6.3E-06
	Xylenes	8.5E-05	8.5E-05	2.5E-04
Sulphur compounds	CS_2 group	5.69E-06	5.74E-06	1.75E-05
	H_2S group	8.41E-06	9.98E-06	2.31E-05

PDC = Planned Development Case.

Table 3-2 Maximum Acute Inhalation Hazard Quotients for Avian Wildlife

Category	Chemicals of Potential Concern	2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	CO	2.2E-03	2.2E-03	2.2E-03
	SO_2	6.3E-05	6.3E-05	6.4E-05
Organics	Aliphatic C_5-C_8 group	1.3E-03	2.6E-03	3.0E-03
	Hexane	1.2E-04	1.2E-04	1.3E-04

PDC = Planned Development Case.

All predicted acute HQ values for all assessment cases were below 1.0 (i.e., predicted exposures were less than the TRVs) for both mammalian and avian receptors. Thus, it was concluded that predicted acute inhalation exposures to the COPCs assessed would not have an adverse effect on either avian or mammalian wildlife in the region.

3.2 Chronic Inhalation Assessment

The chronic inhalation assessment evaluates the potential health risks associated with continuous exposure to predicted maximum annual average air concentrations. The maximum predicted chronic inhalation HQ values are provided in Table 3-3 for the mammalian and Table 3-4 for the avian wildlife receptors.

With the exception of NO_2, none of the predicted chronic inhalation HQ values exceed 1.0 (i.e., predicted exposures were less than the exposure limits) for the three assessment cases (i.e., 2013 Base Case, 2013 PRM Application Case and 2013 PDC) for the mammalian and avian wildlife receptors. Therefore, it was concluded that predicted chronic inhalation exposures to the COPCs assessed would not have an adverse effect on avian or mammalian wildlife receptors in the region. The risks associated with the predicted chronic NO_2 inhalation HQ values of 1.4, 1.7 and 1.8 for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC are discussed below.

Table 3-3 Chronic Inhalation Hazard Quotients for Mammalian Wildlife

Category	Chemicals of Potential Concern	2013 Base Case	2013 PRM Application Case	2013 PDC
Criteria Air Contaminants	NO_2	**1.4E+00**	**1.7E+00**	**1.8E+00**
	SO_2	2.5E-03	2.5E-03	2.1E-03
Metals	Aluminum	1.8E-05	2.4E-05	2.5E-05
	Arsenic	7.7E-09	9.0E-09	1.9E-08
	Barium	2.9E-06	3.5E-06	7.5E-06
	Cadmium	4.8E-03	6.5E-03	6.8E-03
	Chromium	4.1E-06	4.7E-06	6.4E-06
	Chromium VI	7.6E-07	8.7E-07	1.2E-06
	Cobalt	1.2E-04	1.6E-04	1.7E-04
	Copper	1.1E-06	1.4E-06	1.7E-06
	Manganese	5.6E-07	7.1E-07	7.6E-07
	Mercury	3.7E-07	4.4E-07	9.6E-07
	Nickel	1.4E-05	1.4E-05	2.6E-05
	Vanadium	2.4E-05	2.6E-05	5.4E-05
Organics	1,3-Butadiene	5.2E-05	8.8E-05	8.9E-05
	Acetaldehyde	1.4E-04	2.3E-04	2.3E-04
	Acetone	7.1E-07	1.2E-06	1.2E-06
	Acrolein	9.0E-04	1.5E-03	1.5E-03
	Aliphatic C_5-C_8 group	1.5E-04	4.4E-04	4.7E-04
	Aliphatic C_9-C_{16} group	1.8E-03	1.8E-03	2.8E-03
	Aromatic C_9-C_{16} group	6.4E-04	6.4E-04	9.7E-04
	Benzene	1.3E-04	2.5E-04	2.6E-04
	Cyclohexane	2.8E-05	2.8E-05	4.2E-05
	Dichlorobenzene	7.6E-09	7.7E-09	1.7E-08
	Ethylbenzene	7.0E-04	7.0E-04	1.1E-03
	Ethylene	1.4E-06	1.4E-06	3.5E-06
	Formaldehyde	2.1E-03	3.5E-03	3.6E-03
	Hexane	1.8E-05	1.8E-05	2.7E-05
	Isopropylbenzene (cumene)	1.1E-04	1.1E-04	1.7E-04
	Methyl ethyl ketone group	1.7E-06	2.9E-06	2.9E-06
	Naphthalene and substituted naphthalenes	9.0E-07	2.2E-06	2.4E-06
	Propylene	1.2E-07	1.2E-07	3.0E-07
	Propylene oxide	4.6E-09	1.3E-08	1.6E-08
	Toluene	4.1E-03	4.2E-03	6.2E-03
	Trimethylbenzene	1.0E-05	1.0E-05	1.5E-05
	Xylenes	6.9E-03	6.9E-03	1.0E-02
Sulphur Compounds	CS_2 group	6.0E-06	1.6E-05	2.8E-05
	H_2S group	1.7E-04	5.8E-04	6.1E-04

PDC = Planned Development Case.

Table 3-4 Chronic Inhalation Hazard Quotients for Avian Wildlife

COPC	Chemical	2013 Base Case	2013 PRM Application Case	2013 PDC
Organics	Aliphatic C_5-C_8 group	7.7E-03	2.3E-02	2.5E-02
	Hexane	3.0E-04	3.0E-04	4.5E-04

PDC = Planned Development Case.

The exceedance of the chronic mammalian wildlife TRV for NO_2 is not expected to result in adverse effects for the following reasons:

- The use of a No-Observable-Adverse-Effect-Level of 0.10 mg/m³ to derive the TRV used for NO_2 is conservative. A Lowest-Observable-Adverse-Effect-Level of 1 mg/m³ (i.e., 10 times higher than the NOAEL) was also identified from the same study (Tabacova et al. 1985). Comparison of the highest predicted NO_2 concentration, occurring at the maximum point of impingement of 45 µg/m³, to a LOAEL-based TRV of 250 µg/m³ suggests that adverse effects are not expected, as the predicted maximum concentration is well below the LOAEL-based TRV.
- The exceedances of NO_2 are not expected to represent a true risk to wildlife in the PRM area, since the HQ values were conservatively based on the predicted maximum annual NO_2 concentrations. Actual inhalation exposure to wildlife in the region would be expected to be lower than the maximum predicted values as animals move around within their home range.

