2014 | Project No. 10710

Phase 2

Detailed Human Health Risk Assessment of Oil and Gas Activities in Northeastern British Columbia

Prepared for: British Columbia Ministry of Health

Intrinsik Environmental Sciences | 736-8th Avenue SW, Suite 1060 | Calgary, Alberta T2P 1H4 | Tel: (403) 237-0275 | Fax: (403) 237-0291 | intrinsik.com

DISCLAIMER

Intrinsik Environmental Sciences Inc. (Intrinsik) provided this report for the British Columbia Ministry of Health (hereafter referred to as the MoH) solely for the purpose stated in the report. The information contained in this report was prepared and interpreted exclusively for the MoH and may not be used in any manner by any other party. Intrinsik does not accept any responsibility for the use of this report for any purpose other than as specifically intended by the MoH. Intrinsik does not have, and does not accept, any responsibility or duty of care whether based in negligence or otherwise, in relation to the use of this report in whole or in part by any third party. Any alternate use, including that by a third party, or any reliance on or decision made based on this report, are the sole responsibility of the alternative user or third party. Intrinsik does not accept responsibility for damages, if any, suffered by any third party as a result of decisions made or actions based on this report.

Intrinsik makes no representation, warranty or condition with respect to this report or the information contained herein other than that it has exercised reasonable skill, care and diligence in accordance with accepted practice and usual standards of thoroughness and competence for the profession of toxicology and environmental assessment to assess and evaluate information acquired during the preparation of this report. Any information or facts provided by others, and referred to or utilized in the preparation of this report, is believed to be accurate without any independent verification or confirmation by Intrinsik. This report is based upon and limited by circumstances and conditions stated herein, and upon information available at the time of the preparation of the report.

Glossary

%	percent
>	greater than
≤	less than or equal to
µg/kg bw/d	micrograms per kilograms per unit of body weight per day
µg/L	microgram per litre
µg/m^3	micrograms per cubic metre
AAQO	Ambient Air Quality Objective
AAQS	Ambient Air Quality Standard
ACGIH	American Conference of Governmental Industrial Hygienists
ADI	acceptable daily intake
AHW	Alberta Health and Wellness
atm-m^3/mol	Henry's Law Constant in atmospheres cubic meter per mole
ATSDR	Agency for Toxic Substances and Disease Registry
BC ACF	British Columbia Agriculture in the Classroom Foundation
BC EMS	British Columbia Environmental Monitoring System
BC MOE	British Columbia Ministry of the Environment
BC OGC	British Columbia Oil and Gas Commission
BCS	Bureau of Chemical Safety
BLIERS	Base Level Industrial Emission Requirements
BMC	benchmark concentration
BMD	benchmark dose
CAAQS	Canadian Ambient Air Quality Standard
CAC	criteria air contaminants
CAL EPA	California Environmental Protection Agency
CALMET	three-dimensional meteorological data model
CALPUFF	a multi-layer, multi-species, non-steady-state puff dispersion modelling software
CAPP	Canadian Association of Petroleum Producers
CARB	California Air Resources Board
CAS	Chemical Abstract Service
CCHS	Canadian Community Health Survey
CCME	Canadian Council of Ministers of the Environment
CCS	Canadian Cancer Society
CIHI	Canadian Health Institutes Initiative
cm^2	centimetre squared
COPC	chemicals of potential concern
COPD	chronic obstructive pulmonary disease
DM	District Municipality
DNA	deoxyribonucleic acid - genetic material
e.g.	Latin "for example"
et al.	Latin for "and other authors"
etc.	Latin for "and other"
FBC	Fraser Basin Council
FEV1	forced expiratory volume in 1 second
FNFNES	First Nations Food Nutrition and Environment Survey
g/cm^2/day	gram per square metre per day
g/day	grams per day
g/mol	grams per mol (molecular weight)
H_2S	hydrogen sulphide
HHRA	human health risk assessment

HSDB	Hazardous Substances Data Bank
HVP	high vapour pressure pipeline
i.e.	Latin for “such as”
ILCR	incremental lifetime cancer risk
ISA	Integrated Science Assessment
IUR	inhalation unit risk
kg	kilogram
km	kilometers
L/day	litre per day
L/hour	litre per hour
LCR	lifetime cancer risk
LHA	local health area
LOAEL	lowest-observed-adverse-effect level
Log K_{ow}	logarithmic octanol-water partition coefficient
LRDW	Land Resource Data Warehouse
LVP	low vapour pressure pipeline
m	metres
m^3	cubic metres
m^3/day	cubic metres per day
MA DEP	Massachusetts Department of Environmental Protection
MAML	Mobile Air Monitoring Laboratory
MM	million
mmHg	millimetres of mercury (vapour pressure)
MoH	Ministry of Health
MPOI	maximum point of impingement
n/a	not applicable
NAAQS	National Ambient Air Quality Standards
NAPS	National Air Pollution Surveillance Network administered by Environment Canada
NAQS	National Air Quality Standards
NAS	National Academies of Science
NCS	Nutrition Canada Survey
NE BC	Northeastern British Columbia
NIOSH	National Institute of Occupational Safety and Health
NO_2	nitrogen dioxide
NOAEL	no-observed-adverse-effects level
NO_x	nitrogen oxides
NPRI	National Pollutant Release Inventory
NTS	National Topographic System
OEHHA	California Office of Environmental Health Hazard Assessment
OG	oil and gas
PAH	polycyclic aromatic hydrocarbons
PDF	portable document format
PDI	permissible daily intake
PEF	potency equivalency factors
PHAC	Public Health Agency of Canada
PHC	petroleum hydrocarbons
$PM_{2.5}$	fine particulate matter less than 2.5 µm in diameter
POD	point of departure
ppb	parts per billion
RfC	reference concentration
RfD	reference dose

RIVM	Netherlands National Institute of Public Health and the Environment
RQ	risk quotients
RsC	risk-specific concentration
RsD	risk-specific dose
SAG	stakeholder advisory group
SF	slope factor
SLRA	screening level risk assessment
SMR	standardized mortality rates
SO_2	sulphur dioxide
SRC	Syracuse Research Corp.
TCEQ	Texas Commission on Environmental Quality
TDI	tolerable daily intake
TEQ	toxic equivalency quotient
TPHCWG	Total Petroleum Hydrocarbon Working Group
US EPA	United States Environmental Protection Agency
VOC	volatile organic compounds
vs.	Latin “versus”
WCSB	Western Canadian Sedimentary Basin
WHO	World Health Organization

EXECUTIVE SUMMARY

The Ministry of Health (MoH) has contracted a team led by Intrinsik Environmental Sciences (Intrinsik) to complete Phase 2 of the Human Health Risk Assessment (HHRA) of oil and gas activities in northeastern British Columbia (NE BC). In addition to Intrinsik itself, the companies that make up the study team include: RWDI Air, Matrix Solutions and Skystone Engineering. The team also includes a three member Advisory Panel to provide an independent perspective on the design and approach of the Phase 2 HHRA project, and the interpretation of the results. In accordance with the terms of reference compiled by the MoH, the Phase 2 HHRA is intended to investigate the potential impact of oil and gas activities on human health in Local Health Areas 59, 60 and 81 (the Region).

This report presents the detailed HHRA component of the Phase 2 project. The objectives of this HHRA are to provide a comprehensive and focused assessment of potential health risks that may exist for people living in proximity to oil and gas activities in NE BC.

A Screening Level Risk Assessment (SLRA) was completed with the objective of guiding the scope of work for the detailed HHRA. As part of this SLRA, a qualitative risk-ranking exercise was completed for 50 different oil and gas emission scenarios. From this analysis, two air emission scenarios were selected for further evaluation in the detailed HHRA:

1. Continuous air emissions from gas processing plants.
2. Continuous air emissions from production facilities.

These two scenarios and the numerous associated emission sources within each category are considered together to represent continuous emissions from oil and gas activity within this detailed HHRA. By combining the emissions from the gas processing plants and production facilities into a single emission scenario representing oil and gas activities, the potential influence on air quality (and consequently human health) was addressed on a cumulative basis. In addition, information regarding potential emissions from regional sources from other non-oil and gas activities was incorporated into the detailed HHRA.

The HHRA used a widely accepted approach for assessing environmental risks that has been endorsed in the past by regulatory agencies throughout Canada and across the globe. The HHRA was performed step wise following a conventional paradigm and involved the following main steps:

- Problem formulation
- Exposure assessment
- Toxicity assessment
- Risk characterization

A large study area (150 km by 176 km) was defined for the purposes of the HHRA, and was selected such that the most densely populated areas and several First Nations in the NE BC region were included, and also the most concentrated oil and gas development in the region was captured. The major communities in the study area include Fort St. John, Dawson Creek and Chetwynd, along with smaller communities and First Nation lands.

A comprehensive emission inventory of the continuously emitting oil and gas facilities was compiled for the study area. This inventory incorporated several thousand individual emission sources. In addition, to further characterize air quality on a cumulative basis and in order to compare air quality associated with oil and gas activities with those associated with non-oil and gas emission sources, two scenarios were considered in the HHRA:

1. **Oil and Gas Scenario:** includes all on-going emissions from gas processing plants and various production facilities within the HHRA study area. These sources include, but are not limited to significant emitters such as, sweet and sour gas plants, compressor stations, and fugitive emissions from tank storage.
2. **Cumulative Scenario:** includes the oil and gas sources from the oil and gas scenario, as well as emissions from background sources such as other industries (*e.g.,* forestry and mining), transportation, and community activities (*e.g.,* residential wood burning).

A total of 26 community locations were evaluated individually within the HHRA along with the maximum predicted ground-level concentrations of each chemical of potential concern (COPC) (*e.g.*, the maximum point of impingement or MPOI).

A brief review of existing health status in the region conducted as part of the HHRA revealed that there are a number of possible sensitive sub-populations in the area.

To account for potential differences in exposures between individuals in the area, consideration was given to differences in exposure parameters (*e.g.* body weight, types and amounts of foods consumed) between age groups and community type (*e.g.*, residents in Aboriginal, rural/agricultural, or more urban communities).

Results were presented and described for inhalation on a short-term and long-term basis, and for all possible routes of exposure on a long term basis. The predicted risk estimates involved the comparison of estimates of exposure with health-based exposure limits developed by various regulatory organizations (*e.g.,* Health Canada, United States Environmental Protection Agency, World Health Organization). Separate assessments were completed for short-term and long-term exposures, and for carcinogenic and non-carcinogenic COPC.

A brief summary of the results is as follows:

- In general, the predicted short-term air concentrations of the COPC were less than their health based exposure limits. As well, the potential combined risks of these COPC were not predicted to result in adverse health effects in people living or visiting the study area. However, the predicted exposures at some locations were found to exceed exposure limits for certain individual COPC (acrolein, formaldehyde, NO_2, SO_2, $PM_{2.5}$) and the mixtures that these COPC were part of (the eye, nasal and respiratory irritants). The exceedances for formaldehyde, NO_2 and SO_2 were found to be attributable to Oil and Gas emission sources, with some contributions from other sources in the area. Due to the rare nature of these exceedances and the margin of safety built into the HHRA, these exposures are not expected to result in adverse health effects.
- Overall, long-term inhalation exposures to the COPC were predicted to be associated with a low potential for adverse health effects. For fine particulate matter ($PM_{2.5}$), exceedances of the BC Ambient Air Quality Objective were predicted for only the Cumulative Scenario at two remote locations where people are unlikely to be regularly exposed. For formaldehyde, potential cancer risks were predicted for a remote location

in close proximity to an oil and gas site. However, further analysis of this exceedance indicating that the probability for people to be exposed to formaldehyde concentrations at the predicted level over a lifetime was very low. When the potential combined, additive effects of the COPC were evaluated, nasal and respiratory irritant mixtures were predicted to have elevated risk estimates. However, given the locations of where the maximum concentrations for these chemicals were expected to occur (*e.g.* formaldehyde), and the degree of conservatism incorporated into the assessment, the potential mixture risks were determined to have a low potential for adverse health effects.

- In the assessment of potential exposures to the COPC that people in the area might receive over the long term through the consumption of locally-grown foods, drinking water, *etc.*, it was determined that the potential for adverse human health effects is low.

The overall findings of the detailed HHRA of oil and gas activity in NE BC suggest that, while there is some possibility for elevated COPC concentrations to occur at some locations, the probability that adverse health impacts would occur in association with these exposures is considered to be low.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

APPENDICES

1.0 INTRODUCTION

In response to concerns expressed by residents of northeastern British Columbia (NE BC), the British Columbia Ministry of Health (MoH) commissioned a human health risk assessment (HHRA) with a focus on the potential impacts of oil and gas activity on human health. The HHRA scope of work was segregated into three phases by the MoH:

- Phase 1 HHRA. Identification of Health Concerns Relating to Oil and Gas Development in Northeastern BC. Completed in March 2012 by the Fraser Basin Council.
- Phase 2 HHRA. Human Health Risk Assessment of Northeastern British Columbia Oil and Gas Activity.
- Phase 3 HHRA. Communication of overall results. Timeline: To Be Determined.

The MoH has contracted a team led by Intrinsik Environmental Sciences (Intrinsik) to complete Phase 2 of the HHRA of oil and gas activities in NE BC. In addition to Intrinsik, the companies that make up the study team include: RWDI Air, Matrix Solutions and Skystone Engineering. The team also includes a three member Advisory Panel to provide an independent perspective on the design and approach of the Phase 2 HHRA project, and the interpretation of the results.

This report presents the detailed HHRA task for Phase 2, the objectives of which are to understand what potential health risks exist for residents and First Nations members in NE BC living in proximity to oil and gas activities.

In accordance with the MoH terms of reference, the Phase 2 HHRA is intended to investigate the potential impact of oil and gas activities on human health in Local Health Areas 59, 60 and 81 (the Region).

Based on the decisions made in the Screening Level Risk Assessment (SLRA) (Intrinsik 2014a), the HHRA involves a comprehensive and focused assessment of the potential adverse health risks in relation to oil and gas activity in NE BC. Two air emission scenarios have been selected for inclusion:

1. Continuous emissions from gas processing plants.
2. Continuous emissions from production facilities.

These two scenarios, and the numerous associated emission sources within each category are considered together to represent continuous emissions from oil and gas activity within this detailed HHRA. By combining the emissions from the gas processing plants and production facilities into a single emission scenario, the potential influence on air quality (and consequently human health) was addressed on a cumulative basis. In addition, information regarding potential emissions from regional sources not directly associated with oil and gas activities in the region.

No water emissions scenarios were carried forward from the SLRA (Intrinsik 2014a) for quantitative assessment in the HHRA. Additional information regarding water emissions scenarios and the rationale behind their exclusion from the detailed HHRA is provided in the SLRA (Intrinsik 2014a). Several water-related scenarios are also discussed as part of the review of regulatory frameworks (Intrinsik 2014b).

This HHRA is one of a series of reports generated as part of the Phase 2 HHRA project, and is intended to capture the oil and gas emissions in the region that pose the greatest potential risk to human health. The objectives of the HHRA do not include:

- A comprehensive assessment of work completed to date;
- The study of epidemiology, or explore potential cause-effect relationships between exposure and health effects or diseases in the region;
- A Health Impact Assessment or an evaluation of social determinants of health in the region (health care, addictions, mental health, *etc.*); or,
- Detailed discussions of regulations or industry practices. This type of review will be completed as part of the regulatory review component of the Phase 2 HHRA project.

This report has been organized into the following sections:

- Work completed to date – a brief description of the work that has been completed by the study team
- Assessment of potential air quality impacts from oil and gas activity in NE BC
- Overview of the human health risk assessment processSummary
- Next steps

2.0 WORK COMPLETED TO DATE

This detailed HHRA has been formulated based on the outcome of several information collection steps that have been completed by the study team. Some of the key findings associated with this work are presented in Sections 2.1 to 2.4.

2.1 Summary of Phase 1 Human Health Risk Assessment

The Fraser Basin Council (FBC 2012) completed Phase 1 of the HHRA project, which included a public engagement process to identify issues of concern surrounding human health and potential changes in land, air, drinking water and food quality and preparation of a report outlining their findings. Area residents, including First Nations residents, were included in the Phase 1 work. Public engagement activities were carried out from mid-January to early March 2012 in order to provide the public, governments, organizations, and other stakeholders with the opportunity to voice their concerns regarding current and future oil and gas development in NE BC.

A high-level summary of some of the key concerns raised in the Phase 1 report are presented in Table 2–1, along with a discussion of how the issues are addressed in the Phase 2 HHRA Project.