The overall conclusion of the chronic inhalation assessment is that the PRM would pose negligible to low inhalation health risks to mammalian and avian wildlife in the region.

3.3 Chronic Soil Quality Assessment

Chronic risk estimates were based on comparison of predicted soil concentrations with their applicable guidelines. Most soil concentrations were below their respective guidelines, indicating that the predicted soil concentrations for these COPCs are not expected to have an adverse effect on wildlife populations in the study area. Although ESRD SQGs were not exceeded, estimated soil concentrations for antimony, selenium and vanadium, were greater than their US EPA Eco-SSLs. These exceedances and their significance are discussed further below.

A comparison of the maximum predicted soil concentrations and soil quality guidelines/standards/screening levels is provided in Table 3-5 for mammalian and avian wildlife receptors.

Table 3-5 Comparison of Predicted Soil Concentrations with Soil Quality Guidelines Protective of Wildlife [mg/kg]

Chemical	2013 Base Case	2013 PRM Application Case	2013 PDC	SQG	OMOE 2011 Standard[2]		ECO SSL - US EPA 2007[3]	
				ESRD 2010[1]	Avian	Mammalian	Avian	Mammalian
Metals								
Aluminum	3.2E+03	3.2E+03	3.2E+03	ND	ND	ND	ND	ND
Antimony	**3.8E-01**	**3.8E-01**	**3.8E-01**	ND	ND	25	ND	0.27
Arsenic	3.1E+00	3.1E+00	3.1E+00	380	333	51	43	46
Barium	1.3E+02	1.3E+02	1.3E+02	ND	672	394	ND	2,000
Beryllium	3.2E-01	3.2E-01	3.2E-01	ND	13	ND	ND	21
Cadmium	2.3E-01	2.6E-01	2.6E-01	3.8	1.9	2.4	0.8	0.36
Chromium	4.5E+00	4.5E+00	4.5E+00	ND	161	3,000	26	34
Chromium VI	3.7E-01	3.7E-01	3.7E-01	ND	ND	914	ND	130
Cobalt	1.1E+01	1.1E+01	1.1E+01	ND	180	239	120	230
Copper	7.4E+00	7.4E+00	7.4E+00	300	3,060	283	28	49
Lead	5.8E+00	5.8E+00	5.8E+00	70	32	1,760	11	56
Manganese	7.9E+02	7.9E+02	7.9E+02	ND	ND	ND	4,300	4,000
Mercury	7.1E-02	7.1E-02	7.2E-02	ND	20	32	ND	ND
Methylmercury	0.0E+00	0.0E+00	0.0E+00	ND	0.034	0.11	ND	ND
Molybdenum	6.4E-01	6.4E-01	6.4E-01	ND	74	6.9	ND	ND
Nickel	6.4E+00	6.4E+00	6.4E+00	355	5,430	5,010	210	130
Selenium	**7.1E-01**	**7.1E-01**	**7.1E-01**	4.5	5.5	2.4	1.2	0.6
Silver	4.0E-03	4.7E-03	4.8E-03	ND	ND	ND	4.2	14
Strontium	4.3E+01	4.3E+01	4.3E+01	ND	ND	ND	ND	ND
Uranium	7.8E-01	7.8E-01	7.8E-01	33	ND	33	ND	ND
Vanadium	**1.2E+01**	**1.2E+01**	**1.2E+01**	ND	18	108	7.8	280
Zinc	2.9E+01	2.9E+01	2.9E+01	640	337	4,200	46	79
Organics								
Acetaldehyde	1.0E-07	1.7E-07	1.7E-07	ND	ND	ND	ND	ND
Acetone	4.9E-07	8.2E-07	8.3E-07	ND	ND	32	ND	ND
Acrolein	5.8E-09	9.7E-09	9.8E-09	ND	ND	ND	ND	ND
Aliphatic aldehydes	1.5E-06	2.5E-06	2.5E-06	ND	ND	ND	ND	ND
Acenaphthenes/acenaphthylenes	2.6E-08	7.9E-08	8.3E-08	21.5	ND	6,630	ND	ND
Aliphatic C9-C16 group	4.8E-04	4.8E-04	7.7E-04	9,800	ND	ND	ND	ND
Aliphatic C_{17}-C_{34} group	1.4E-04	1.4E-04	2.6E-04	16,000	ND	ND	ND	ND
Aromatic C_9-C_{16} group	4.5E-09	1.3E-08	1.4E-08	9,800	ND	ND	ND	ND
Aromatic C_{17}-C_{34} group	3.6E-07	1.1E-06	1.1E-06	16,000	ND	ND	ND	ND
Anthracene/phenanthrenes	2.0E-01	2.0E-01	2.0E-01	43	ND	38,000	ND	ND
Biphenyl	1.2E-08	1.2E-08	1.2E-08	ND	ND	ND	ND	ND

Table 3-5 Comparison of Predicted Soil Concentrations with Soil Quality Guidelines Protective of Wildlife [mg/kg] (continued)

Chemical	2013 Base Case	2013 PRM Application Case	2013 PDC	SQG	OMOE 2011 Standard[2]		ECO SSL - US EPA 2007[3]	
				ESRD 2010[1]	Avian	Mammalian	Avian	Mammalian
Carcinogenic PAH Group 1	1.0E-05	2.1E-05	2.3E-05	0.6	ND	1,600	ND	ND
Carcinogenic PAH Group 2	4.5E-05	1.3E-04	1.4E-04	6.2	ND	ND	ND	ND
Carcinogenic PAH Group 3	7.3E-06	2.0E-05	2.1E-05	6.2	ND	ND	ND	ND
Fluorenes/fluoranthenes and substituted fluorenes	6.1E-06	1.8E-05	1.9E-05	15.4	ND	0.69	ND	ND
Naphthalene	1.0E-02	1.0E-02	1.0E-02	8.8	ND	379	ND	ND
Pyrenes and substituted pyrenes	1.0E-02	1.1E-02	1.1E-02	7.7	ND	2,700	ND	ND
Formaldehyde	5.0E-09	8.5E-09	8.6E-09	ND	ND	ND	ND	ND
Methyl ethyl ketone group	3.3E-07	5.2E-07	5.3E-07	ND	ND	5,680	ND	ND
Propylene oxide	2.0E-05	5.7E-05	6.7E-05	ND	ND	ND	ND	ND
HMW PAH group [4]	6.9E-05	1.9E-04	2.0E-04	ND	ND	ND	ND	1.1
LMW PAH group [5]	1.7E-08	2.5E-08	2.6E-08	ND	ND	ND	ND	100
F2 Fraction	4.8E-04	4.8E-04	7.7E-04	9,800	ND	ND	ND	ND
F3 Fraction	2.0E-04	3.1E-04	4.5E-04	16,000	ND	ND	ND	ND

Notes:

Bold values indicate that the value exceeded the ESRD SQG, OMOE soil standard, or US EPA ECO-SSL guideline.

ND = SQG or Eco-SSL not available.

(1) Alberta SQGs are referenced from ESRD (ESRD 2010) Surface Soil Remediation Guideline Values for Natural Area Land Use - Wildlife Soil and Food Ingestion (Table A-1).

(2) OMOE soil standards are referenced from (OMOE 2011) Rationale for the Development of Soil and Ground Water Standards for use at Contaminated Sites in Ontario.

(3) US EPA Eco-SSLs are referenced from US EPA (2010) for metals, or US EPA (2007) for PAH Eco-SSLs (Table 2.1).

(4) HMW PAH includes all PAHs with 4 or more rings (CCME 2008; US EPA 2007).

(5) LMW PAH includes all 2 and 3 ring PAHs (CCME 2008; US EPA 2007).

SQG = Soil Quality Guideline; ECO-SSL = Ecological Soil Screening Level; AEW = Alberta Environment and Water; OMOE = Ontario Ministry of Environment; PDC = Planned Development Case.

3.3.1 Antimony

The predicted soil concentration for antimony was 0.38 mg/kg under all three assessment cases. Of the two antimony samples that were collected, one sample was non-detect. Antimony concentrations range from 0.06 to 0.38 mg/kg and an average concentration of 0.22 mg/kg. The soil concentration under the 2013 Base Case is due entirely to the measured concentrations of antimony in soils in the JME and PRM lease areas (maximum of 0.38 mg/kg), which exceeds the US EPA Eco-SSL of 0.27 mg/kg for mammalian ground insectivore (i.e., shrew). The 2013 Base Case concentration does not exceed the Eco-SSL of 10 mg/kg for mammalian herbivores (i.e., vole) or the Eco-SSL of 4.9 mg/kg for mammalian carnivores (i.e., weasel).

The US EPA Eco-SSL is conservative since it is lower than the range of reported typical background antimony concentrations in U.S. soils. The predicted antimony concentration of 0.38 mg/kg is below the typical U.S. background soil range of approximately 0.5 to 3.6 mg/kg. The lack of increase from the 2013 Base Case to 2013 PRM Application Case and 2013 PDC indicates a negligible impact from the PRM or future emission sources in the area. Antimony concentrations are well below the mammalian OMOE soil standard of 25 mg/kg. As part of the Alberta Ambient Soil Quality Database, antimony concentrations have been measured in soil at various sites (i.e., transportation, parkland and commercial) around Alberta (AEW 1996). The mean antimony concentration across the various sites was 0.26 mg/kg, with minimum concentrations below the analytical detection limits and a maximum of 1.86 mg/kg. This natural mean concentration of 0.26 mg/kg is comparable to the maximum and mean antimony concentration measured in the lease areas of 0.38 and 0.22 mg/kg, respectively. Therefore, the risks posed to mammals by antimony concentrations in the PRM area are no greater than those in other parts of the province. This suggests that measured Baseline and predicted long-term soil concentrations associated with PRM and planned projects and activities are not expected to adversely affect wildlife populations in the region.

3.3.2 Selenium

The estimated soil concentration for selenium is 0.71 mg/kg under all three assessment cases. This soil concentration is equal to the 95 UCLM of measured concentrations of selenium in 114 soil samples near JME and PRM. The lack of increase from the 2013 Base Case to 2013 PRM Application Case and 2013 PDC indicates a negligible impact from the PRM.

The selenium soil concentration only exceeded the most conservative mammalian Eco-SSL of 0.63 mg/kg for mammalian ground insectivores (i.e., shrew). The estimated selenium concentration of 0.71 mg/kg is below the Eco-SSLs derived for other trophic levels of mammals, such as the mammalian herbivores (i.e., vole) with an Eco-SSL of 2.7 mg/kg and the mammalian carnivores (i.e., weasel) with an Eco-SSL of 2.8 mg/kg.

Median concentrations of selenium in surface soils of the Prairie Provinces were reported to be 0.5 mg/kg in 1992 by the Geological Survey of Canada (CCME 2009). In Alberta, selenium soil levels ranged from 0.1 to 2.7 mg/kg with a mean (±s.d.) of 0.55 mg/kg (±0.28). In 2002, the Alberta Environmentally Sustainable Agriculture Soil Quality Monitoring Program conducted a survey of agricultural soils to provide a benchmark database of elemental concentrations in Alberta (GOA 2004). From 129 agricultural sites across the province, total selenium levels ranged from 0.1 to 1.6 mg/kg in the 0 to 15 cm depth samples, and from 0.001 to 2.3 mg/kg in the 15 to 30 cm depth samples. The estimated concentration of selenium for all three cases (0.71 mg/kg for the 95 UCLM, and 0.59 mg/kg for the average) falls within the range of typical selenium levels in Alberta.