Table 2–1 Summary of Key Issues Identified in the Phase 1 HHRA Report and Discussion of Integration into the Phase 2 Human Health Risk Assessment

Issue Identified in Phase 1 Report	Issue Addressed in Phase 2 HHRA Project?	Comment
1. Personal Health Issues		
a. Lung related issues such as asthma and bronchitis	Yes	The Phase 2 HHRA Project evaluates the potential health impacts associated with the chemical emissions from oil and gas activity on human health, including potential respiratory health impacts. Individuals potentially sensitive to the effects of airborne chemicals, such as asthmatics and the chronically ill, are considered in the detailed HHRA. However, the HHRA does not evaluate whether any specific diseases in the region have been caused by exposure to specific contaminants or activities.
b. Cancer	Yes	The Phase 2 detailed HHRA evaluates the potential health impacts associated with the chemical emissions from oil and gas activity on human health, including potential cancer risks associated with exposure. However, the HHRA does not evaluate whether any specific diseases in the region have been caused by exposure to specific contaminants or activities.
c. Quality of life for local residents	No	The Phase 2 HHRA Project focuses on health issues potentially related to chemical exposures associated with oil and gas activity.
d. Stress and sleep deprivation	No	Non-chemical related health determinants are beyond the scope of the Phase 2 HHRA Project.
2. Environmental Pathways of Exposure		
a. Emissions from oil and gas activity and potential impact on human health	Yes	The Phase 2 HHRA Project evaluates the potential health impacts associated with the chemical emissions from oil and gas activity in NE BC.
b. Acute and chronic exposure to H_2S, SO_2 and other emissions	Yes	Both short-term and long-term exposures to chemicals of concern associated with oil and gas activity are evaluated in the detailed HHRA, including H_2S and SO_2.
c. Potential impacts of SO_2 and H_2S on sensitive or susceptible individuals	Yes	Individuals who are potentially sensitive to the effects of airborne chemicals, such as asthmatics and the chronically ill, are considered.
d. Characterization of chemicals of concern associated with oil and gas activity	Yes	Literature reviews were used to identify chemicals of potential concern associated with oil and gas activities that are relevant to NE BC. Information was also collected from local health authorities, provincial ministries, and industry in order to identify chemicals of concern for the detailed HHRA.
e. Effects of fugitive emissions on human health	Yes	Consideration was given to potential fugitive emissions of chemicals of concern in the Project, where information is available.
f. Contamination of water from activities associated with oil and gas development, including the construction, operation, spills, and waste management practices of industry	Yes	Consideration was given to potential chemical contamination of surface and ground water in association with oil and gas activity, where adequate information was available.

Issue Identified in Phase 1 Report	Issue Addressed in Phase 2 HHRA Project?	Comment
g. Potential impacts of oil and gas emissions on locally grown food, including agricultural products, fish and wildlife	Yes	The Phase 2 HHRA Project considers the potential chemical contamination of local soil, water, traditional and country food sources in association with oil and gas activity, where adequate information is available. The assessment of the existing health or potential impacts of oil and gas activities on crop, livestock or wildlife health specifically are beyond the scope of the assessment.
h. Cumulative impacts of emissions from various sources in the area (transportation, industry, *etc.*)	Yes	The Phase 2 HHRA Project focused on emissions associated with oil and gas activity. Relevant baseline, measured concentrations of chemicals in environmental media from the area were considered where possible and relevant. The full extent of the cumulative effects assessment was determined in the SLRA and the detailed HHRA.
i. Emissions from oil and gas activity and potential impact on livestock health	No	The Phase 2 HHRA Project focuses only on the potential impacts to human health. Effects on livestock and agricultural operations are outside the Project scope.
j. Emissions from oil and gas activity and potential impact on ecological health (wildlife, aquatic receptors, *etc.*)	No	Effects on ecological receptors are outside the scope of the Project.
k. Impacts of oil and gas activity on water quantity (*i.e.,* use of large volumes of water)	No	The potential non-chemical impacts on water sources and water availability are beyond the scope of this Project.
l. Impact on agricultural operations or adherence to Environmental Farm Plan guidelines	No	The impact of oil and gas activity on agricultural operations is outside the scope of this Project.
3. Related Environmental Issues		
a. Explosions and accidental releases	Yes	Consideration was given to potential human health impacts associated with accidents, in relation to oil and gas activity in the area as part of the Project. This was addressed in the Screening Level Risk Assessment (SLRA) and in the Review of the Regulatory Framework.
b. Impacts on water bodies and soil from spills, leaks, and waste disposal on health	Yes	The potential impacts of accidental releases and spills of water used by oil and gas activities in the region was considered in this Project, specifically in the SLRA and the Review of the Regulatory Framework.
c. Destruction of wildlife and aquatic habitat	No	An evaluation on habitat impacts is beyond the scope of the Phase 2 HHRA Project.
d. Increased traffic	No	The Phase 2 HHRA focused on chemical emissions associated with oil and gas activity and potential human health effects.
e. Potential impact of noise and light pollution on health	No	The assessment of non-chemical related health determinants are beyond the scope of the Phase 2 HHRA.
4. Changes to Community		
a. Impacts on access to community services, including health care and social services	No	The assessment of non-chemical related health determinants are beyond the scope of the Project.
b. Increased impacts due to growth on municipal and regional infrastructure (water and wastewater facilities, housing, security, parking, transportation issues)	No	The assessment of non-chemical related health determinants are beyond the scope of the Project.

Issue Identified in Phase 1 Report	Issue Addressed in Phase 2 HHRA Project?	Comment
c. Impact of oil and gas activity on social endpoints (addictions, family structure, mental health)	No	The assessment of non-chemical related health determinants are beyond the scope of the Project.
5. Oil and Gas Operational Issues		
a. Impacts of oil and gas exploration activities	Yes	The potential impacts of exploration activities were considered in the Phase 2 HHRA Project. The extent to which these will be assessed was determined in the SLRA.
b. Potential for increased road accidents due to increased traffic, including accidents involving hazardous waste vehicles	No	The evaluation of traffic patterns and the potential for accidents is beyond the scope of this assessment.
c. Impact of physical aspects of oil and gas activity such as facility density, potential for earthquakes	No	An evaluation of these types of physical hazards is beyond the scope of the HHRA, as the Phase 2 HHRA focused on chemical emissions associated with oil and gas activity and potential human health effects.
6. Institutional Framework		
a. Evaluation of regulations and policies relating to the oil and gas activity in NE BC	Yes	The Phase 2 HHRA includes a regulatory review of existing relevant environmental frameworks (*i.e.*, the Review of the Regulatory Framework).
b. Recommendations regarding emission management and reduction	Yes	The potential development of such recommendations will be based on the findings of the detailed HHRA and the regulatory review Where appropriate, recommendations regarding emission management strategies may be made.
c. Evaluation of emergency response planning in the area and impacts on people	Yes	The existing emergency response protocols and practices will be considered in the Phase 2 HHRA, specifically in the Review of the Regulatory Framework.
d. Communication of environmental monitoring information to the public	In part	As part of the Review of the Regulatory Framework, existing regulations will be evaluated and recommendations made based on the findings of the review.
e. Communication between operators, health authorities and the public with respect to operations, emergency response, enforcement and compliance activities	In part	As part of the Review of the Regulatory Framework, existing regulations will be evaluated and recommendations made based on the findings of the review.
f. Establishment of a monitoring program or framework for environmental media (air, water, soil)	In part	Developing specific environmental quality monitoring programs are outside the scope of the Phase 2 HHRA Project. However, based on the findings of the Phase 2 HHRA, recommendations regarding the development and implementation of monitoring programs may be made.
g. Tracking and reporting of adverse health effects	No	The Phase 2 HHRA Project does not include the collection and tracking of specific adverse health effects of people in the area.

2.2 Summary of Phase 2 Direction Document

This document represented a revised work plan for the Phase 2 HHRA, with additional depth on some topics. The information covered in this document included:

- Review of Phase 1 report by Fraser Basin Council (2012)
- Discussion of perceived health concerns from the Phase 1 report in relation to the Phase 2 HHRA project
- Discussion of oil and gas activity in NE BC
- Existing health status and available health data
- Existing environmental data for the region
- Potential chemicals and exposure pathways of interest

2.3 Review of the Nature and Extent of Oil and Gas Activity in Northeastern British Columbia

Oil and gas production has been an important and prevalent source of economic development in western Canada since the early part of the 20th century. Traditional products of the oil and gas industry have included oil, natural gas, natural gas liquids and sulphur extracted from the large geological feature referred to as the Western Canadian Sedimentary Basin (WCSB). Technological advances in well drilling, completions and stimulation have allowed the exploitation of less traditional reservoir types, including shales and 'tight' (geological formations having naturally low primary permeability) clastic formations.

Northeastern BC contains the western edge of the WCSB, and as such, has seen considerable oil and gas activity. Much of BC's production has been conventional gas, with approximately 25% of Canada's gas production coming from NE BC (Center for Energy 2013). Gas production in BC continues to increase over time and in 2011, 40.5 MM m^3 was produced (BC OGC 2013). Traditional gas reserves have been developed in predominantly clastic formations of Cretaceous and Triassic age, as well as the Devonian-aged carbonates (Mossop and Shetsen 1994).

In 2008 through 2010, significant increases in BC's gas reserves were booked, or recorded as assets by oil and gas companies. These increases are primarily 'unconventional' reserves contained within Devonian-aged shales in the Horn River Basin and the Triassic Montney tight gas trend (BC OGC 2013). The primary drivers for these new reserves have been the widespread drilling of horizontal wells in these shales, combined with multi-stage hydraulic fracturing as a production stimulation method. Together, production from these two plays accounted for approximately 40% of BC's 2011 gas production (BC OGC 2013).

Significant oil production is also sourced in NE BC; however, this production peaked in 1998 at over 2.5 MM m^3, and has declined steadily to slightly more than 1.1 MM m^3 in 2011. Oil reserves have also declined since 2001.

Both sweet and sour oil and gas resources in conventional and unconventional forms are present in NE BC, and thus are of potential relevance to the Phase 2 HHRA project.

2.4 Summary of Literature Review

A screening level literature review was completed in spring 2013 by the study team (Intrinsik 2013), consisting of a comprehensive search of reports that involved the assessment of potential health effects associated with oil and gas development. This search and review was designed to be consistent with the *Cochrane Handbook for Systematic Reviews of Interventions* (Cochrane 2008), and included a critical review of the available scientific peer-reviewed literature as well as 'grey' literature (reports published by government, academia, non-profit organizations or industry). A comprehensive list of search terms was developed by Intrinsik and sent to a professional medical librarian. The abstracts collected during this search were critically reviewed. Only documents that met pre-defined inclusion criteria were selected for further review. The inclusion criteria were as follows:

- Published in English
- Published 1990 to present
- Human health study – community or occupational studies

The original draft document included a total of 27 peer-reviewed articles and 11 documents from the grey literature, all of which met the study criteria. Additional documents were suggested by the Advisory Panel based on their review of the draft, and were subsequently incorporated into the final report.

The general conclusions of the literature review were as follows:

- In the studies that evaluated cancer morbidity and mortality, the types of cancer most frequently reported included bladder, kidney, acute myelogenous leukemia, other leukemias and melanoma. Some variation was observed in results in relation to the types of effects observed. There is an apparent need for additional studies with case-control or cohort study designs to evaluate the potential association between cancer incidence and oil and gas activity.
- There is an overall lack of published research regarding respiratory health effects and oil and gas activities. Although there is a wealth of information regarding hydrogen sulphide (H_2S), sulphur dioxide (SO_2) and exposure to petroleum hydrocarbons from downstream oil and gas activities (*i.e.,* refineries), there is limited information with respect to other chemicals, and emissions from upstream oil and gas facilities and respiratory health.

Other health outcomes of interest reported within the key studies included autoimmune diseases, reproductive, cardiovascular and neurological effects. Those studies that were identified in the literature review that evaluated autoimmune diseases and cardiovascular effects were sometimes of low quality.

The diseases of concern in relation to upstream oil and gas activity that needed to be considered in the Phase 2 HHRA, in order of priority, were identified as:

- Respiratory diseases and cancers
- Reproductive, neurological and acute (short-term, mild, transient) effects
- Autoimmune disease and cardiovascular effects

The majority of the studies evaluated as part of this review lacked information regarding exposure pathways of interest, exposure concentrations, or chemicals of potential concern (COPC). As such, the results of the literature review did not provide any recommendations regarding chemicals or exposure pathways of concern in relation to oil and gas activity and human health.

The findings of the review did not provide any information regarding specific types of oil and gas activities or scenarios that should be considered further.

2.5 Summary of the Screening Level Risk Assessment

The intention of the SLRA was to identify the potential oil and gas related emission sources in the region that presented the greatest potential risk to human health, and to provide a means of prioritizing scenarios for a quantitative assessment in the detailed HHRA. In order to do so, a comprehensive qualitative analysis of a spectrum of potential emissions sources to air and water was completed, with the aim of identifying emission sources that pose the greatest potential risk to people in NE BC. This screening exercise was conducted through the use of matrices, statistics and professional judgment. Particular consideration was given to potential adverse health impacts, likelihood of occurrence, scale of potential impacts, and magnitude of potential exposures in order to identify the emission sources and scenarios presenting the greatest potential risk.

A study area for the detailed HHRA work was proposed within the SLRA, taking into consideration a number of different factors, including:

- Population density
- Estimates of emission density of Criteria Air Contaminants (CACs) and total Volatile Organic Compounds (VOCs)
- Known locations of wells, pipelines, gas plants, and oil and gas processing facilities (*e.g.*, batteries and compressor stations)

The identified study area is centred on Fort St. John, and also includes several of the larger communities in the region: Dawson Creek, Pouce Coupe, Hudson's Hope and Taylor, the Blueberry and Doig River First Nations, and the northern boundary of the Tumbler Ridge area. This area represents the most densely populated area in the region under study, as well as the area with the highest density of continuous emission sources. Additional information regarding this map was provided in Section 6.0 of the SLRA (Intrinsik 2014a).

The SLRA identified a series of potential emission scenarios related to oil and gas activity in the region that could present risks to human health. Risk-based matrices were developed in order to qualitatively evaluate and rank the potential risks associated with the different air emission and water release scenarios in the study area. Each of the identified scenarios were assessed according to its potential exposure and health hazard, and relative numerical rankings or 'scores' were assigned to each scenario to qualitatively describe potential human health risk.

The exposure scores were based on a combination of the likelihood of an emission/release event occurring, the duration and areal extent associated with that event, and the overall magnitude of the event's exposure. On the hazard side, the score was based entirely on the potential health impact associated with a particular event. To the greatest extent possible, numerical risks were based on actual data, predominantly as these relate to the likelihood of

events occurring. However, when such data were unavailable, the scoring system relied on a combination of past experience, information retrieved from oil and gas related HHRAs and exposure studies, and professional judgment. The risk matrices provided the relative 'score' of the potential health risks on the basis of varying types of oil and gas activity. However, whether or not an exposure scenario ultimately would be included in the HHRA depended not only on the significance of the risk (*i.e.,* its relative numerical ranking in the matrix), but also on the availability and adequacy of environmental data that allows for the health risks to be quantitatively assessed on a regional scale.

Based on the findings of the risk matrix for air emissions, two scenarios emerged as the top priorities for further evaluation in the detailed HHRA:

1. Continuous emissions associated with gas processing plants
2. Continuous emissions from oil and gas production facilities (including batteries, storage tanks, compressors, dehydrators, *etc.*).

The estimated risks for all the other emission scenarios considered in the risk matrix were orders of magnitude less than the two continuous (or ongoing) emission scenarios.

The SLRA completed for water emission sources determined that the two scenarios with the highest numerical rankings included:

1. In-ground fluid pits and flare systems, and
2. Pipeline ruptures.

The scenarios relating to historical and current fluid management practices are associated with a relatively lower consequence to human health, however, the potential likelihood of occurrence is higher due to the number of historical and active wells. The potential health risks associated with these site-specific issues can only be assessed on a case-by-case basis using measured, site-specific data. There are a number of data gaps in publicly available information regarding groundwater and surface water resources in NE BC that may impact the ability to complete site-specific assessments.

The pipeline rupture scenario is associated with a relatively lower likelihood of occurrence, but a higher potential consequence to human health in the event of a release. However, these types of release events can be mitigated through emergency planning and response practices, communication, and site-specific activities (such as evacuation or closure of drinking water intakes) to reduce the potential for human exposure. As a result, these scenarios were not carried forward into the detailed HHRA, which is intended to have a regional focus (as opposed to a local or site-specific focus).

In addition, a process for further evaluation of oil and gas sites on a site-specific basis that could be used to assess potential water-related human health risks was presented in the SLRA. The described process takes into consideration an existing site classification system from the BC Oil and Gas Commission (OGC) that could be utilized for this purpose.

The justification for the technical aspects of the detailed HHRA was provided within the SLRA. Based on the selected emission scenarios, a proposed approach to the detailed HHRA was formulated, following an approach consistent with those established by regulatory organizations such as Health Canada, the United States Environmental Protection Agency and the World

Health Organization. Based on the findings of the Literature Review (Intrinsik 2013) and an additional review completed as part of the SLRA (see Intrinsik 2014a, Appendix D), a list of representative chemicals that are known to be associated with the selected emission sources and have the potential to, at high enough concentrations, cause a number of the health effects identified in the Literature Review were included in the detailed HHRA. A number of potential exposure pathways were identified, and conceptual models were constructed.

3.0 ASSESSMENT OF POTENTIAL AIR QUALITY IMPACTS FROM OIL AND GAS ACTIVITY IN NE BC

3.1 HHRA Overview and Description of Methods

The overall approach that was followed in performing the detailed HHRA is outlined below, including a generic description of the methods followed and the various steps and guiding principles involved.

The detailed HHRA component of the Phase 2 HHRA followed methods that are consistent with those developed by:

- Health Canada (Health Canada 2012, 2010, 2009)
- Canadian Council of Ministers of the Environment (CCME 2006)
- United States Environmental Protection Agency (US EPA 1989; US EPA OSW 2005)
- BC Ministry of the Environment (BC MOE 2012a). Technical Guidance on Contaminated Sites. October 2012
- Alberta Health (AHW 2011)

The HHRA uses a widely accepted approach for assessing environmental risks that has been endorsed in the past by regulatory agencies throughout Canada and across the globe. The HHRA was performed step-wise following a conventional paradigm (see Figure 3-1) and involved the following five main steps:

- Problem formulation
- Exposure assessment
- Toxicity assessment
- Risk characterization
- Risk management

A general overview of each step is provided in Section 3.1.2 below. Specifics are provided in subsequent sections.