The ESRD SQG of 4.5 mg/kg was derived to protect primary consumers (domestic and wild animals) from exposure via ingestion of food and soil on agricultural lands (ESRD 2010). In addition, the OMOE soil standard of 2.4 for mammalian wildlife and 5.5 for avian wildlife were the most conservative OMOE standards presented for all mammals and avian species evaluated. As the 95 UCLM selenium concentration of 0.71 mg/kg is below the ESRD SQG and the OMOE soil standard, and within the typical range of selenium levels across Alberta, it is unlikely that the baseline and predicted long-term selenium concentrations as a result of PRM and other existing and planned developments would affect wildlife populations in the vicinity of the PRM.

3.3.3 Vanadium

The estimated soil concentration for vanadium was 12 mg/kg for the 2013 Base Case, 2013 PRM Application Case and 2013 PDC. The measured soil concentration is based on the 95 UCLM of 114 measured soil samples in the study area, and exceeds the most conservative US EPA avian Eco-SSL of 7.8 mg/kg derived for ground insectivores (i.e., woodcock), but does not exceed the Eco-SSL of 13 mg/kg derived for avian herbivores (i.e., dove) or the Eco-SSL of 140 mg/kg derived for the avian carnivore (i.e., hawk). Additionally, the vanadium soil concentrations did not exceed the Eco-SSL for mammals of 280 mg/kg and also did not exceed the OMOE soil standard of 18 mg/kg for avian wildlife or 108 mg/kg for mammalian wildlife.

All soil samples collected from the site had detectable levels of vanadium (with the exception of one non-detect sample), with concentrations ranging from 0.06 to 52 mg/kg and an average concentration of 11 mg/kg. According to the soil metal database, natural mean concentrations of vanadium in Canadian soils range from 38 mg/kg to 42 mg/kg (CCME 1997). Similarly, the mean vanadium concentration reported in the Alberta Ambient Soil Quality Database across Alberta was 36 mg/kg, with a minimum concentration of 10 mg/kg and a maximum of 70 mg/kg (AEW 1996). The potential vanadium-related risks posed to regional wildlife are no greater than those in other parts of Alberta or the country. This suggests that measured and predicted soil concentrations associated with PRM and planned projects and activities are not expected to adversely affect wildlife populations in the LSA.

3.4 Chronic Surface Water Assessment

Chronic risk estimates associated with surface water ingestion exposure pathways were based on comparison of predicted surface water concentrations to relevant SWQGs, as identified previously. Where predicted surface water concentrations were below their respective guidelines, it was concluded that predicted long-term surface water concentrations would not adversely impact terrestrial wildlife populations.

A comparison of predicted surface water concentrations and SWQGs for wildlife is provided in Table 3-6 for wildlife receptors.

With the exception of phenol (i.e., total phenolic compounds), predicted surface water concentrations did not exceed any of the SWQGs for wildlife under any of the three assessment cases (i.e., 2013 Base Case, 2013 PRM Application Case, and 2013 PDC), indicating that predicted surface water concentrations associated with the PRM and planned projects and activities will not adversely affect wildlife populations in the LSA. Further discussion of the phenol exceedances are discussed below.

Table 3-6 Comparison of Predicted Surface Water Concentrations with Surface Water Quality Guidelines Protective of Wildlife [mg/L]

Category	Chemical	2013 Base Case	2013 PRM Application Case	2013 PDC	ESRD 2010[(1)]
Metals	Aluminum	2.7E-01	3.0E-01	2.9E-01	5
	Antimony	7.0E-04	7.0E-04	7.7E-04	–
	Arsenic	1.5E-03	1.5E-03	1.6E-03	0.025
	Barium	7.1E-02	8.1E-02	8.0E-02	–
	Beryllium	2.8E-04	2.9E-04	3.4E-04	–
	Boron	9.0E-02	9.5E-02	2.0E-01	5
	Cadmium	7.6E-05	8.1E-05	1.6E-04	0.08
	Chromium	1.5E-03	1.9E-03	1.8E-03	0.05
	Chromium VI	0.0E+00	0.0E+00	0.0E+00	–
	Cobalt	1.2E-03	1.3E-03	2.3E-03	–
	Copper	1.7E-03	1.8E-03	2.0E-03	0.5
	Lead	3.8E-04	4.1E-04	4.6E-04	0.1
	Lithium	3.8E-02	3.8E-02	4.3E-02	–
	Manganese	4.6E-01	4.5E-01	5.8E-01	–
	Mercury	9.1E-07	1.9E-06	2.1E-06	0.003
	Methyl mercury	0.0E+00	0.0E+00	0.0E+00	–
	Molybdenum	4.9E-04	5.7E-04	2.1E-02	–
	Nickel	3.5E-03	3.7E-03	4.5E-03	1
	Selenium	4.7E-04	4.8E-04	5.1E-04	0.05
	Silver	1.5E-05	1.5E-05	2.0E-05	0.05
	Strontium	1.9E-01	2.1E-01	2.5E-01	–
	Thallium	3.4E-05	3.6E-05	3.9E-05	–
	Uranium	2.6E-04	2.8E-04	4.5E-04	0.2
	Vanadium	2.4E-03	2.6E-03	3.2E-03	–
	Zinc	1.4E-02	1.4E-02	1.4E-02	50
Organics	Acetaldehyde	0.0E+00	0.0E+00	0.0E+00	–
	Acetone	0.0E+00	0.0E+00	0.0E+00	–
	Acrolein	0.0E+00	0.0E+00	0.0E+00	–
	Aliphatic aldehydes	0.0E+00	0.0E+00	0.0E+00	–
	Acenaphthenes/acenaphthylenes	0.0E+00	2.0E-05	2.1E-05	–
	Aliphatic C_9-C_{16} group	0.0E+00	0.0E+00	0.0E+00	42.6
	Aliphatic C_{17}-C_{34} group	0.0E+00	0.0E+00	0.0E+00	69.0
	Aromatic C_9-C_{16} group	0.0E+00	0.0E+00	0.0E+00	42.6
	Aromatic C_{17}-C_{34} group	0.0E+00	0.0E+00	0.0E+00	69.0
	Ammonia	1.6E-01	2.1E-01	2.4E-01	–
	Anthracene/phenanthrenes	0.0E+00	1.8E-04	1.9E-04	–
	Biphenyl	0.0E+00	3.8E-10	1.4E-06	–
	Carcinogenic PAH group 1	0.0E+00	2.5E-05	2.6E-05	–
	Carcinogenic PAH group 2	0.0E+00	1.1E-04	1.1E-04	–
	Carcinogenic PAH group 3	0.0E+00	1.5E-06	2.0E-06	–
	Fluorenes/fluoranthenes	0.0E+00	2.3E-04	2.4E-04	–
	Naphthalene	0.0E+00	5.0E-06	7.1E-06	–
	Pyrenes and substituted Pyrenes	0.0E+00	2.4E-05	2.7E-05	–
	Formaldehyde	0.0E+00	0.0E+00	0.0E+00	–
	Methyl ethyl ketone group	0.0E+00	0.0E+00	0.0E+00	–

Table 3-6 Comparison of Predicted Surface Water Concentrations with Surface Water Quality Guidelines Protective of Wildlife [mg/L] (continued)

Category	Chemical	2013 Base Case	2013 PRM Application Case	2013 PDC	ESRD 2010[1]
Organics (continued)	Phenol	**4.5E-03**	**6.5E-03**	**6.8E-03**	0.002
	Propylene oxide	0.0E+00	0.0E+00	0.0E+00	–
	F2 Fraction[2]	0.0E+00	3.8E-10	1.4E-06	42.6
	F3 Fraction[3]	0.0E+00	1.3E-04	1.3E-04	69.0

Notes:
Bold values indicate that the value exceeded the Alberta SQG or US EPA ECO-SSL guideline.
— = SWQG not available.
(1) Alberta SWQGs are referenced from ESRD (2010) Surface Water Quality Guidelines for Livestock and Wildlife Water (Table C-11).
PDC = Planned Development Case.

Predicted concentrations of phenol (i.e., total phenolic compounds) in rivers and streams were predicted to exceed the Alberta SWQG in the 2013 Base Case, 2013 Application Case and 2013 PDC. The toxicological basis of the phenol SWQG for the protection of livestock watering is uncertain and was derived by CCME in 1987. Unfortunately, there is no fact sheet available from CCME to present the basis of the livestock SWQG and describe the risk associated with wildlife consumption of phenol concentrations in surface water based on the CCME SWQG. Given the lack of documentation supporting the derivation of the phenol SWQG a literature search was performed to identify wildlife toxicity reference values for mammals and birds to develop a water quality guideline.

A NOAEL TRV of 12 mg/kg/day for mammals (i.e., rats) was developed by the US ARMY (USACHPPM 2008) based on developmental effects (i.e., reduced fetal body weights). A LOAEL of 360 mg/kg/day and a NOAEL of 120 mg/kg/day was derived in the Proctor & Gamble (P&G 1997) study based on developmental effects. The recommended NOAEL-based TRV was divided by an uncertainty factor of 10 to account for potential interspecies differences. No data for avian species were found.

The NOAEL-based TRV of 12 mg/kg/day was used to calculate a receptor-specific water quality guideline (WQG) for the moose and snowshoe hare, based on methods described by ESRD (2010). Table 3-7 presents the assumptions used to calculate the receptor-specific WQGs for phenol and the receptor-specific surface water quality guidelines for the protection of wildlife.

Table 3-7 Receptor-specific water quality guidelines derived for phenol

Parameter	Units	Moose	Snowshoe Hare
Toxicity Reference Value (TRV)	mg/kg/day	12	12
Body Weight (BW)	kg	450	1.4
Water Ingestion Rate (WIR)	L/day	24	0.13
Water Quality Guideline (WQG)[1]	mg/L	225	129

Notes:
(1) Calculated with the following formula:
WQG= (TRV * BW)/ WIR

Comparison of the predicted maximum phenol concentrations (0.0045 to 0.0068 mg/L) to the receptor-specific WQGs (129 to 225 mg/L) indicates that adverse effects from exposure to phenol are not expected.

Finally, total phenols are naturally high in the LSA and maximum observed concentrations range from 0.025 to 0.050 mg/L. The source of the naturally occurring phenols is soil with a high content of oil sands. Further details are provided in the Aquatic Resources assessment (Appendix 2, Section 3.3.3.1).

3.5 Summary of Conservative Assumptions Used in the Screening Level Wildlife Risk Assessment

Conservative assumptions applied to the 2013 SLWRA include:

- Wildlife receptors were assumed to be exposed to the maximum predicted 1-hour or 24-hour (acute) air concentrations and continuously exposed to maximum predicted annual average (chronic) air concentrations in the LSA.
- Wildlife receptors were assumed to be exposed to maximum predicted air concentrations for the entire durations of their lifetimes; in actuality, most wildlife species move around within their home ranges or migrate, meaning that they will not be continuously exposed to maximum predicted air concentrations from PRM over their entire lifetimes.
- Soil concentration calculations did not include certain known chemical loss mechanisms (i.e., soil erosion and leaching), but did include abiotic and biotic degradation and volatilization losses for organic COPCs.
- Chronic inhalation TRVs were developed using the lowest NOAELs or LOAELs selected for the most sensitive species available.

3.6 Conclusions

On both an acute and chronic basis, maximum predicted air concentrations did not exceed TRVs protective of avian and mammalian wildlife with the exception of NO_2 on a chronic basis.