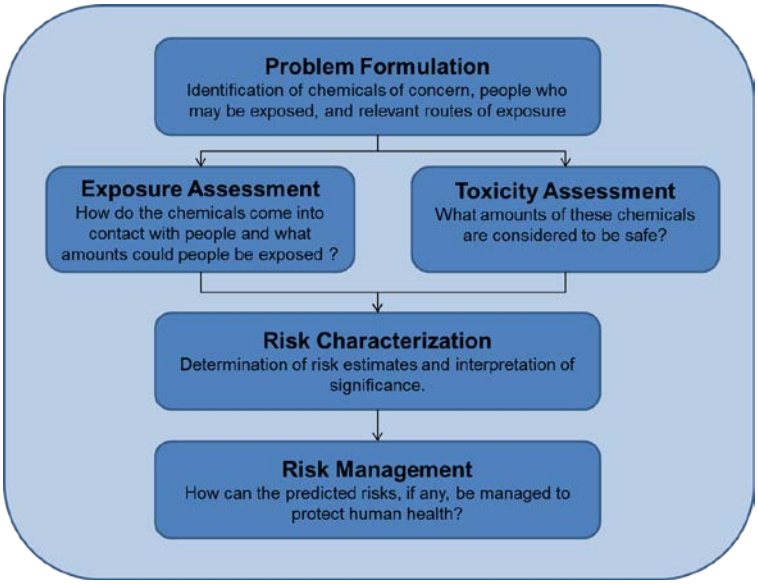


Figure 3-1 Human Health Risk Assessment Paradigm

3.1.1 Guiding Principles

As stated earlier, the detailed HHRA is being conducted in order to identify and understand the potential health risks that could be presented to people in the study area from chemical exposures resulting from the oil and gas activities identified in the SLRA. The assessment required consideration of both the toxic properties of the chemicals as well as the amounts of the chemicals to which people might be exposed. For the purposes of the HHRA, the term 'risk' was used in the following context:

RISK	=	TOXICITY	×	EXPOSURE

Certain guiding principles common to the study of the potential health risks presented by chemicals, regardless of source, were embraced by the work. These principles are:

- All chemicals, regardless of type or source, possess some degree of intrinsic toxicity. This principle is easily appreciated for chemicals such as arsenic, cyanide, strychnine and other well-known poisons. However, the principle applies equally to over-the-counter medications (such as cough syrups, vitamin supplements and analgesics), to common food ingredients (such as granulated sugar and table salt), as well as to the chemicals associated with the oil and gas activities in NE BC. Each of these chemicals possesses the capacity to cause harm.
- The health effects caused by any chemical are dependent not only on the intrinsic toxicity of the substance (*i.e.,* the capacity to cause harm), but equally on the exposure or dose of the chemical that is received. This principle forms the basis of the statement: "*All substances are poisons … there is none which is not a poison … the right dose differentiates a poison and a remedy*", first penned by Paracelsus more than five centuries ago. Irrespective of the intrinsic toxicity of a chemical, health effects will not occur in the absence of exposure.
- With very few exceptions, the intrinsic toxicity of a chemical is only expressed provided the exposure exceeds a critical threshold level. Below this threshold dose, injury does not occur since the body is capable of tolerating minor exposures as a result of detoxification, elimination and/or repair processes that act to neutralize the chemical and reduce the prospect for harm. If the threshold dose is exceeded, health effects may occur. The severity of these effects will depend on the level of exposure received, with more severe effects occurring with higher doses. This is commonly referred to as the 'dose-response' principle of toxicology.
- One possible exception to the threshold principle involves certain chemical carcinogens that act via genetically-mediated mechanisms to produce certain forms of cancer. Some regulatory agencies consider that no safe dose levels exist for these carcinogens and as such develop an acceptable or negligible (*i.e., de minimus*) risk level. The negligible risk level is commonly set at the dose where cancer risk is increased by a level of one in a hundred thousand (1 in 100,000). Some authorities also insist that a threshold dose does not exist for various morbidity and mortality outcomes that can follow exposure to some substances (*e.g.*, fine particulate matter ($PM_{2.5}$)). However, the apparent lack of a threshold level in this case is not based on mechanistic considerations, but rather simply on an inability to discern a threshold dose because of experimental limitations. In these cases, non-cancer based *de minimus* risk levels must be established.

- The health effects produced by a chemical vary depending on the amount, duration and frequency of exposure. It is important to distinguish between the health effects (and associated health risks) which may result from acute exposures of short duration *vs.* the effects which may follow longer-term exposures lasting several days to several weeks *vs.* the effects which may follow chronic exposures lasting several months or years, even up to a lifetime.
- The toxicity of any chemical is dependent on its molecular structure. Within limits, chemicals having similar structures will produce similar toxic responses. This principle allows the health effects of a chemical of unknown toxicity to be predicted on the basis of the health effects known to be caused by a second 'surrogate' chemical of similar molecular structure. The term 'read across' has been coined to describe the process by which the health effects data for the surrogate chemical are applied to other structurally-related compounds.

A further guiding principle concerns the uncertainty that can surround the prediction of any health risks, regardless of type or source. This uncertainty can take several forms, including: uncertainty due to lack of information; uncertainty due to the variability intrinsic to living systems; and, uncertainty due to experimental and measurement error. These and other forms of uncertainty can confound the interpretation of the meaning and significance of any health risks that might be revealed by the work. By convention, the uncertainty is accommodated, in part, through the use of assumptions which embrace a high degree of conservatism and are often intentionally selected to represent worst-case or near worst-case conditions. Using this approach, any health risks identified by the assessment are unlikely to be understated, but may be considerably overstated.

3.1.2 The Risk Assessment Paradigm

As described, the HHRA paradigm (see Figure 3-1) is recognized world-wide, and its use has been endorsed by a number of leading federal and provincial regulatory agencies, including Health Canada, Environment Canada, the Canadian Council of Ministers of the Environment (CCME), the US Environmental Protection Agency (US EPA), and the BC Ministry of Environment (MOE). Highlights of the five steps of the paradigm are outlined below.

Step One – Problem Formulation

This step is concerned with defining the scope and nature of the assessment, and setting practical boundaries on the work such that it is directed at the principal areas of concern. The Problem Formulation is focused on four major areas:

- **Identification of the exposure scenarios to be examined.** The scenarios refer to the specific conditions by which people could be exposed to chemicals released, discharged or emitted into the environment, with consideration given to the sources of the chemicals, the nature and duration of the releases (*i.e.*, intermittent *vs.* continuous), and other factors affecting the types and levels of exposure that could be experienced.
- **Identification of the chemicals of potential concern (COPC) to be examined.** The COPC refer to the chemicals contained in the releases, discharges or emissions associated with each Exposure Scenario that may be of concern from a health perspective. Selection of the COPC is based, in part, on the toxicity of the chemical, its rate of release, its regulatory status, and the nature of the health endpoints affected by over-exposure.

- **Identification of people who may be exposed.** Consideration is given to individuals who could be exposed to the COPC (*e.g.*, local residents, people working in the area, people visiting the area). Emphasis often is given to people who might be especially vulnerable to chemical exposures, including infants, young children, the elderly and individuals with compromised health.
- **Identification of the exposure pathways to be examined.** The pathways refer to the avenues and modes by which the people could be exposed to the COPC. The pathways often are distinguished as being primary or secondary in nature. The former pathways are dictated by the manner in which the COPC are emitted, discharged or released into the environment and represent direct avenues by which the chemicals can reach individuals (*e.g.*, breathing in an air-borne chemical); whereas, the latter pathways represent secondary routes by which the COPC might reach people depending on the substance's environmental fate and behaviour (*e.g.*, exposure via the food chain). The mode of exposure refers to the actual manner in which the substance can enter the body, with the principal modes being inhalation, ingestion and dermal contact.

Details surrounding each of the above items as it relates specifically to the detailed HHRA can be found in Section 3.2.

Step Two – Exposure Assessment

This step is concerned with estimating the level of exposure to the COPC that might be received by individuals *via* the various exposure pathways. The process of exposure assessment often relies on one or more forms of predictive modelling to arrive at the exposure estimates, with specific reliance on air dispersion modelling in the case of air-borne contaminants. Factors that can influence the amount of exposure received, such as the behaviour of the COPC in the environment and the characteristics of individuals who may be exposed (*e.g.*, body weight, breathing rate) are integrated into the assessment. Apart from estimating the exposures received from the selected air emission sources, consideration also is often given to background exposures contributed by existing sources of the COPC to arrive at estimates of cumulative exposures.

Distinction is made between exposures of a short-term (or 'acute') nature extending over a few minutes to several hours *vs.* long-term (or 'chronic') exposures lasting for several months or years, possibly up to a lifetime.

Step Three – Toxicity Assessment

This step of the risk assessment process is concerned with identifying and understanding the potential health effects that can be caused by each of the COPC (acting either singly or in combination), and the conditions under which the effects can occur. This step revolves around the guiding principle that the dose of a chemical largely dictates the nature and severity of any health effects that might be observed. Careful consideration is given to understanding the influence of the amount, duration and frequency of exposure on the nature and severity of the health effects (*i.e.,* the dose-response relationship) that may exist in humans and other species. A principal outcome of this step is the determination of exposure limits for the COPC, which refer to the safe levels of exposure (*i.e.,* the dose of the COPC that would not be expected to cause harm). The limits are typically based on guidelines, objectives or standards established by government agencies charged with the protection of public health, and incorporate a high margin of safety to ensure the protection of even vulnerable members of the population.

The choice of exposure limits to be used in the assessment typically requires that several criteria be met:

- Health-based (as opposed to being based on endpoints such as protection against the detection of unpleasant odours or ecological effects).
- Developed by a reputable government or scientific authority with the requisite technical knowledge.
- Adequately protective of the health of the general population, including infants, children, the elderly and people with compromised health.
- Documentation supporting the exposure limit should be available outlining the basis of its selection and the manner in which it was derived.

Step Four – Risk Characterization

This step is concerned with quantifying the potential health risks that could be presented to individuals in the area by comparing the exposure estimates determined as part of Step Two (Exposure Assessment) to the corresponding exposure limits determined as part of Step Three (Toxicity Assessment). If the exposure estimates are shown to be less than the exposure limits, the likelihood of adverse health impacts is considered to be low. On the other hand, if the exposure estimates are determined to be greater than the exposure limits, some prospect for adverse health impacts exists. The interpretation of the significance of the exceedances involves the consideration of a number of factors, including the magnitude of the exceedance, the underlying basis of the selected exposure limit, and the overall degree of conservatism incorporated into the assessment. More detailed information as to how risks were interpreted in the detailed HHRA is provided in Section 3.5 (Risk Characterization).

Step Five – Risk Management

This step is directed at identifying options for eliminating or minimizing any unacceptable health risks through implementation of engineering, operational, administrative and/or other mitigation measures that will act to reduce the potential exposures that might be received by the people in the area. As well, this can include options for measuring or monitoring health risks associated with oil and gas activity in NE BC.

As part of the Phase 2 HHRA project, the study team will be formulating several recommendations for consideration by the MoH based on the outcomes of all deliverables associated with the project, of which the detailed HHRA is only one. As a result, specific recommendations regarding risk management have not been made as part of the detailed HHRA report, but will be presented as part of a separate report.

Additional Considerations

Several additional factors were considered in the design, conduct and reporting of the health risk assessment and the interpretation of the outcomes. An appreciation of these factors is necessary for understanding the strengths and limitations of the process. They are:

- Virtually all health risk assessments are encumbered by uncertainty, the source of which relates, in part, to: i) the variability in responses to chemical exposures that can exist within and between species; ii) the reliance on predictive models to estimate the

exposures that might be experienced by people in the area; iii) the gaps in information that often exist concerning the dose-response characteristics of the COPC; and, iv) the variability in the quantity and quality of data used to derive the exposure estimates and exposure limits that form the basis of the risk calculations.

- To accommodate the uncertainty, conservatism is invariably incorporated into the assessment as a means to avoid overlooking or understating any potential health risks. The conservatism is commonly introduced through a combination of: i) reliance on worst-case exposure scenarios; ii) use of conservative assumptions with respect to exposure modelling parameters; and, iii) use of uncertainty factors in the derivation of the exposure limits to enhance the level of protection. These uncertainty factors are meant to accommodate the variability in the responsiveness to chemical exposures that can exist between and within species, as well as the uncertainty that can surround the type and nature of the responses themselves. The use of the uncertainty factors results in the exposure limits corresponding to concentrations that are well below the levels of the chemicals known to cause adverse health effects, even among the most sensitive species. Although the conservatism is meant to offset the uncertainty intrinsic to the assessment, it can contribute to exaggeration of the toxic potencies of the COPC and/or the exposures that might be received by the people in the HHRA study area. In fact, the compounding of these conservative elements often can lead to risk estimates that represent strictly hypothetical constructs of questionable practical meaning. Accordingly, if adverse health impacts are indicated by the assessment, the need exists to interpret the significance of the outcomes in the context of the conservatism embraced by the work. The conservatism can extend to all facets of the assessment, including (but not limited to) the choice of exposure scenarios, discrete locations, exposure pathways, and exposure limits.

3.2 Problem Formulation

Within the context of the Phase 2 HHRA, the SLRA served as the foundation for the detailed HHRA, and included a preliminary Problem Formulation discussion. This section has been updated and expanded as part of the detailed HHRA.

Work specific to the detailed HHRA began with Step One (*i.e.*, Problem Formulation) of the paradigm. As already indicated, this step was concerned with defining the scope and nature of the assessment, beginning with determining the exposure scenarios of interest, and then identifying the COPC, locations, and exposure pathways that were assessed under each scenario. Details are given below.

3.2.1 Description of HHRA Study Area

The HHRA study area was developed using a multi-step process as part of the SLRA (see Intrinsik 2014a, Section 6.2). A general outline of this process is provided below, with additional details available in the SLRA.

Step One - Map Construction

The starting point for the selection of a study area for the HHRA involved the construction of a map of NE BC using ESRI's ArcGIS® software. This layered map identified cities and town sites across the region, along with First Nations communities. The map also illustrated the various oil and gas infrastructure densities, including pipelines, wells, batteries and gas plants. In addition,

the map could be used to characterize the different emission densities for a number of chemicals or chemical groups according to the highlighted emission sources. This is described further below. The map was distributed to the Advisory Panel and Steering Committee as a Geo-referenced PDF.[1]

The map focused on the geographic area covered by the three local health areas addressed in the Phase 2 HHRA (LHA 59, 60 and 81). For screening level purposes, a grid resolution corresponding to the National Topographic System (NTS) was applied.[2]

Step Two - Consideration of Population Density

In order to determine where the most populated areas within the region are, and to aid in the evaluation of the relative proximity of the population to oil and gas activities, a separate layer of the map was constructed. Population data for the region from the 2011 Census were obtained from Statistics Canada (2013, 2011), including for the First Nation communities in the region. Population density was incorporated into the map by first determining the population density of each census subdivision (population divided by surface area), and then an average population density was calculated for each grid block by taking an average of the population densities for all census subdivisions that resided in each block. The outcome of this was a map with a layer that clearly showed the population densities using coloured grid blocks.

Step Three - Locations of Various Oil and Gas Activities

In an effort to gain a clear understanding of where the majority of oil and gas activities in the region were located, the locations of wells, pipelines, batteries, gas plants, and other facilities (*e.g.,* compressor stations) and their corresponding emissions and emission densities were incorporated into the map, and were layered over the geographical and population map sheets to visually provide a sense of the overall distribution of existing oil and gas infrastructure in the region. Oil and gas infrastructure data were queried using a commercially available database ('IHS Energy's Energy Information, Software & Solutions EGIS', IHS Energy 2013a,b; n.d.), which is compiled from various government and industry sources on facilities, wells, and pipelines. Infrastructure densities were determined by finding the quantity in each grid block and dividing by the surface area of the block (in square kilometres).

Pipelines were split into two main categories based on their H_2S content (sweet being less than 1% and sour being equal or more than 1%), and further split into subcategories based on substance: 'gas', 'oil', 'water', 'high vapour pressure pipeline (HVP)/low vapour pressure pipeline (LVP)' and 'other'. Facilities were split into 'batteries', 'gas plants', and 'compressor stations', the status of which were identified in EGIS as 'operating', and 'other facilities' which included all other types of facilities, regardless of operational status. Both bottom and surface holes were included on the map for wells.

[1] A 'Geo-referenced PDF' refers to a PDF file that allows users to obtain geographic coordinates and map distances directly from the PDF map using a mouse pointer or using reference points located on the map.
[2] The NTS mapping system is used by Natural Resources Canada to provide general-purpose topographic maps within Canada.

Step Four - Consideration of Potential Emission Densities

To provide a sense of emission density in relation to population and oil and gas infrastructure locations for mapping purposes, emissions data were collected from Environment Canada's National Pollutant Release Inventory (NPRI) database (Environment Canada 2014). This step was intended to provide a general, preliminary sense of where the greatest emission density of certain chemicals may be occurring in relation to larger emission sources (*e.g.*, gas plants). To achieve this, a list of all gas plants in the map area and their emissions (in tonnes) for volatile organic compounds (VOCs), $PM_{2.5}$, NO_x, and SO_2 was compiled. Data from the 2011 NPRI reporting year was used, unless no data were available. Emission densities were created by summing the tonnes of emissions of all gas plants located in a grid block, and dividing by the surface area of that grid block.

Step Five - Selection of HHRA Study Area

Upon detailed examination, one area emerged in the SLRA as having a relatively populated area, as well as the greatest density of oil and gas emission sources. This area, which is centred on Fort St. John, includes Dawson Creek, Pouce Coupe, Hudson's Hope and Taylor, the Blueberry River and Doig River First Nations, and the northern boundary of Tumbler Ridge. It represents the most densely populated area in NE BC, as well as the area with the highest density of continuous emission sources, as illustrated by Figure 3-2. The selected study area excluded the Fort Nelson LHA, due to the relatively lower population, facility and emission densities.