Chronic inhalation HQs of 1.4, 1.7, and 1.8 were predicted for NO_2 under the 2013 Base Case, 2013 PRM Application Case, and 2013 PDC, respectively. The predicted exceedances are attributable to the conservative assumptions incorporated in the 2013 SLWRA, including the use of a conservative TRV and the maximum predicted annual NO_2 air concentrations. The predicted NO_2 air concentrations are below levels at which effects have been observed in animals. The overall conclusion is that the predicted NO_2 air concentrations are not expected to have an adverse effect on wildlife.

For the most part, the soil concentrations for COPCs are not predicted to measurably increase from the 2013 Base Case to the 2013 PRM Application Case or 2013 PDC. This indicates that the emissions from PRM and other sources in the area will have negligible impacts on terrestrial wildlife health.

The soil concentrations in the 2013 SLWRA did not exceed relevant ESRD soil quality guidelines for any of the COPCs. In addition, the soil concentrations did not exceed any of the OMOE soil standards intended for the protection of wildlife.

Exceedances of the US EPA ecological soil screening levels (Eco-SSL) were discussed for antimony, selenium, and vanadium. For these COPCs, the predicted exceedances were associated with the most conservative avian and mammalian Eco-SSLs derived for the protection of ground insectivores. However, soil concentrations do not exceed the guidelines derived for avian or mammalian herbivores and carnivores.

The antimony, selenium, and vanadium concentrations estimated for the 2013 Base Case are entirely due to existing (i.e., measured) concentrations in the PRM area. The measured concentrations of the three metals are within the range of typical, background concentrations for Albertan or Canadian soils. As such, the risks posed to avian and mammalian wildlife by antimony, selenium and vanadium concentrations in the PRM area are no greater than those in other parts of the province.

The surface water concentrations in the 2013 SLWRA did not exceed any of the ESRD surface water quality guidelines intended for the protection of wildlife, with the exception of phenol. However, the calculation of receptor-specific water quality guidelines for phenol demonstrated that adverse effects are not expected.

The results of the 2013 SLWRA indicate that the overall risks posed to wildlife health will be low. Therefore, no impacts to wildlife populations are expected based on estimated wildlife exposures to predicted maximum acute and chronic air concentrations or predicted soil and surface water concentrations. These conclusions are consistent with those presented in the original WHRA.

As the findings of the 2013 SLWRA did not markedly differ from those presented in the EIA WHRA, the scope of the current assessment was not expanded to the detailed quantitative risk assessment originally presented in the EIA. Further study is considered unnecessary as the 2013 SLWRA, like the EIA WHRA, did not identify an impact to the terrestrial wildlife as a result of the PRM emissions.

4.0 REFERENCES

Abou-Donia, M.B., Hu, Z.H. Lapadula, D.M., et al. 1991. *Mechanisms of joint neurotoxicity of n-hexane, methyl isobutyl ketone and O-ethyl O-4-nitrophenyl phenylphosphonothioate in hens*. J Pharmacol Exp Ther 257(1): 282-289.

AEW (Alberta Environment and Water). 1996. *Alberta Ambient Soil Quality Database. Letter received from: Ted Nason*. Alberta Environmental Protection, Chemicals Assessment and Management Division, Soil Protection Branch. February 1996.

ACGIH (American Conference of Governmental Industrial Hygienists). 1996. *Acetone*. CAS: 67-64-1. Documentation of the Threshold Limit Values and Biological Exposure Indices. ACGIH®, Cincinnati. OH

ACGIH. 2005. *Ethylene*. CAS: 74-85-1. Documentation of the Threshold Limit Values and Biological Exposure Indices. ACGIH®, Cincinnati. OH.

ACGIH. 2006. *Propylene*. CAS: 115-07-1. Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. ACGIH®, Cincinnati. OH.

ATSDR (Agency for Toxic Substances and Disease Registry). 1994. *Toxicological Profile for Acetone*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. May 1994

ATSDR. 1996. *Toxicological Profile for Carbon Disulfide*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. August 1996.

ATSDR. 1999a. *Toxicological Profile for n-Hexane*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. July 1999.

ATSDR. 1999b. *Toxicological Profile for Mercury*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. March 1999.

ATSDR. 2000. *Toxicological Profile for Toluene*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. September 2000.

ATSDR. 2004. *Toxicological Profile for Cobalt*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. April 2004.

ATSDR. 2005a. *Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. August 2005.

ATSDR. 2005b. *Toxicological Profile for Nickel*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. August 2005.

ATSDR. 2006. *Toxicological Profile for Hydrogen Sulfide*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. July 2006.

ATSDR. 2007a. *Toxicological Profile for Arsenic*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. August 2007.

ATSDR. 2007b. *Toxicological Profile for Xylenes*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. August 2007.

ATSDR. 2008a. *Toxicological Profile for Aluminum*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. September 2008.

ATSDR. 2008b. *Draft Toxicological Profile for Cadmium*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. September 2008.

ATSDR. 2008c. *Toxicological Profile for Chromium*. Atlanta, GA: United States Department of Health and Human Services, Public Health Service. September 2008.

ATSDR. 2009. *Draft Toxicological Profile for 1,3-butadiene*. US Department of Health and Human Services, Public Health Service. September 2009

ATSDR. 2012. *Toxicological Profile for Manganese*. Atlanta, GA: US Department of Health and Human Services, Public Health Service. September 2012

BC MWLAP (British Columbia Ministry of Water, Land and Air Protection). 1998. *Recommended Guidance and Checklist for Tier 1 Ecological Risk Assessment of Contaminated Sites in British Columbia*. Prepared for BC Ministry of Water, Land and Air Protection by W.G. Landis, A.J., Markiewicz, V. Wilson, A. Fairbrother, and G. Mann.

CCME (Canadian Council of Ministers of the Environment). 1996. *A Framework for Ecological Risk Assessment: General Guidance*. Winnipeg, MB: The National Contaminated Sites Remediation Program, Canadian Council of Ministers of the Environment.

CCME. 1997. *Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Vanadium*. Canadian Council of Ministers of the Environment.