Slight changes in the dimensions of the HHRA study area have been made from what was proposed in the SLRA (the green square in Figure 3-2) as a result of the refinements made during the configuration of the air quality dispersion model. The revised study area is shown in Figure 3-3.

The study area for the HHRA now extends from just north of Tumbler Ridge from the south to approximately 30 km north of Wonowon. The west boundary of the study area is located approximately 40 km west of Hudson's Hope and extends to the BC/Alberta border. The major communities in the study area include Fort St. John, Dawson Creek and Chetwynd, along with smaller communities and First Nation lands.

Locations or points where COPC concentrations were predicted in the air dispersion model are called 'receptors' as per guidance from BC Guideline for Air Quality Dispersion Modelling (BC MOE 2008). Since no major emission sources or communities were identified on the outskirts of the HHRA study area to the west and the north and in an effort to reduce the model run time, the receptor grid extent was reduced to 150 km by 176 km (slightly smaller than the HHRA study area originally proposed in the SLRA). Over the entire study area, receptor grid spacing was set at 2 km apart, while a finer receptor grid spacing (*i.e.*, 250 m apart) was used in areas of interest. The areas with 'finer' grid spacing include:

- The larger communities in the region including Fort St. John, Dawson Creek, Chetwynd, Hudson's Hope, Pouce Coupe and Taylor.
- Areas incorporating the First Nation lands of West Moberly Lake, East Moberly Lake, Halfway River, Doig River and Blueberry River.
- Locations within 3 km from the largest 17 oil and gas emitters in the HHRA study area (based on NPRI 2010 emission rates) as displayed in Figure 3-4. Facilities included in

these large emitters include gas plants (12), large compressor stations (3) and booster stations (2).

A single point in each of the 13 smaller communities that were shown in Figure 3-4 was also added. A total of 15,676 receptors were used for this study in the prediction of air quality dispersion modelling results (Figure 3-4).

Although oil and gas activity exists outside of the study area, the intent of the detailed HHRA was to capture worst-case or near worst-case estimates of exposure that are relevant to the greatest number of people. By focusing the detailed HHRA on an area with a diverse range of relatively densely developed oil and gas activity and the greatest population density, evaluation of this area provides the most conservative and meaningful assessment of potential health risks associated with the COPC. Also, with the focus on the most populated area in the region, it was feasible to assume that potential vulnerable sub-populations may be present.

A survey distributed to the BC Air Shed Stakeholder Advisory Group (SAG 2013) and the NE BC Oil and Gas Health Advisory Committee members revealed that the top priorities for monitoring air quality in the region, based on feedback received from the survey respondents, included:

- Air quality in the most populated areas
- Air quality in the most developed areas with respect to oil and gas
- Estimation of highest and average human exposures
- Exposure of sensitive individuals

The study area selected for the evaluation of airborne emission sources in the detailed HHRA addressed these priorities.

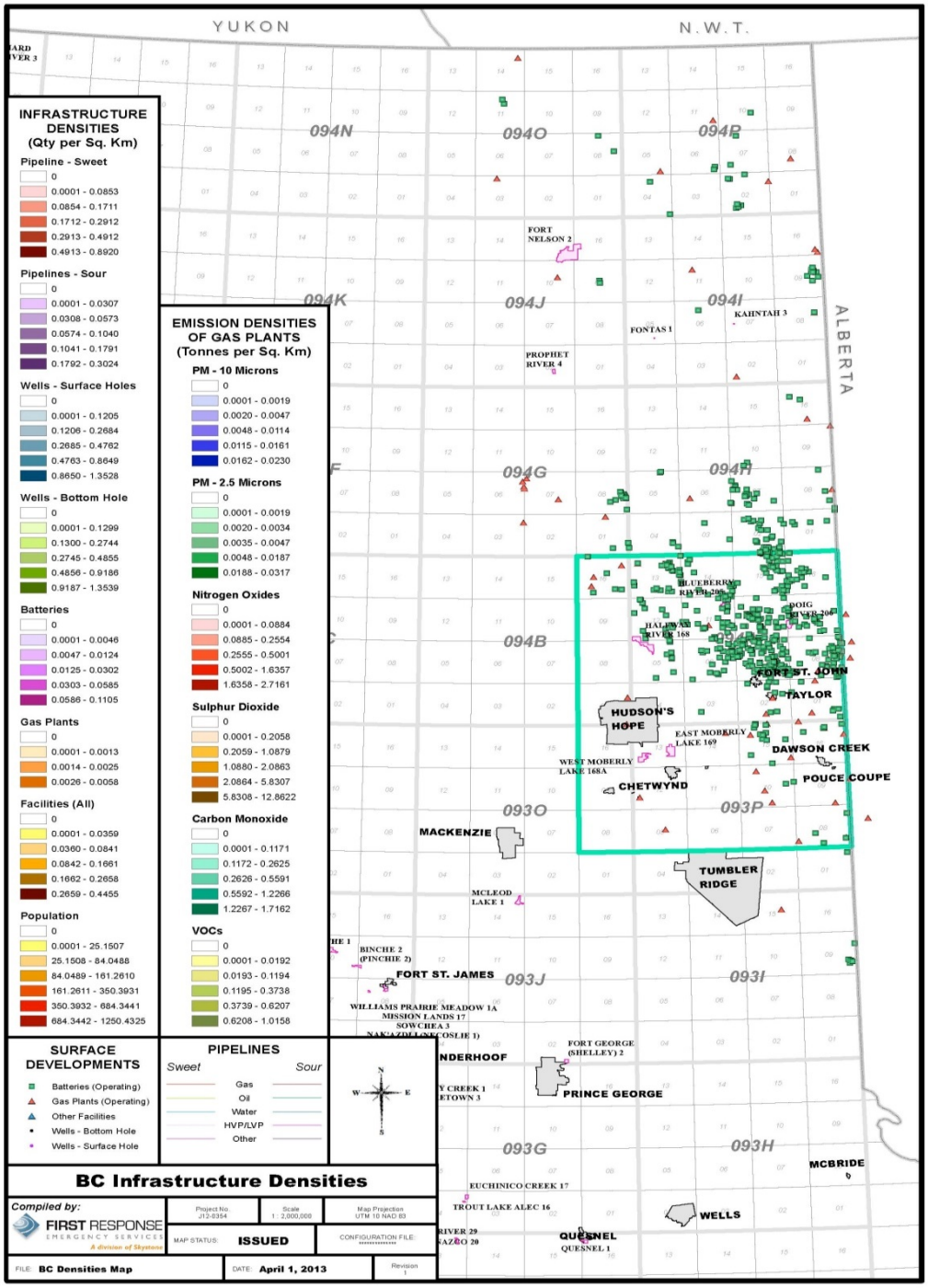


Figure 3-2 Map of Sweet and Sour Gas Plants (red triangles) and Batteries (green squares) in NE BC, Intrinsik (2014a)[3]

[3] This Figure originated from a layered map

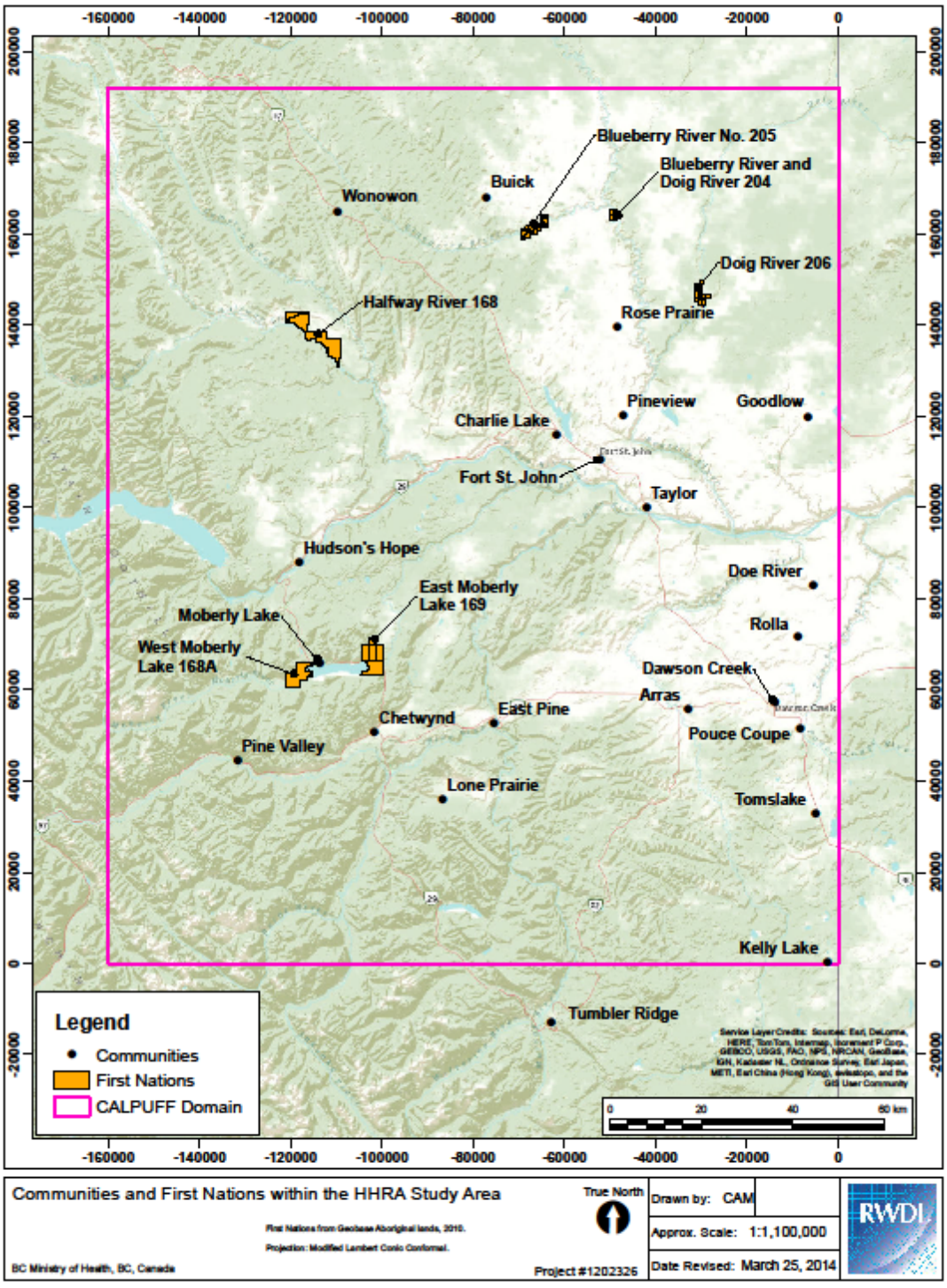


Figure 3-3 Human Health Risk Assessment Study Area and Locations Included in Detailed HHRA

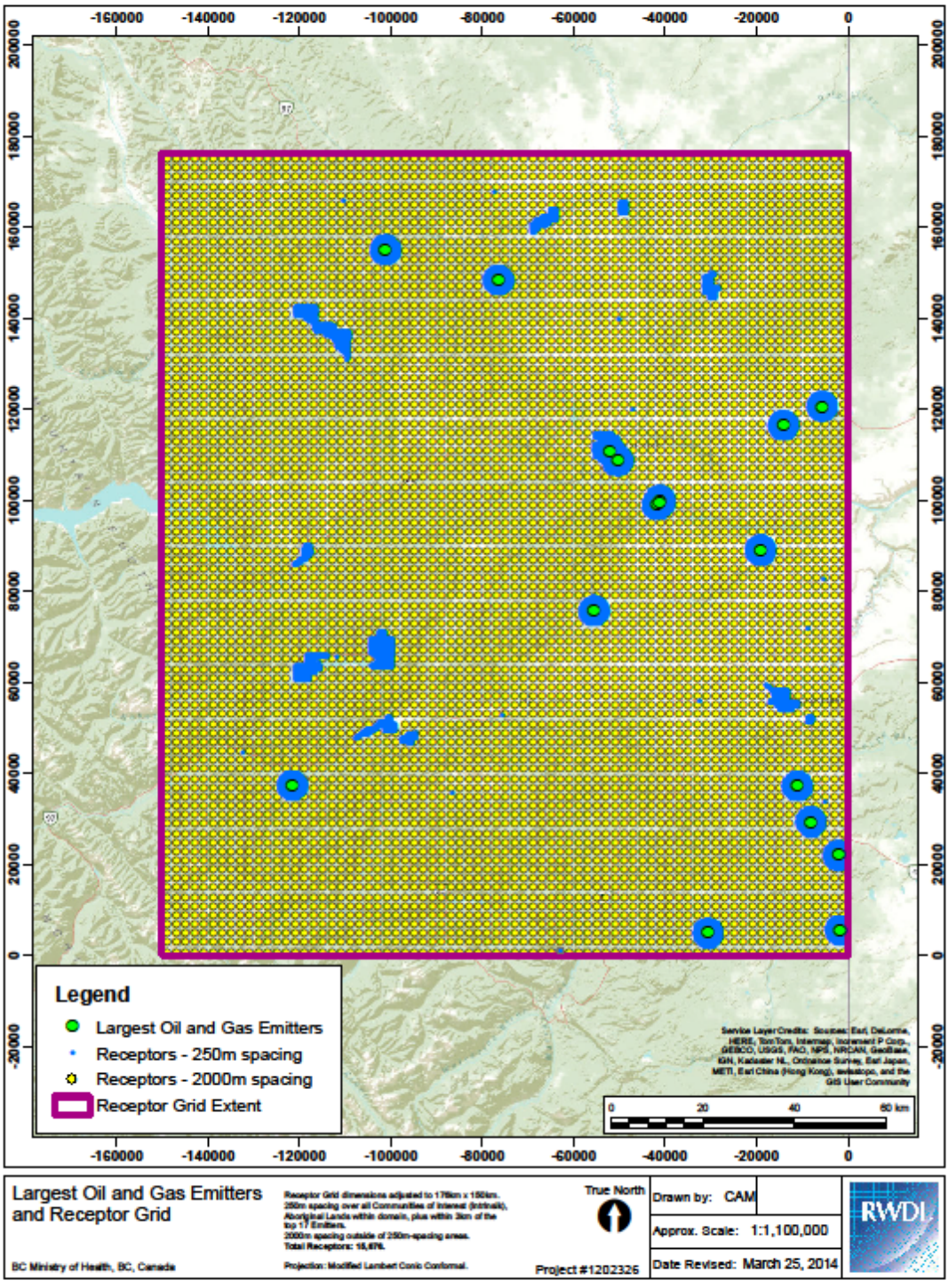


Figure 3-4 Receptor Grid for the Air Quality Dispersion Modelling

3.2.2 Exposure Scenarios

Based on the findings of the SLRA, the detailed HHRA focused on the assessment of the potential adverse health risks related to two air emission source types:

- Continuous emissions from gas processing plants
- Continuous emissions from production facilities

These air emission source types were identified as posing the greatest potential risk to the health in the region (when compared to the other air-related emission scenarios). To ensure that the potential influence on air quality (and consequently human health) was addressed on a cumulative basis, the emissions from the gas processing plants and production facilities were combined for a cumulative air quality assessment of the potential impacts of oil and gas activity on human health.

To further characterize air quality on a cumulative basis and in order to compare air quality associated with oil and gas activities with those associated with non-oil and gas emission sources, two scenarios were considered.

1. **Oil and Gas Scenario:** includes all on-going emissions from gas processing plants and various production facilities within the HHRA study area. These sources include, but are not limited to significant emitters such as, sweet and sour gas plants, compressor stations, fugitive emissions from tank storage.
2. **Cumulative Scenario:** includes the oil and gas sources from the first scenario, as well as emissions from background sources such as transportation and agriculture, and community activities (*e.g.,* residential wood burning). Other industrial sectors including paper and pulp, forestry and mining were also represented in this second scenario.

A large amount of information was collected and considered in the compilation of emissions data for both of these scenarios. Additional information regarding this process is available in Appendix A.

The objectives of the emission inventory and dispersion modelling were to provide detailed information regarding predicted concentrations of COPC that are emitted by oil and gas activities, and the cumulative effect when combined with other emission sources within the HHRA study area. This assessment consisted of three steps, which are further described below.

Step One - Collection of Supporting Information

The completion of dispersion modelling typically requires the collection and integration of meteorological data, topographical and land use data, and information on each of the emission sources.

Meteorological inputs to the model for this HHRA were taken from several surface stations for the period of January 16, 2011 to January 15, 2012, including:

- BC Ministry of Environment stations at Kwoen Gas Plant, Pine River Hasler and Taylor Town site
- BC Ministry of Forestry stations at Hudson’s Hope and Wonowon

- BC Ministry of Transportation stations at 73 Mile and Braden Road
- Three airports including Fort St. John, Chetwynd and Dawson Creek

Meteorological, topographical and land use data were obtained from public databases. To improve the meteorological simulation, more detailed meteorological data were obtained from BC Hydro as an input to the air dispersion modelling. The BC Hydro data included measurements from their monitoring network that was constructed in support of the Site C project near Fort St. John (Personal Communication, Al Strang 2014).

Step Two - Development of an Emissions Inventory

As described by BC MOE, an emissions inventory is "a complex process that involves estimating and compiling emissions activity from hundreds of point, area and mobile sources in an airshed" (BC MOE 2014d).

A considerable amount of effort was expended to create an up-to-date emissions inventory for the selected facility types within the HHRA study area (Figure 3-3). The procedures for developing the emissions inventory for the oil and gas sector and other sources of interest are briefly summarized below.