CCME. 2006. *A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines*. Winnipeg, MB: Canadian Council of Ministers of the Environment.

CCME. 2008. *Canada-Wide Standards for Petroleum Hydrocarbons (PHCs) in Soil: Scientific Rationale*. Supporting Technical Documentation. Canadian Council of Ministers of the Environment. January 2008.

CCME. 2009. *Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health: Selenium*. Canadian Council of Ministers of the Environment, revised 2009.

ChemIDplus. 2013. *Database on-line search. ChemIDplus, Toxicology Data Network (TOXNET), United States National Library of Medicine*. Available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?CHEM

CICAD (Concise International Chemical Assessment Document). 2002. *Concise International Chemical Assessment Document 40*. World Health Organization: Geneva, 2002. Available at: http://www.inchem.org/documents/cicads/cicads/cicad40.htm

Environment Canada. 1994. *A Framework for Ecological Risk Assessment at Contaminated Sites in Canada: Review and Recommendations*. Prepared by C. Gaudet, EVS Environment Consultants and Environment and Social System Analysts for Environment Canada.

ESRD (Alberta Environment and Sustainable Resource Development). 2010. *Alberta Tier 1 Soil and Groundwater Remediation Guidelines*. Edmonton, AB: Alberta Environment. December 2010.

Fedde, M.R. and Kuhlmann, W.D. 1979. *Cardiopulmonary responses to inhaled sulphur dioxide in the poultry*. Poultry Science 58(6): 1584-1591.

Gaudet, C., EVS Environmental Consultants, and Environmental and Social Systems Analysts. 1994. *A framework for ecological risk assessment at contaminated sites in Canada: Review and recommendations*. Scientific Series No. 199. Ottawa, ON: Environment Canada, Ecosystem Conservation Directorate, Evaluation and Interpretation Branch.

GOA (Government of Alberta, Agriculture and Rural Development). 2004. The Micronutrient and Trace Element Status of Forty-Three Soil Quality Benchmark Sites in Alberta: Results and Summary. Available at: http://www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/aesa8885

Government of Canada. 1992. *Canadian Environmental Protection Act*. Priority Substances List Assessment Report No. 4. Toluene. Environment Canada and Health Canada. Ottawa, Ontario: Minister of Supply and Services Canada, 1992. ISBN 0-662-19950-2.

Government of Canada. 1993. *Canadian Environmental Protection Act*. Priority Substances List Assessment Report. Benzene. Environment Canada and Health Canada. Ottawa, Ontario: Minister of Supply and Services Canada, 1993. ISBN 0-662-20434-4.

Government of Canada. 1994. *Canadian Environmental Protection Act*. Priority Substances List Assessment Report. Cadmium and Compounds. Environment Canada and Health Canada. Ottawa, Ontario: Minister of Supply and Services Canada, 1994. ISBN 0-662-2046-3.

Government of Canada. 2000a. *Canadian Environmental Protection Act*. Priority Substances List Assessment Report. Acetaldehyde. May 2000.

Government of Canada. 2000b. *Canadian Environmental Protection Act, 1999*. Priority Substances List Assessment Report. Carbon Disulfide. Environment Canada and Health Canada. Minister of Public Works and Government Services, 2000. ISBN 0-662-28496-8.

HSDB (Hazardous Substances Data Bank). 2013. *Database on-line search. Hazardous Substances Data Bank (HSDB), Toxicological Data Network (TOXNET), United States National Library of Medicine*. Available at: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB.

IPCS (International Programme on Chemical Safety). 1993. *Environmental Health Criteria 150*. Benzene. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

IPCS. 1996. *Environmental Health Criteria 186*. Ethylbenzene. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

IPCS. 1998. *Environmental Health Criteria 200*. Copper. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

IPCS. 2001. *Concise International Chemical Assessment Document 32*. Beryllium and Beryllium Compounds. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

IPCS. 2002. Concise International Chemical Assessment Document 46. Carbon Disulfide. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

IPCS. 2006. *Concise International Chemical Assessment Document 69*. Cobalt and Inorganic Cobalt Compounds. Geneva, Switzerland: International Programme on Chemical Safety, United Nations Environment Programme, International Labour Organization, World Health Organization.

MA DEP (Massachusetts Department of Environmental Protection). 2003. *Updated petroleum hydrocarbon fraction toxicity values for VPH / EPH / APH methodology, Final*. Boston, MA: Office of Research and Standards, Massachusetts Department of Environmental Protection. November 2003.

NIOSH (National Institute for Occupational Safety and Health). 1996. *Arsenic (inorganic compounds, as As) IDLH Documentation*. Available at: http://www.cdc.gov/niosh/idlh/7440382.html.

NIOSH. 2007. *NIOSH PPT Program Evidence Package Aug 30, 2007 Appendix A List of Acronyms*. Available at: http://www.cdc.gov/niosh/nas/ppt/pdfs/PPT_EvPkg_092407_appA.pdf

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. *Determination of Noncancer Chronic Reference Exposure Levels*. Chronic Toxicity Summary. Nickel and Nickel Compounds. Available at: http: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=420

OEHHA. 2008. TSD for Noncancer RELs. Manganese and Compounds Reference Exposure Levels. December 2008. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=170

OMOE (Ontario Ministry of the Environment). 2006. *Rationale for the Development of Ontario Air Standards for Trimethylbenzenes: 1,2,3-Trimethylbenzene; 1,2,4-Trimethylbenzene; 1,3,5-Trimethylbenzene*. Standards Development Branch, Ontario Ministry of the Environment.

OMOE. 2011. *Rationale for the Development of Soil and Ground Water Standards for use at Contaminated Sites in Ontario*. April 15, 2011 Prepared by: Standards Development Branch Ontario Ministry of the Environment PIBS 7386e01

P&G. 1997. *Oral (gavage) developmental toxicity study of phenol in rats with cover letter dated 07/29/97*. Proctor & Gamble, Inc. EPAOTS0573686.

Ramamoorthy, S., E.G., Baddaloo and S. Ramamoorthy. 1995. *Handbook of Chemical Toxicity Profiles of Biological Species*. Volume II. Avian and Mammalian Species. Boca Raton, FL: CRC Lewis Publishers.

RIVM (National Institute of Public Health and the Environment, NIPHE). 2001. *Re-evaluation of human toxicological maximum permissible risk levels*. RIVM Report 711701 025. March 2001.

Sample, B.E. and G.W. Suter. 1994. *Estimating exposure to terrestrial wildlife to contaminants*. Oak Ridge National Laboratory, Oak Ridge TN. ES/ER/TM-86/R3.

Sample, B.E., D.M. Opresko, G.W. Suter II. 1996. *Toxicological Benchmarks for Wildlife: 1996 Revision*. Risk Assessment Program, Health and Sciences Division, Oak Ridge, Tenn. 37831 - prepared for U.S. Dept. of Energy

Suter II, G.W., Efroymson, R.A., Sample, B.E. and Jones, D.S. 2000. *Ecological Risk Assessment for Contaminated Sites*. Boca Raton, FL: Lewis Publishers, CRC Press.

Tabacova, S., Nikiforov, B. and Balabaeva, L. 1985. *Postnatal effects of maternal exposure to nitrogen dioxide*. Neurobehav Toxicol Teratol 7(6):785-789.

TPHCWG (Total Petroleum Hydrocarbon Working Group). 1997. *Development of Fraction Specific Reference Doses (RfDs) and Reference Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH), Volume 4*. Amherst, MA: Amherst Scientific Publishers.

USACHPPM. 2008. *Wildlife Toxicity Assessment for Phenol, Project Number 87-MA02T6-05E, U.S.* Aberdeen Proving Ground, Maryland.

US EPA (United States Environmental Protection Agency). 1991a. *IRIS Summary of Ethylbenzene (CASRN 100-41-4)*. Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0051.htm#refinhal.

US EPA. 1991b. *IRIS Summary of Formaldehyde (CASRN 50-00-0)*. Animal Carcinogenicity Data. Available at: http://www.epa.gov/ncea/iris/subst/0419.htm#refinhal

US EPA. 1991c. *IRIS Summary of Propylene Oxide (CASRD 75-56-9).* Animal Carcinogenicity Data. Available at: http://www.epa.gov/iris/subst/0403.htm#refinhal

US EPA. 1995. IRIS Summary of Carbon disulfide (CASRN 75-15-0). Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/iris/subst/0217.htm#refinhal

US EPA. 1996. *IRIS Summary of 1,4-Dichlorobenzene (CASRN 106-46-7)*. Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0552.htm#refinhal

US EPA. 1997. *Mercury Study Report to Congress. Volume III: Fate and Transport of Mercury in the Environment*. EPA-452/R-97-005. December 1997.

US EPA. 1998. *IRIS Summary of Barium and Compounds (CASRN 7440-39-3)*. Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0010.htm#refinhal.

US EPA. 2003a. *IRIS Summary of Acrolein (CASRN 107-02-8).* Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/iris/subst/0364.htm#refinhal

US EPA. 2003b. *IRIS Summary of Cyclohexane (CASRN 110-82-7).* Reference Concentrations for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/iris/subst/1005.htm#refinhal

US EPA. 2003c. *IRIS Summary of Methyl ethyl ketone (MEK) (CASRN 78-93-3)*. Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0071.htm#refinhal

US EPA. 2003d. *IRIS Summary of Hydrogen sulphide (CASRN 7783-06-4). Reference Concentration for Chronic Inhalation Exposure (RfC)*. Available at: http://www.epa.gov/ncea/iris/subst/0061.htm#refinhal.

US EPA. 2005a. *Guidance for Developing Eco-SSLs*. Revised February 2005. Washington, DC: Office of Solid Waste and Emergency Response, United States Environmental Protection Agency.

US EPA. 2005b. *IRIS Summary of n-Hexane (CASRN 110-54-3)*. Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0486.htm#refinhal

US EPA. 2007. *Ecological Soil Screening Levels for Polycyclic Aromatic Hydrocarbons (PAHs)*. Interim Final. OSWER Directive 9285.7-78. Washington, DC: Office of Solid Waste and Emergency Response, United States Environmental Protection Agency.

US EPA. 2010. *Ecological Soil Screening Levels Interim Eco-SSL document*. Available at: http://www.epa.gov/ecotox/ecossl

US EPA OSW (United States Environmental Protection Agency Office of Solid Waste). 1999. *Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. United States Environmental Protection Agency Region 6. Multimedia Planning and Permitting Division. Center for Combustion Science and Engineering. Office of Solid Waste. EPA 530-D-99-001A. August 1999.

US EPA OSW. 2005. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Final*. Database: Chemical-Specific Parameter Values. United States Environmental Protection Agency Region VI. Multimedia Planning and Permitting Division. Center for Combustion Science and Engineering. Office of Solid Waste and Emergency Response. EPA 530-R-05-006. September 2005.

WHO (World Health Organization). 1998a. *Environmental Health Criteria 200*. Copper. WHO: Geneva, 1998. ISBN 92 4 157200 0.

WHO. 1998b. *Environmental Health Criteria 207.* Acetone. WHO: Geneva, 1998. ISBN 92 4 157207 8

WHO. 1999. *Concise International Chemical Assessment Document 18.* Cumene. WHO: Geneva, 1999. ISBN 92 4 153018 9

WHO. 2001a. *Concise International Chemical Assessment Document 33*. Barium and Barium Compounds. WHO: Geneva, 2001. ISBN 92 4 153033 2.

WHO. 2001b. *Concise International Chemical Assessment Document 29*. Vanadium Pentoxide and Other Inorganic Vanadium Compounds. WHO: Geneva, 2001. ISBN 92 4 153029 4.

WHO. 2006. *Concise International Chemical Assessment Document 69. Cobalt and Inorganic Cobalt Compounds. WHO: Geneva 2006. ISBN 92 4 153069 3.*