Emissions for the selected oil and gas activities in the HHRA study area were obtained from two main databases maintained by Environment Canada (2014). The first is the National Pollutant Release Inventory (NPRI) 2010 database (discussed in detail in Section 6.2 of the SLRA). The year 2010 was selected for use in this assessment for three reasons:

- Informal guidance from Environment Canada indicated that some quality assurance issues were still being addressed for some of NPRI 2011 records and NPRI year 2010 was recommended, being fully reviewed and confirmed.
- The NPRI 2010 emission inventory would coincide with the available 2010 Upstream Oil and Gas Emission Inventory allowing for direct comparison between the two inventories (this aided in identifying facilities that were considered in both inventories).
- Phase 1 of the SLRA (Intrinsik 2014a) considered NPRI 2010 data for the purposes of selecting the HHRA study area. To stay consistent with the reported emission intensities and other results in the SLRA, NPRI 2010 was used for the more detailed analysis.

Where required, information was supplemented with data from 2012 (Additional information is provided in Appendix A). Over 190 oil and gas facilities report to the NPRI and include both sweet sour and sweet gas plants along with many compressor stations. With assistance from several members of the Canadian Association of Petroleum Producers (CAPP) who operate in the NE BC region, individual facility operators for larger gas plants were also approached with respect to obtaining site specific information such as stack parameters and site plot plans. Five CAPP member companies provided detailed information of their facilities that was not available in NPRI. In addition, site specific information was collected from other non-CAPP operators where possible.

The second database provided by Environment Canada relied upon total annual emissions of small and temporary upstream oil and gas facilities such as well drilling sites and batteries that may not need permits through the BC MOE (Personal communication, Mourrand Sassi, January 2014). The small upstream oil and gas inventory provided annual emissions of nitrogen oxides (NO_x), sulphur dioxide (SO_2), fine particulate matter ($PM_{2.5}$) and total volatile organic compounds

(VOCs). In total, 6,034 facilities (including flares[4], storage facilities, compressors, leaks, *etc.*) from this inventory were identified in the HHRA study area. The base year of this inventory was 2010.

For the second scenario (cumulative), the NPRI database was used to include large emitters from the mining, forestry and pulp and paper industries (representing a total of nine facilities in the HHRA study area). A more complete emissions inventory of background sources such as road and non-road transportation, agricultural activities and residential heating was built from databases provided by Environment Canada (Personal Communications, Mike Moran 2013).

To estimate individual VOCs that were not reported to NPRI, emission profiles were applied to the annual total VOC emissions based on land use (such as residential, transportation and commercial) or industry classification, location and time of year of the predicted emissions. Estimation of individual polycyclic aromatic hydrocarbons (PAHs) was based on published emission factors from the United States Environmental Protection Agency (US EPA 1996) and other references found through a literature review (*i.e.*, Hytonen *et al*. 2009; Strosher 1999).

Step Three - Dispersion Modelling

Dispersion modelling was conducted using the BC MOE approved CALPUFF dispersion model (Version 5.8.4). CALPUFF is a multi-layer, multi-species, non-steady-state puff dispersion modelling software. It simulates the effects of time and space-varying meteorological conditions on pollutant, transport, transformation and deposition. For this study, three-dimensional meteorological data estimated by the CALMET meteorological model were used as input into the study. The dispersion model (CALPUFF) was used to predict ground-level air concentrations of the COPC throughout the HHRA study area.

3.2.3 Identification of People Who Might Be Exposed

The region that is the focus of the detailed HHRA is made up of a number of communities spread over a relatively large area (Figure 3-3), which could be potentially impacted by a number of different emission sources. Within each of these communities are people of different ages, with differences in existing health status, and lifestyle practices.

The larger communities that fall within the study area include:

- Fort St. John (pop. 20,268 in 2013)[5]
- Dawson Creek (pop. 12,285 in 2013)[5]
- Pouce Coupe (pop. 748 in 2013)[5]
- Hudson's Hope (pop. 1,039 in 2013)[5]
- Chetwynd (pop. 2,724 in 2013)[5]
- Taylor (pop. 1,488 in 2013)[5]
- Tumbler Ridge (pop. 2,785 in 2013)[5]

[4] Flares were only included if reported as part of the NPRI database for a facility. Flares that were intermittent and or used only during emergencies were not considered as point sources.
[5] Source: BC Stats 2013. Sub-Provincial Population Estimates. Released: January 31, 2014.

A number of First Nations communities are also located within the study area:

- Blueberry River First Nation (pop. 220)[6]
- Doig River First Nation (pop. 120) [6]
- Halfway River First Nation (pop. 155) [6]
- West Moberly Lake First Nation (no data)
- East Moberly Lake First Nation/Saulteau First Nation (pop. 340)[6]

In addition to oil and gas development, agriculture and tourism are important industries in the study area. The commercial farming of crops such as wheat, barley, canola, oats, various forage crops and vegetables is prevalent in the area. There are also commercial cattle, game and exotic livestock farming operations in the area (FBC 2012; BC ACF 2008). Over 300,000 people are estimated to visit the region every year for recreational purposes (FBC 2012).

Overall, there may be considerable diversity in the lifestyles and behaviours of people in the area that are relevant to the detailed HHRA. Based on the above information, it is reasonable to conclude that the study area includes:

- Individuals who may practice a subsistence lifestyle, and consume a large proportion of their diet from natural or traditional foods
- Individuals who live in a more urban environment and have ready access to supermarket foods but have limited access to natural foods
- People who live on farms or others who may consume a diet high in local agricultural foods
- People who may visit the area on a short-term basis for business or recreational purposes
- Sensitive individuals who may be more susceptible to the effects of chemical exposure, due to age, or pre-existing health conditions.

To the extent reasonably possible, these characteristics will be accounted for in the Exposure Assessment (Section 3.3).

In order to gain a better understanding of people who live in the HHRA study area, summaries of demographic and health status information have been completed (Sections 3.2.3.1 and Section 3.2.3.3).

3.2.3.1 Demographic Information

To gain a better understanding of the existing population in the study area, recent demographic information from Statistics Canada 2013 was compiled for the larger communities and regional districts within the HHRA study area. A summary of this information has been compiled in Table 3–1.

[6] Source: Statistics Canada 2013.

Table 3–1 Summary of Recent Demographic Information for Population within the HHRA Study Area[1]

	Age Groups														
	0-4 years			5-9 years			10-19 years			20-79 years			80+ years		
	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female
Blueberry River	20	5	10	20	5	15	45	25	20	135	70	55	-	-	-
Chetwynd	190	100	95	180	80	95	385	210	185	1,845	950	885	25	10	15
Dawson Creek	775	395	385	675	340	340	1,525	795	730	8,115	4,010	4,120	495	185	315
Doig River[2]	30 (total males and females)						15	-	-	75 (total males and females)					
Doig River[2]	10	5	5	10	10	5	20	10	10	90	55	30	0	0	0
East Moberly	25	15	10	20	10	10	60	40	25	210	115	100	0	-	5
Fort St. John	2,225	1,130	1,095	1,685	865	820	3,475	1,755	1,720	18,590	9,710	8835	420	155	265
Halfway River	15	10	10	20	15	5	20	15	5	100	40	65	-	-	-
Hudson's Hope	50	25	20	45	30	20	135	70	60	735	370	350	10	5	5
Peace River (Regional District)[3]	4,595	2,350	2,245	3,925	1,960	1,965	8,440	4,305	4,125	41,875	21,620	20,265	1,250	505	740
Pouce Coupe[2]	60	35	30	40	15	20	80	35	40	525	285	255	25	10	20
Taylor	130	60	65	100	45	55	170	85	80	970	510	455	15	10	5
Tumbler Ridge	180	95	85	155	75	85	330	160	175	2,025	1,130	900	20	10	5
West Moberly	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Notes:
- data not available
1 Source: Statistics Canada (2013). Several inconsistencies were identified between the 'Total' and the numbers of males and females within each age group. These discrepancies occur within the Statistics Canada tables as presented in the original source.
2 Data from Statistics Canada (2013) were not available. Alternatively, data from Statistics Canada (2012) were used to represent these communities.
3 The Peace River Regional District includes all communities within the HHRA study area.

Northern Health (2013a,b) has forecasted the expected degree of population growth in the region by age groups. Although the relative percentage of seniors is the smallest compared to the other health regions in BC, the senior population in NE BC is expected to grow over the next 15 years. The estimated growth by age group for each of the two LHA in the study area and the NE BC region is presented in Table 3–2. The information in these tables is based on data relating to trends in births, deaths, migration, fertility, and age-group survival. This information suggests that in the future, there may be a larger group of potentially sensitive individuals in the study area.

Table 3–2 Forecasted Population Growth by Local Health Area, 2015 to 2030 (in % growth)

Age Groups	Peace River North LHA 60	Peace River South LHA 59	NE BC
< 20	21	1.8	12.1
20 to 44	8.5	3.5	5.3
45 to 64	28.1	-4.3	11.9
> 65	113	81.5	100.8
Total	146.7	11.1	140.0

Source: Northern Health (2013a,b)

In addition to age, consideration of potential differences in behaviours that may predispose people to the effects of environmental exposure should be considered. This could include the consumption of traditional foods in First Nations communities. Recent information from Statistics Canada census data (Statistics Canada 2013) was compiled to obtain a better understanding of the relative proportion of individuals residing in the HHRA study area who identify as being Aboriginal (First Nations, Inuit, Metis) for the purposes of the Canadian census. This information is summarized in Table 3–3 for communities in the HHRA study area for which data were available.

Table 3–3 Summary of Relative Proportion of Individuals Identifying Aboriginal in 2011 Statistics Canada Census

Community	Individuals within Population	% Aboriginal in Population
Blueberry River	210	100
Chetwynd	2,600	12
Dawson Creek	1,650	15
Doig River	130	100
East Moberly	310	97
Fort St. John	3,025	12
Halfway River	165	100
Hudson's Hope	960	17
Peace River (Regional District)	8,135	14
Pouce Coupe	-	-
Taylor	180	13
Tumbler Ridge	350	12
West Moberly	70	100

Source: Statistics Canada (2013)
Notes: - data not available

Four of the communities in Table 3–3 appear to be populated completely by individuals who identify as Aboriginal (Blueberry River, Doig River, Halfway River, West Moberly). In addition, the community of East Moberly had 97% of the residents surveyed as being First Nations. In the larger communities or populations in the study area (Fort St. John and Dawson Creek) and the Peace River Regional District as a whole (which includes all of the smaller communities included in the HHRA), a smaller percentage of the population identified themselves as being Aboriginal (less than 15%). Some general conclusions may be drawn from this information:

- The HHRA study area includes a number of communities that are entirely Aboriginal in composition. It is expected that within these communities there are individuals who practice a traditional lifestyle, with a high reliance on foods such as game, fish and traditional plants. As such, these exposure pathways needed to be considered in the HHRA.
- The predominantly Aboriginal communities are smaller relative to more populated areas such as Fort St. John and Dawson Creek.
- The more populated areas have a smaller proportion of Aboriginal individuals. As a result, the HHRA must differentiate between exposures in urban *vs.* rural areas.

3.2.3.2 Behavioural Considerations

Both the region as a whole and the study area are inhabited by Aboriginal and non-Aboriginal communities. As a result, there are potential differences in behaviour patterns that could influence how much of a chemical a person is exposed to. For example, a person who actively hunts and harvests food from the region may experience different levels of exposure than a person who consumes primarily store-bought foods. For the purpose of the detailed HHRA, four general categories of individuals will be considered in the multiple pathway assessment in an effort to capture the various exposures that people in the region might receive:

- **Aboriginal group.** Individuals in this group were assumed (for the purposes of the detailed HHRA) to primarily consume locally sourced foods, including game animals, fish, berries and traditional plants, and garden vegetables. Recognizing that people identifying themselves as being of First Nations or Aboriginal descent may spend their entire lives in the region, it was assumed that people within this group are exposed to oil and gas emissions for 365 days per year, 24-hours per day, over an entire lifetime. Effort has been made to ensure that assumptions regarding potential exposure are appropriate and representative of individuals who practise traditional lifestyles.
- **Community Group.** This group has been assumed to consume a smaller proportion of their diet from local sources (*e.g.*, game, fruits and vegetables that might be grown in a home garden, or obtained from local vendors on occasion, as per Health Canada guidance). These individuals have been assumed to spend their entire lifetimes in the area, and are exposed 365 days per year, 24-hours per day.
- **Agricultural Group.** Various types of farming take place in the area, and as a result people may be exposed to chemicals through the consumption of local agricultural foods (beef, chicken, dairy, fruit, vegetables). To capture potential exposures that might be received by agricultural residents who consume a diet with a high proportion of local foods, it has been assumed that these individuals rely entirely on local foods.
- **Maximum Point of Impingement (MPOI).** This group represents hypothetical individuals could be present at the MPOI for each COPC. This assessment is intended to be very conservative and worst-case.

All information sources used in formulating and conducting the detailed HHRA will be provided, along with reference citations.

As it is likely that many residents could live in the NE BC region for extended periods and possibly over their entire lifetime, all age classes (life stages) have been considered in the detailed HHRA. The five life stages that will be included in the detailed HHRA are consistent with Health Canada guidance (Health Canada 2012):

- 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 stages of a person's life (*e.g.*, from infant to adult) is used to represent cumulative exposure over an 80-year lifetime.

To account for the physical and behavioural differences that exist between the different groups, several assumptions regarding exposure potential have been made. Additional information regarding these approaches is provided in Section 3.3 (Exposure Assessment).

3.2.3.3 Identification of Potentially Sensitive Individuals

As part of the Problem Formulation, Intrinsik has completed a general review of the existing health status in the region. The objective of this review is to gain a better understanding regarding the chronic diseases and conditions that are of relevance to the people in the area and the chemicals being assessed, and to further identify any potential sensitive sub-populations in the region who might be adversely impacted. The evaluation presented in this section is intended to provide a general, qualitative overview of some publicly available data only, and does not represent an epidemiological assessment of potential cause-effect relationships and is not intended to be a comprehensive review.

The focus of this health status review is on the Peace River North and Peace River South Local Health Areas (LHA 59 and 60, respectively) due to the range of the selected study area for the detailed HHRA. Peace River North LHA includes Fort St. John, Hudson's Hope, and Taylor, while the Peace River South LHA includes Chetwynd, Tumbler Ridge, Pouce Coupe, and Dawson Creek. As discussed earlier, the Fort Nelson LHA is outside of the HHRA study area.

Information was obtained from the following organizations as part of this review:

- BC Vital Statistics Agency
- Northern Health Authority
- Provincial Health Services Authority
- Statistics Canada

Where possible, information for other areas of BC or the Province as a whole is presented for comparison purposes.

The findings of the Literature Review (Intrinsik 2013) indicated that health conditions of interest with respect to oil and gas activity include:

- Acute (short-term, mild, transient) effects
- Respiratory diseases
- Reproductive effects
- Neurological effects
- Cardiovascular effects
- Autoimmune diseases
- Cancer

Accordingly, the emphasis of this health status review is on information relating to these conditions for the population within the selected HHRA study area, where information was available.

Review of Non-communicable Disease Mortality

To better understand the existing disease burden within the HHRA study area, mortality data were evaluated for the area, with a focus on the short-list of diseases and conditions identified from the Literature Review (Intrinsik 2013).

Recent mortality statistics from the Northern Health Authority (Northern Health 2012) and Statistics Canada (Statistics Canada 2013) provide comparative data for various Health Areas relative to the rest of the Province. A tabular summary of this information in relation to the selected list of diseases and conditions relevant to this HHRA is provided in Table 3–4 and Table 3–5. Some of the general findings, by disease or condition, are:

- **Cancer:** Mortality rates for all cancers (per 100,000 population) were higher for males and females in NE BC than for the Province or Canada (Northern Health 2012; Statistics Canada 2013).
- **Prostate and Breast Cancer:** Mortality rates were higher in NE BC compared to the Province (Northern Health 2012; Statistics Canada 2013).
- **Colorectal Cancer:** Mortality rates were higher for females in NE BC than the Provincial average, while mortality rates were lower for males (Northern Health 2012). However, Statistics Canada (2013) found that mortality rates for both males and females in NE BC were slightly higher than the Province.
- **Lung:** No notable differences in mortality for females were identified between NE BC and the Province, but mortality rates were observed to be higher for males in NE BC in comparison to both the Province and Canada (Northern Health 2012; Statistics Canada 2013).
- **Cardiovascular and Circulatory Diseases:** Slightly higher mortality rates for both genders as a result of ischemic heart disease (*i.e.*, coronary artery disease) were observed in NE BC than for the Province or Canada (Northern Health 2012; Statistics Canada 2013).
- **Cerebrovascular Disease:** In NE BC, mortality rates as a result of cerebrovascular disease were slightly higher for both genders than for the Province or Canada (Northern Health 2012; Statistics Canada 2013).

- **Chronic Respiratory Diseases**: mortality rates for bronchitis (emphysema and asthma), as well as for pneumonia and influenza were higher in NE BC when compared to rates for the Province and Canada (Northern Health 2012; Statistics Canada 2013).

Table 3–4 Comparison of 2011 Mortality Rates per 100,000 population for NE BC, Province of BC and Canada for Respiratory Diseases

Disease/Condition	NE BC			Province of BC			Canada		
	Male	Female	Total	Male	Female	Total	Male	Female	Total
Respiratory Diseases (all)	77.1	41	57.8	56.5	37.4	45.3	59.4	361	45
• Pneumonia and influenza	-	-	20.2	16.1	12.1	13.8	14.5	10	11.7
• Bronchitis, emphysema and asthma	-	-	5.1	3.4	2.4	2.8	3.0	2.0	2.4
• All other Respiratory Diseases	43.2	23.6	32.4	37	23	28.7	41.9	24	30.8

Source: Statistics Canada (2013)
Notes: - no data

Table 3–5 Comparison of 2011 Mortality Rates per 100,000 population for NE BC, Province of BC and Canada for Cancer

Disease/Condition	NE BC			Province of BC			Canada		
	Male	Female	Total	Male	Female	Total	Male	Female	Total
Cancer (all)	232.9	157	191.6	180.6	131.2	152.5	202.1	141.1	166.4
• Colorectal cancer	17.5	18.6	19	18.6	12.7	15.4	22.4	14.3	17.9
• Lung cancer	67.5	36.9	52.1	46.9	35	40.2	57.8	36.1	45.4
• Breast cancer	-	28.9	14.7	-	19.3	10.3	-	21.8	11.9
• Prostate Cancer	26.7	-	12.4	20.2	-	8.4	21	8.3	-

Source: Statistics Canada (2013)
Notes: - no data

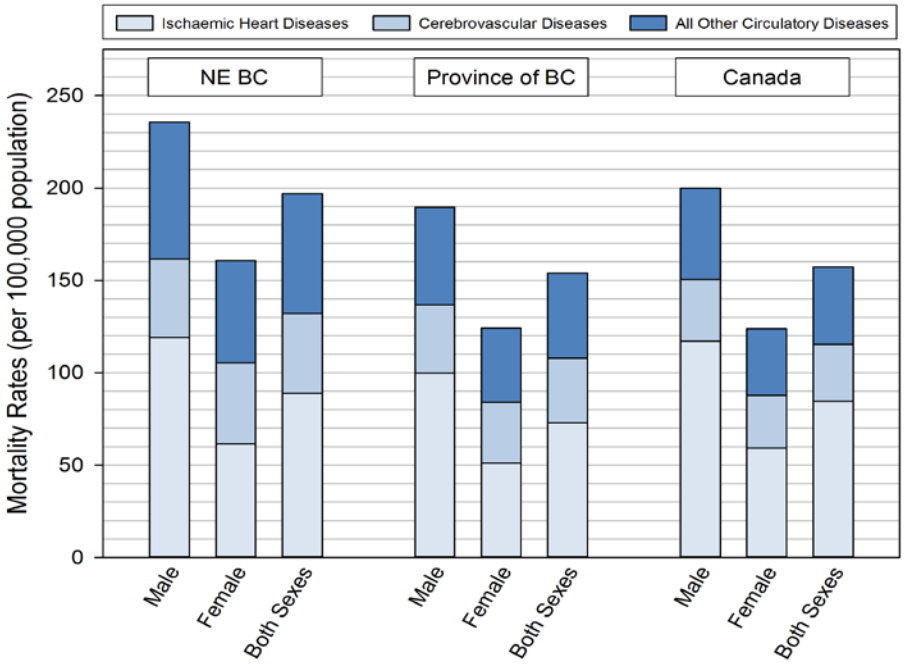


Figure 3-5 Comparison of Mortality Rates (per 100,000) for NE BC, the Province and Canada (Statistics Canada 2013)

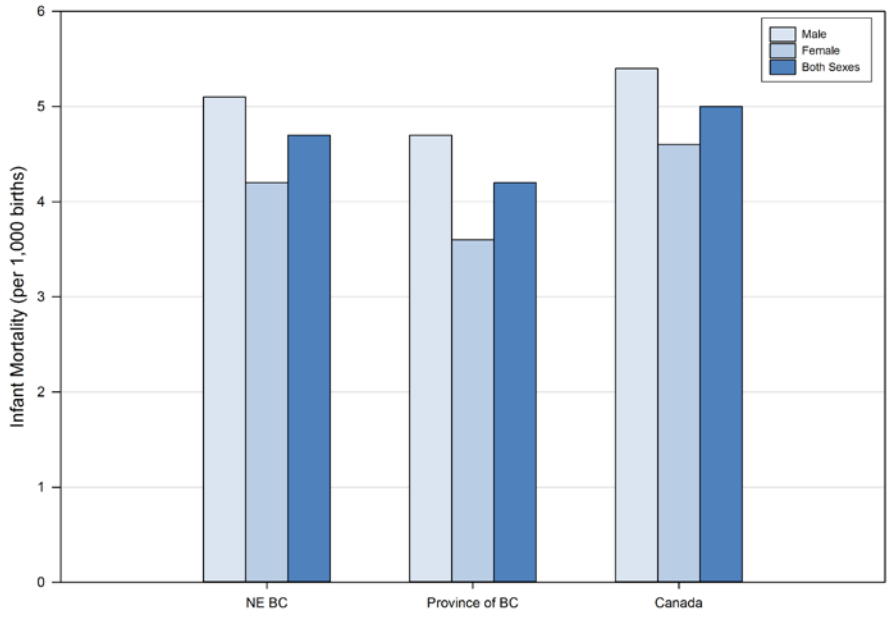


Figure 3-6 Comparison of Infant Mortality Rates for NE BC, the Province of BC and Canada (Statistics Canada 2013)

Data from the BC Vital Statistics Agency (2011) provides additional focus on a more sub-regional scale, with information available for the Peace River North and Peace River South LHA. In this data, standardized mortality rates (SMR) are used to quantify the increase or decrease in mortality for an area compared to the general population. The SMR are presented for Peace River North, Peace River South, and the NE BC region in Figure 3-7. The number of deaths compared to what is expected for each area is:

- **Peace River North:** Number of deaths were higher than expected for lung cancer, circulatory system conditions, ischemic heart disease, respiratory system conditions, pneumonia/influenza, and chronic lung disease (BC Vital Statistics Agency 2011). For cerebrovascular disease/stroke there were lower numbers of deaths than expected (BC Vital Statistics Agency 2011).
- **Peace River South:** Number of deaths were higher than expected for lung cancer, circulatory system conditions, ischemic heart disease, and chronic lung disease (BC Vital Statistics Agency 2011). There were lower numbers of deaths than expected for cerebrovascular disease/stroke, respiratory system conditions, and pneumonia/influenza (BC Vital Statistics Agency 2011).
- **NE BC:** Numbers of deaths were higher than expected for lung cancer, circulatory system conditions, ischemic heart disease, respiratory system conditions and chronic lung disease (BC Vital Statistics Agency 2011). For cerebrovascular disease/stroke and pneumonia/influenza there were lower numbers of deaths than expected (BC Vital Statistics Agency 2011).

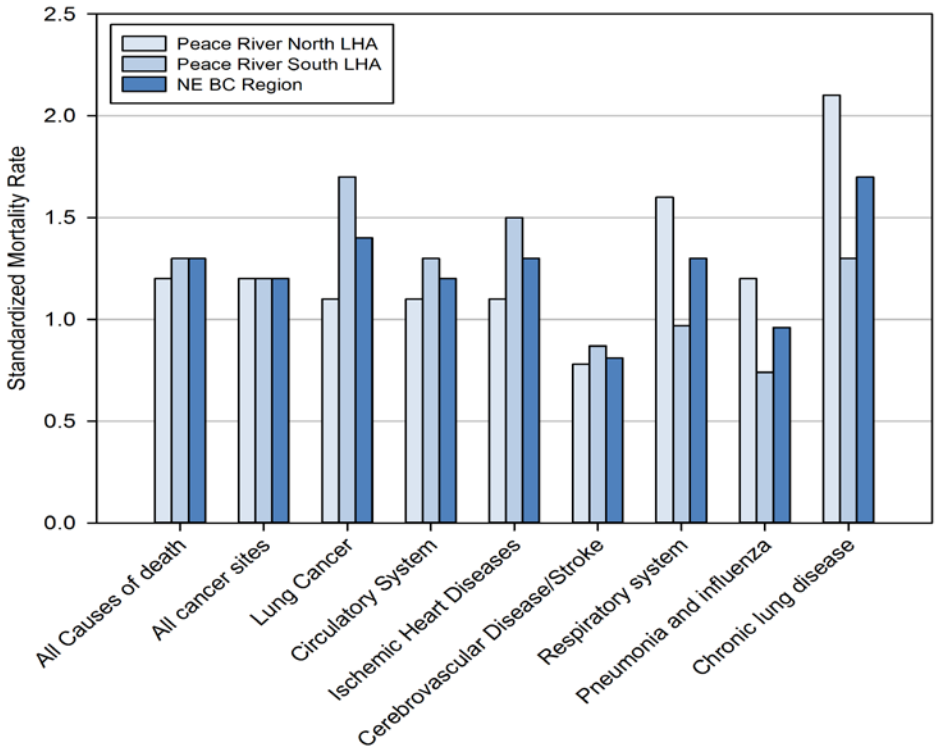


Figure 3-7 Comparison of Standardized Mortality Rates (SMR) for the Peace River North and Peace River South LHA with the NE BC Region

The Community Health Atlas (Northern Health 2013c) provides disease statistics for the Peace River North and South LHA for the years 2007 to 2011. Several of the conditions have been described by Statistics Canada (2013) and BC Vital Statistics Agency (2011). One of the conditions not described in these data sets, infant mortality rates (Northern Health 2013c), is presented in Figure 3-8 for the Peace River North and South LHA from the Community Health Atlas.

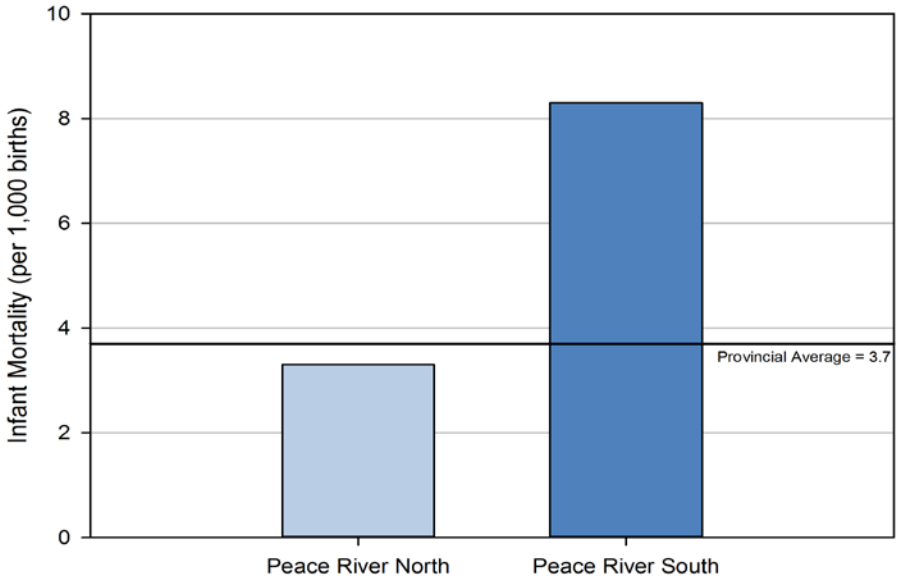


Figure 3-8 Comparison of Infant Mortality Rates (per 1,000 births) with the Provincial Average (Northern Health 2013c)

Non-communicable Disease Morbidity

Relative incidence rates of the identified diseases and conditions were evaluated for the population within the HHRA study area to better understand how these health issues were affecting people in the area.

Recent morbidity statistics from Statistics Canada (2011), the Northern Health Authority (Northern Health 2013c), and the Provincial Health Services Authority (Fang *et al*. 2010) provide comparative data for the Peace River South and Peace River North Health Areas[7] relative to the rest of the Province. This information is summarized in Table 3-6.

[7] Fort Nelson Local Health Area was excluded from this review, as it is considered to be outside the HHRA study area.

Table 3–6 Summary of Existing or Treated Cases of Various Diseases in the Peace River North Local Health Area and the Peace River South Local Health Area

Disease/Condition	Existing or Treated Cases in 2010-2011 (Northern Health 2013c)						
	Fort St. John	Dawson Creek	Hudson's Hope	Peace River North LHA	Chetwynd	Tumbler Ridge	Peace River South LHA
Cardiovascular Disease	650	560	33	1,143	124	127	1,294
Ischemic Heart Diseases	446	388	23	784	86	88	86
Congestive Heart Failure	323	288	16	568	64	65	665
Stroke	92	83	5	161	18	19	192
Hypertension	2,553	2,256	130	4,486	500	511	5,210
Acute Myocardial Infarction	258	202	13	454	45	46	466
COPD	478	346	24	840	77	78	800
Asthma	1,418	849	72	2,483	188	192	1,961
Rheumatoid Arthritis	235	111	12	413	25	25	257

Statistics Canada has compiled data for the NE BC region[8] as a whole expressed in percentage of the population (%) or in rates per 100,000 people, and has compared this regional data with comparable statistics for BC and Canada. The most recent published data (for 2011) are presented in Figures 3.9 to 3.12 for the diseases and conditions of interest to this HHRA.

[8] The data from Statistics Canada (2012, 2013) is presented for the 'North East Health Service Delivery Area'. This area is noted by Statistics Canada to include people living in the entire NE BC region, including the Fort Nelson area. Populations included in these statistics are : City of Fort St. John, City of Dawson Creek, District Municipality (DM) of Tumbler Ridge, DM of Chetwynd, DM of Hudson's Hope, DM of Taylor, Village of Pouce Coupe, Peace River district electoral areas B, C, D, and E, First Nations communities of: East Moberly Lake 169, West Moberly Lake 168A, Doig River 206, Halfway River 168, Blueberry River 205, Lower Post, Liard River 3, Fontas 1, Fort Nelson 2, Kahntah 3, Prophet River 4.

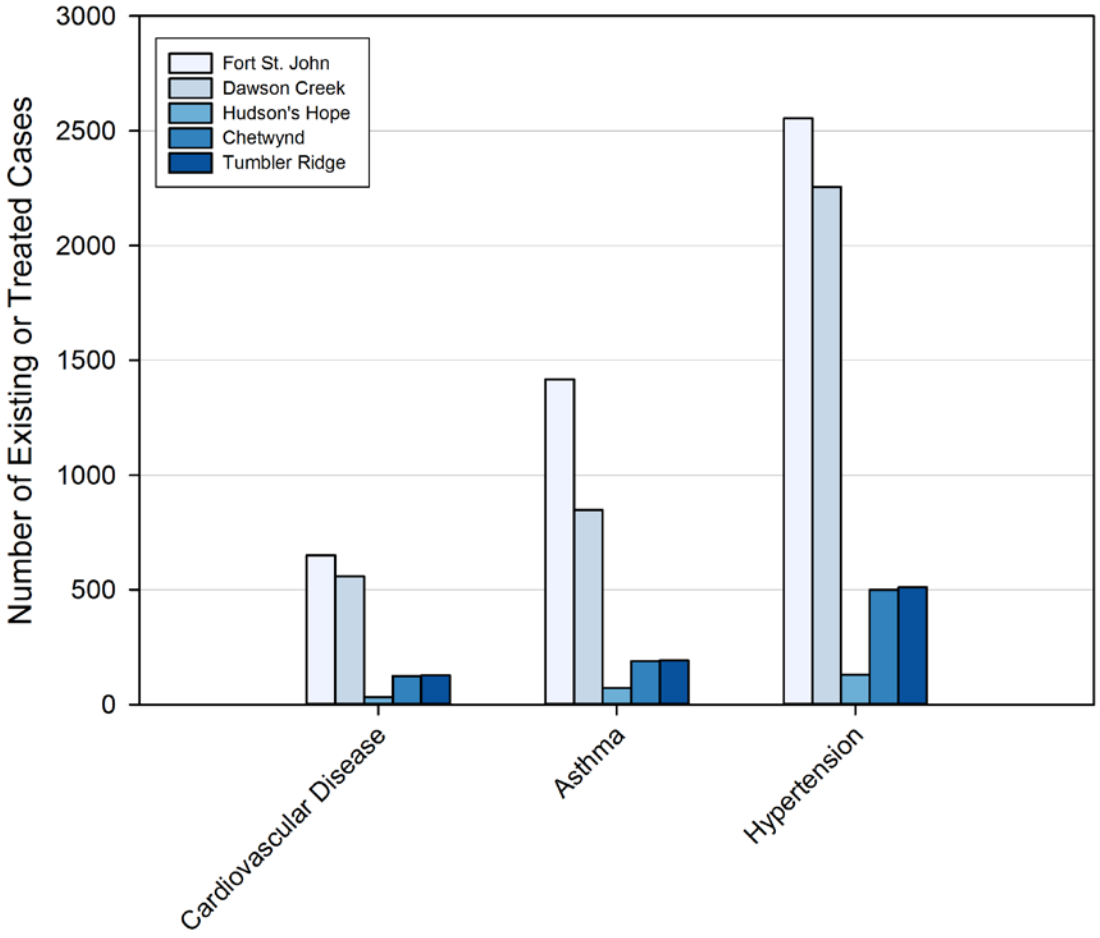


Figure 3-9 Comparison of the Number of Existing or Treated Cases of Cardiovascular Diseases, Asthma and Hypertension between Communities in NE BC in 2011

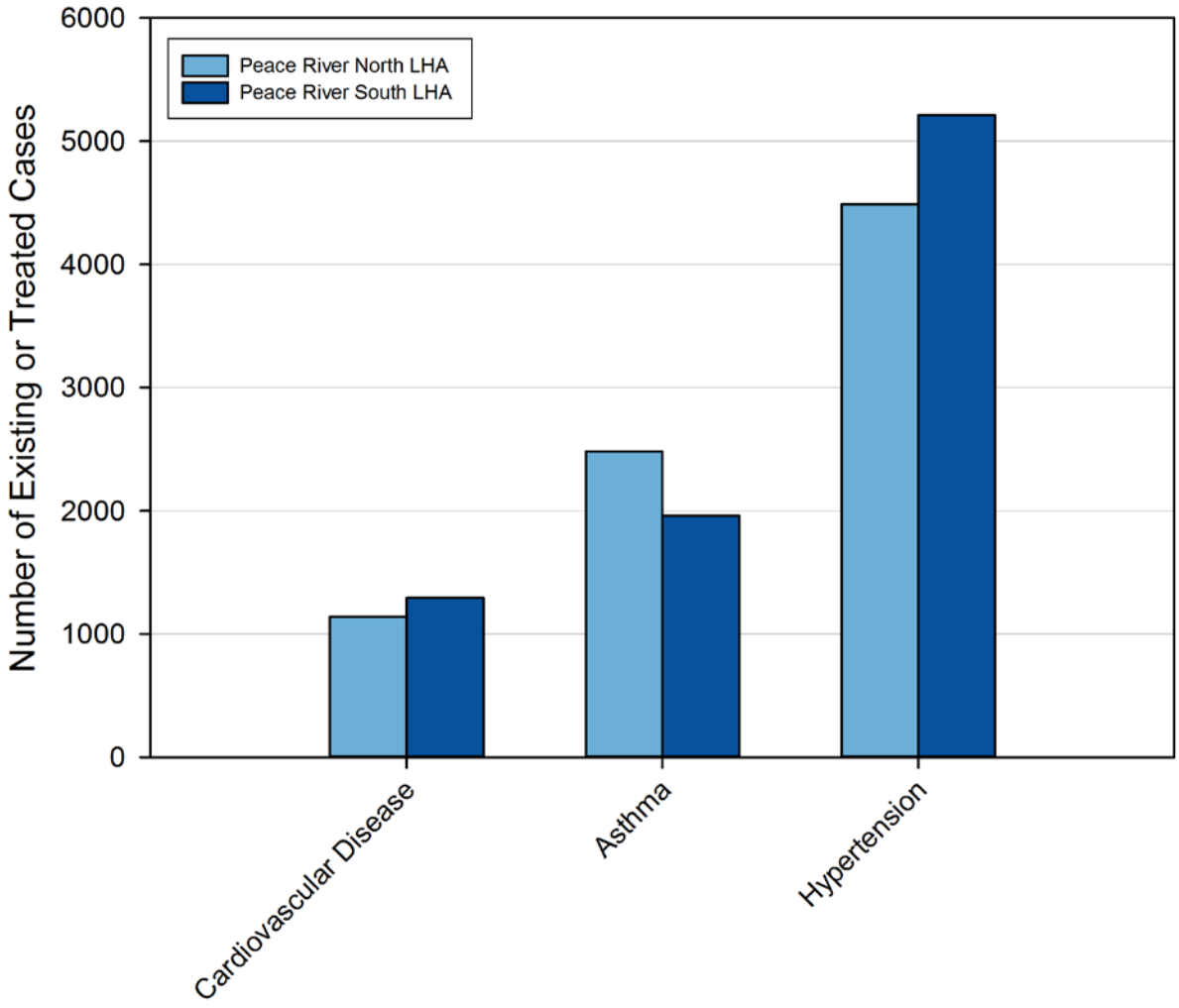


Figure 3-10 Comparison of the Number of Existing or Treated Cases of Cardiovascular Diseases, Asthma and Hypertension between Local Health Areas in NE BC in 2011

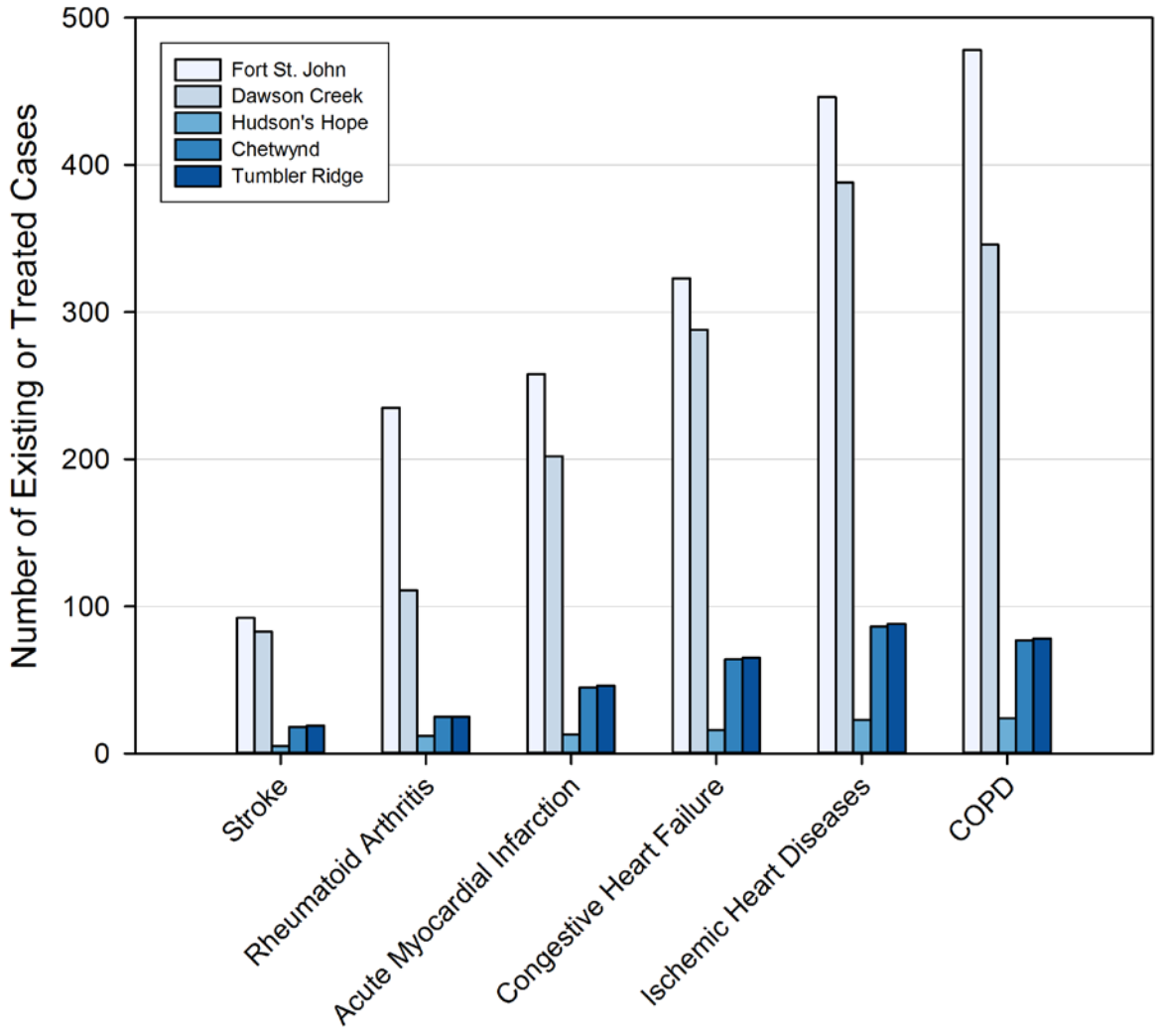


Figure 3-11 Comparison of the Number of Existing and Treated Cases of Various Diseases between Communities in NE BC in 2011

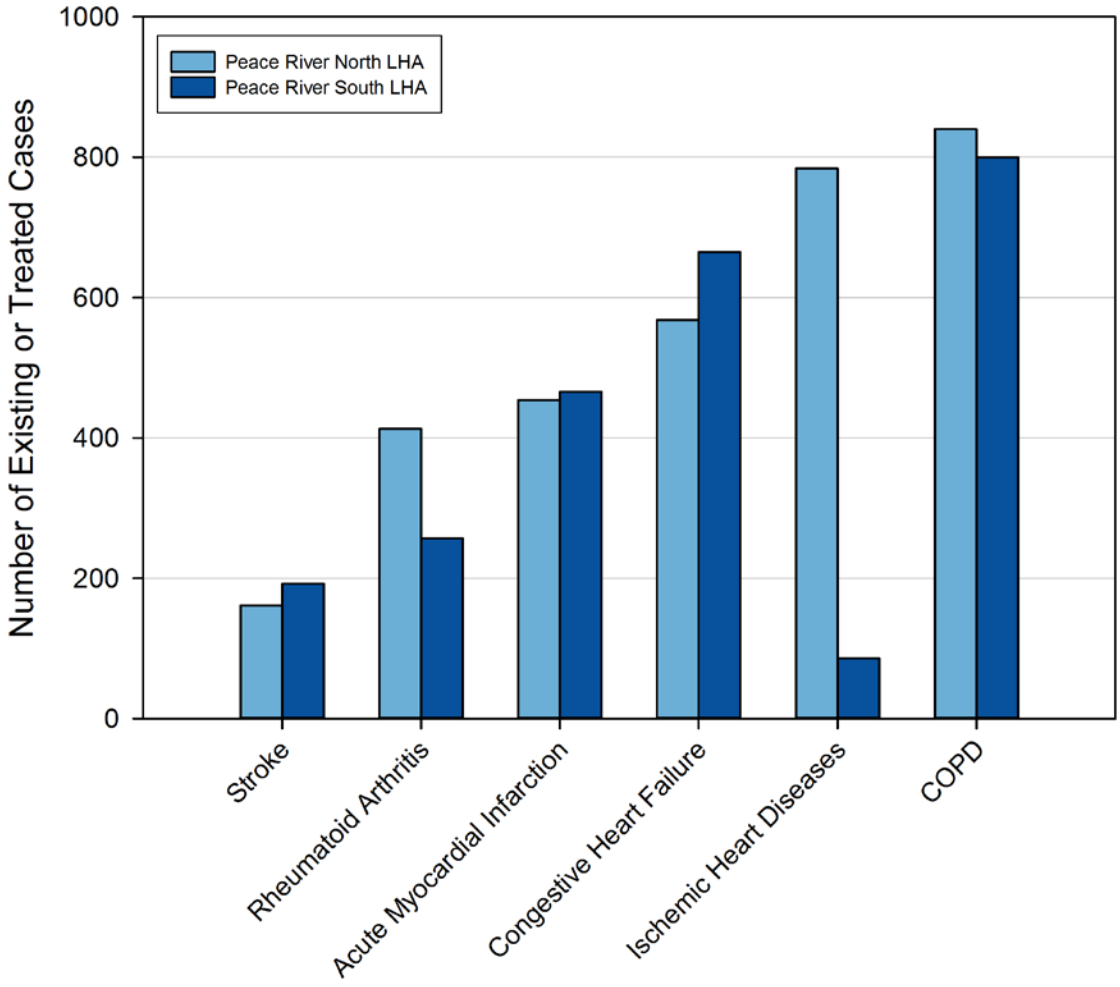


Figure 3-12 Comparison of the Number of Existing and Treated Cases of Various Diseases between Areas in NE BC in 2011

Some additional disease prevalence data for Northern BC have been published by the Provincial Health Services Authority (Fang *et al.* 2010). When each health condition of interest was analyzed individually, the unadjusted prevalence (not age-adjusted) was similar between Northwest BC, Northern Interior BC, and NE BC (Fang *et al.* 2010).

The unadjusted prevalence of diseases in northern BC is presented Table 3-7.

Table 3–7 Prevalence of Diseases in Northern BC in 2007

Health Condition	Health Service Delivery Area	Unadjusted Prevalence (%)		
		Total	Males	Females
Cancer	Northwest	1.1	1.2	1.0
	Northern Interior	1.1	1.1	1.1
	Northeast	0.8	0.8	0.9
COPD	Northwest	4.7	5.0	4.5
	Northern Interior	5.5	5.8	5.3
	Northeast	5.5	5.6	5.4
Asthma	Northwest	12.8	11.3	14.4
	Northern Interior	13.9	12.4	15.4
	Northeast	11.3	10.3	12.4
Cardiovascular Disease	Northwest	4.4	5.2	3.6
	Northern Interior	4.3	5.0	3.5
	Northeast	3.2	3.7	2.6

Source: Fang *et al.* (2010)

Summary of Existing Health Information

Although much of the data cannot be directly compared across the areas due to the various statistics and populations used, some general conclusions can be reached from the review of the mortality and morbidity data for the region:

- Many of the conditions identified as being of interest to the HHRA in the Literature Review (Intrinsik 2013) are prevalent in the population of NE BC.
- A recent report by Northern Health (2012) recognizes that Health status indicators consistently show that the residents of parts of NE BC (*e.g.*, Peace River North LHA 60) are not as healthy as the rest of BC with respect to a number of health indicators. The Fang *et al*. (2010) study of health conditions in the Province also noted that the Northern Health Region had high rates of prevalence and in the rates of increases of certain chronic health conditions (hypertension, cardiovascular disease, asthma). Some of the factors that were identified by Northern Health (2012) as contributing to chronic health conditions in the region include:
  - Physical inactivity
  - Unhealthy eating habits
  - Obesity
  - Tobacco use
  - Alcohol use
- These findings for the NE BC region are generally consistent with the findings of a report by the Canadian Health Institutes Initiative (CIHI 2006), where people living in rural areas showed a health disadvantage with respect to mortality and the incidence of chronic diseases (including cardiovascular and respiratory diseases).
- In some instances, higher rates of mortality or morbidity for respiratory diseases, cardiovascular disease and associated conditions and lung cancer were identified in populations within the NE BC region. This suggests that, of the list of conditions identified in the Literature Review (Intrinsik 2013), these may be the most important to the HHRA study population from a prevalence perspective.

- The selection of exposure limits for use in the Toxicity Assessment needs to take into consideration the diseases of interest from the Literature Review, with particular attention on the potential adverse respiratory, cardiovascular, and lung cancer effects.

Finally, it is apparent that several factors contribute to the development of chronic health conditions, of which chemical exposure is only a part. This detailed HHRA is intended to evaluate only potential chemical exposures from oil and gas activities. However, it is important to acknowledge the existing health status of the region.

3.2.3.4 Locations where People Might Be Exposed

Given the scope of the Phase 2 HHRA project, it is not possible to evaluate every community or individual who may be exposed on an individual basis within NE BC.

As described in Section 3.2.1, the HHRA study area has been selected to focus on an area associated with worst-case or near worst-case conditions with respect to exposure and consequent health risks. The study area is associated with the highest density of continuous emission sources and most densely populated communities. Therefore, the area represents the greatest likelihood of exposure to oil and gas emissions. This approach permits the detailed HHRA to provide conservative, representative estimates of exposure that might be received by people who live in the region.

The study team has selected a number of discrete locations that represent many of the larger and smaller communities in the study area to include in the detailed HHRA. Using site-specific information regarding meteorology, terrain, and other factors, air quality dispersion modelling has been completed for these individual locations as well as for the study area as a whole. The list of discrete locations included in the HHRA, as well as the category assigned (Aboriginal, Agricultural, Community, or MPOI) applied to the different locations (see Section 3.2.3.2) are presented in Table 3–8.

In general, the larger communities (Fort St. John, Dawson Creek) were classified as ‘Community Residents’, and the First Nations as ‘Aboriginal Residents’. The smaller communities were classified as Aboriginal if they were in close proximity to known Aboriginal communities. All other communities were assumed to be ‘Agricultural Residents’.

Visitors or recreational users in the region were not assessed on an individual or discrete basis due to the large area over which these people could be present (*i.e.,* the entire HHRA study area). To capture potential worst-case short-term exposures that these individuals might receive, the maximum predicted air concentrations for each COPC and averaging period for the entire study area (the MPOI[9]) were used to evaluate the potential health risks.

[9] The MPOI (maximum point of impingement) for each COPC and averaging period were determined in the air quality dispersion modelling. Additional information regarding this modelling is provided in Appendix A.

Table 3–8 Discrete Locations Included in the Human Health Risk Assessment

Location	Assigned Group
Arras	Agricultural
Blueberry River First Nation	Aboriginal
Buick[1]	Aboriginal
Chetwynd	Agricultural
Dawson Creek	Community
Doe River	Agricultural
Doig River First Nation	Aboriginal
East Moberly Lake First Nation (Saulteau)	Aboriginal
East Pine	Agricultural
Fort St. John	Community
Goodlow	Agricultural
Halfway River First Nation	Aboriginal
Hudson's Hope	Agricultural
Kelly Lake	Agricultural
Lone Prairie	Agricultural
Moberly Lake[2]	Aboriginal
Pine Valley	Agricultural
Pine View	Agricultural
Pouce Coupe	Agricultural
Rolla	Agricultural
Rose Prairie	Agricultural
Taylor	Agricultural
Tomslake	Agricultural
Tumbler Ridge	Agricultural
West Moberly Lake First Nation	Aboriginal
Wonowon[3]	Aboriginal

Notes:
1 Buick is located in proximity to the Blueberry River First Nation and was considered to be an Aboriginal location for the purposes of the HHRA.
2 Moberly Lake is in proximity to the West and East Moberly Lake First Nations, and was considered to be an Aboriginal location for the purposes of the HHRA.
3 Wonowon is located in proximity to the Halfway First Nation and was considered to be an Aboriginal location for the purposes of the HHRA.

3.2.4 Identification of Chemicals of Potential Concern

As discussed previously, the emission scenarios selected for further evaluation in the detailed HHRA include continuous airborne emissions from gas processing plants and production facilities. Accordingly, the selection of COPC must focus on chemicals that are associated with these emission sources.

The overall objectives of the COPC selection process for the detailed HHRA are to:

- Identify those chemicals that are emitted from gas processing plants and production facilities, based on published documentation.
- Select COPC that may be emitted from these activities that are of particular concern with respect to human health.

- When possible, to select COPC that may be considered to be representative of a number of different COPC, with respect to toxic potential.

As part of the SLRA (Intrinsik 2014a), the study team reviewed the available scientific information regarding chemicals that may be emitted from the identified sources, and compiled a list of COPC that are known to be emitted from gas plants or production facilities and are of particular concern to human health, or are associated with certain health endpoints of interest. By focusing on a list of specific COPC, a more thorough assessment of potential human exposures in the study area may be completed.

Chemicals known to be associated with oil and gas activity that have been selected for further consideration are summarized in Section 3.2.4.1. Further details are available in the SLRA (Intrinsik 2014a). In the development of emission inventories for the HHRA study area, lists of chemicals associated with different types of sources (Oil and Gas, Cumulative, *etc.*) were formulated. This information was considered in the development of a final list of COPC for this detailed HHRA (see Section 3.2.4.2).

3.2.4.1 Selected Chemicals of Potential Concern for Consideration in the HHRA

A summary of the selected COPC associated with continuous air emission sources (gas processing plants, various production facilities) in the area is provided in Table 3–9, along with a general description of the rationale for including each COPC. This list primarily includes COPC with the health endpoints of interest identified from the Literature Review (Intrinsik 2013): cancer, respiratory effects, reproductive/developmental effects, cardiovascular and immunological effects.

Table 3–9 Summary of Selected Chemicals of Potential Concern for Evaluation in the Human Health Risk Assessment of Oil and Gas Activity in Northeastern British Columbia

Selected Chemical of Potential Concern	Rationale for Inclusion	References
Sulphur Dioxide (SO_2)	Presence in combusted sour fuel emissions, potential for adverse respiratory effects	Krzyzanowski 2012; US EPA 2010a; Witter *et al.* 2008
Nitrogen Dioxide (NO_2)	Presence in combustion emissions, potential for adverse respiratory effects	Krzyzanowski 2012; US EPA 2008; Witter *et al.* 2008
Fine Particulate Matter ($PM_{2.5}$)	Presence in combustion emissions, potential for adverse respiratory and cardiovascular effects	CCME 2012a, 2000; CARB 2005
Benzene	Detected in ambient air near upstream oil and gas operations in Western Canada, presence in emissions from gas plants and production facilities, potential for adverse immunological or carcinogenic effects, predicted risks in oil and gas risk assessments	Krzyzanowski 2012; You *et al.* 2008; ATSDR 2007; Burstyn *et al.* 2007; TCEQ 2007a; US EPA 2000; McKenzie *et al.* 2012; Witter *et al.* 2008, 2011
Toluene	Detected in ambient air near upstream oil and gas operations in Western Canada, potential for adverse respiratory and neurological effects	Krzyzanowski 2012; You *et al.* 2008; US EPA 2005; ATSDR 2000; Witter *et al.* 2008
Ethylbenzene	Detected in ambient air near upstream oil and gas operations in Western Canada, potential for adverse reproductive/developmental effects, neurological effects, predicted risks in oil and gas risk assessments	Krzyzanowski 2012; ATSDR 2010; McKenzie *et al.* 2012; Witter *et al.* 2008, 2011; You *et al.* 2008
Xylenes	Detected in ambient air near upstream oil and gas operations in Western Canada, potential for adverse respiratory and neurological effects, predicted risks in oil and gas risk assessments	Krzyzanowski 2012; TCEQ 2009; McKenzie *et al.* 2012; Witter *et al.* 2008, 2011; You *et al.* 2008
1,3-Butadiene	Potential reproductive/developmental effects, carcinogenic effects, predicted risks in oil and gas risk assessments	US EPA 2002; McKenzie *et al.* 2012; Witter *et al.* 2011
Acrolein	Presence in combustion emissions, potential for respiratory effects	OEHHA 2008a
Acetaldehyde	Presence in combustion emissions, respiratory effects and cancer	Krzyzanowski 2012; OEHHA 2008b; Health Canada 2004
Cyclohexane	Associated with risks in oil and gas risk assessments (aliphatic hydrocarbons), potential reproductive effects	McKenzie *et al.* 2012; US EPA 2003a
Formaldehyde	Presence in combustion emissions, potential respiratory and carcinogenic effects	TCEQ 2008; ATSDR 1999; US EPA 1991
n-Hexane	Potential neurological effects, predicted risks (for aliphatic hydrocarbons) in oil and gas risk assessments	TCEQ 2007b; McKenzie *et al.* 2012; Witter *et al.* 2011
Hydrogen sulphide (H_2S)	Presence in sour oil and gas emissions, respiratory, neurological effects	ATSDR 2006; US EPA 2003b
Isopropylbenzene (cumene)	Detected in ambient air surrounding upstream oil and gas operations in Western Canada,	You *et al.* 2008
Naphthalene	Detected in ambient air surrounding upstream oil and gas operations in Western Canada, potential respiratory effects	You *et al.* 2008; US EPA 1998
n-Pentane	Associated with risks in oil and gas risk assessments (aliphatic hydrocarbons), potential neurological effects	You *et al.* 2008; McKenzie *et al.* 2012; TCEQ 2011

Selected Chemical of Potential Concern	Rationale for Inclusion	References
Trimethylbenzenes	Detected in ambient air near upstream oil and gas operations in Western Canada, potential neurological effects; predicted risks in oil and gas risk assessments	US EPA 2010b; McKenzie *et al.* 2012; Witter *et al.* 2011; You *et al.* 2008
Benzo(a)pyrene and other carcinogenic PAHs	Associated with airborne emissions from oil and gas facilities, carcinogenic effects	Ana *et al.* 2012; Health Canada 2012; Krzyzanowski 2012

3.2.5 Identification of Exposure Pathways of Interest

The exposure pathways that are relevant to people in the region vary with the receptor group being assessed (Aboriginal, Community Resident, Agricultural Resident, MPOI). This reflects potential differences in behaviours between these groups, such as the types and quantities of food consumed (see Section 3.3.3). For inhalation exposures, no differences between these groups were assumed.

Both primary and secondary exposure pathways are potentially relevant to the evaluation of exposures that might be received by people as a result of continuous emissions from gas plants and production facilities. Inhalation of COPC from airborne emissions is considered to be a primary pathway. Secondary pathways are the result of deposition or accumulation of COPC within various environmental media that come into contact with air, such as soil, water, and dust as well as foods from these areas that are subsequently consumed by people or animals.

The assumed pathways that the four groups have in common are the inhalation of air and dust, dermal (skin) contact and ingestion of soil and food. The key differences in how each group is assessed are the assumptions applied in the HHRA for ingestion pathways, with respect to what and how much of the food types are consumed. A comparison of the exposure pathways that was given further consideration in the detailed HHRA for the four groups is provided in Table 3-10. Consideration was given to all life stages in the multiple pathway assessment. Going forward, the indirect pathways will be referred to as the '***multiple pathway assessment'***.

Table 3–10 Summary of Potential Exposure Pathways

Exposure Pathway	Aboriginal Group	Community Group	Agricultural Group	MPOI
Inhalation				
Inhalation of air	✓	✓	✓	✓
Inhalation of dust	✓	✓	✓	✓
Ingestion				
Ingestion of soil (inadvertent)	✓	✓	✓	✓
Ingestion of water[1]	✓	✓	✓	✓
Ingestion of local fruit and vegetables	✓	✓	✓	✓
Ingestion of traditional plants	✓	X	x	✓
Ingestion of local berries	✓	✓	✓	✓
Ingestion of local livestock and poultry	x	X	✓	✓
Ingestion of local dairy and eggs	x	X	✓	✓
Ingestion of local wild game	✓	X	x	✓
Ingestion of local fish	✓	✓	✓	✓
Dermal Contact				
Dermal contact with soil	✓	✓	✓	✓
Dermal contact while swimming or bathing	✓	✓	✓	✓

Notes:
1 Includes ingestion of drinking water (ground or municipal) and incidental ingestion while swimming.
✓ Exposure pathway is applicable for the receptor group.
x Exposure pathway is not applicable for the receptor group.

People in the study area are expected to consume local agricultural foods (beef, poultry, dairy, fruits and vegetables), berries, fish, and traditional foods (large and small game animals, traditional plants). To reflect this, human exposure to the COPC via these food-related pathways was given consideration in relation to the assessment of long-term, continuous emission sources to air, where adequate information is available. Additional details as to the methodologies used in estimating exposure are provided in Section 3.3 (Exposure Assessment).

Conceptual models linking the selected emission sources (gas processing plants and production facilities) with the identified exposure pathways were constructed. Separate models for each group of individuals (Aboriginal, Community, Agricultural) were developed, and are presented as Figure 3-11 to Figure 3-13. The assessment of MPOI concentrations was completed using the conceptual models for the other three groups.

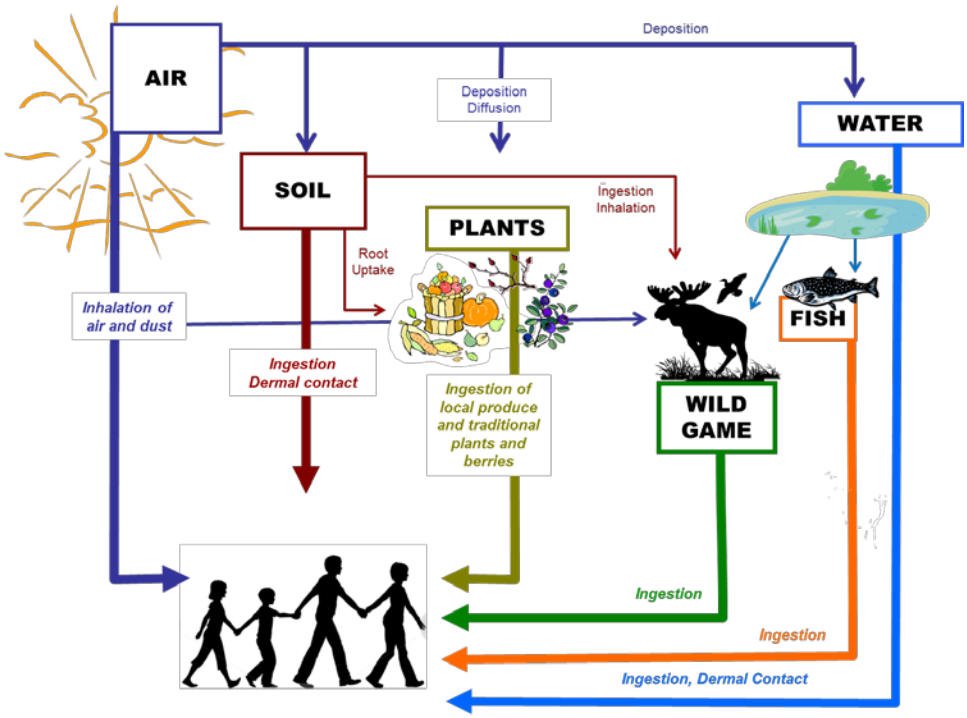


Figure 3-13 Conceptual Model of Potential Exposure Pathways for the Assessment of the Aboriginal Group

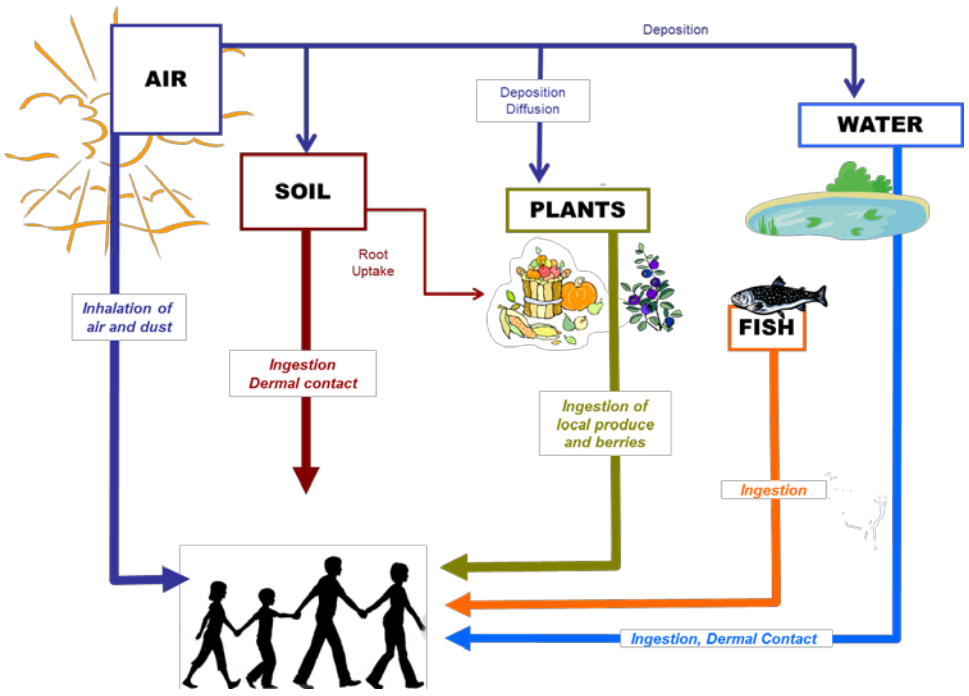


Figure 3-14 Conceptual Model of Potential Exposure Pathways for the Assessment of the Community Group

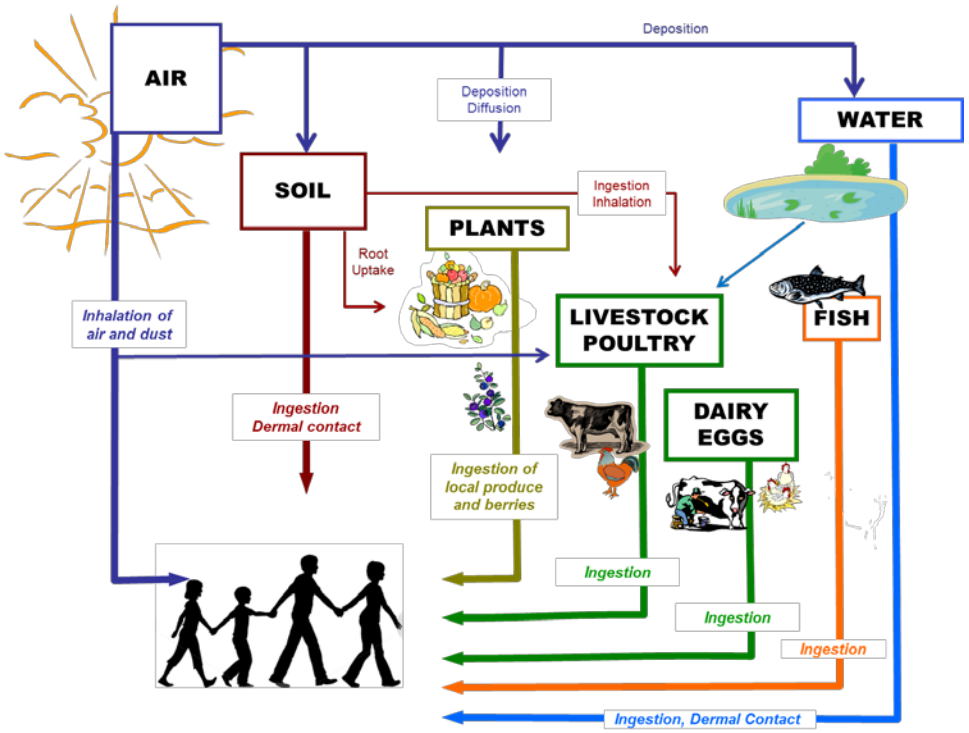


Figure 3-15 Conceptual Model of Potential Exposure Pathways for the Assessment of the Agricultural Group

3.3 Exposure Assessment

The objective of the Exposure Assessment is to estimate potential chemical exposures that might be received by people who live in the study area. To achieve this, a number of conservative or 'reasonable worst case' assumptions were made with respect to how people could be exposed to the various COPC. Due to the nature of the releases of the COPC to air from the continuous emission sources studied in the detailed HHRA, people in the area could be exposed over both the short- and long-term.

As part of the Problem Formulation step of the HHRA, it was determined that exposure could occur through both primary (inhalation) and secondary exposure pathways (*e.g.,* water, soil, food). The methods used to evaluate inhalation and multiple pathway exposures are summarized in Sections 3.3.2 and 3.3.3.

3.3.1 Estimation of Exposures via Air Dispersion Modelling

The Exposure Assessment relied on the results of air dispersion modelling of the various emissions associated with the different exposure scenarios of interest. Modelling was completed for each of the exposure scenarios as outlined below, with the model inputs configured to reflect the operating conditions that were assumed to be in effect. The modelling results reflect the predicted dispersion of emissions under the 'worst-case' of the meteorological conditions modelled. Complete details surrounding the air quality modelling can be found in Appendix A.

3.3.2 Inhalation Assessment

The exposure estimates for the inhalation pathway are based on results of the air quality dispersion modelling. The data generated from the air quality modelling consists of predicted maximum ground-level air concentrations of the COPC for each of the discrete locations identified in Section 3.2.3.4. As no adjustments for exposure were applied to differentiate between sub-populations, the inhalation exposure assessment was completed at all community locations included in the HHRA, as well as for the MPOI.

To evaluate both short-term and long-term health effects, predicted ground-level air concentrations were considered in association with various averaging periods. For the evaluation of potential short-term health risks, peak (*i.e.*, 1st highest) 10-minute, 1-hour, and 24-hour ground-level air concentrations were used to evaluate those exposures lasting 24 hours or less in duration. Annual average air concentrations were used to evaluate potential long-term health risks for the COPC.

Short-term and long-term predicted air concentrations were compared against the exposure limits described in Section 3.4 (Toxicity Assessment), according to the methods outlined in Section 3.5 (Risk Characterization).

3.3.3 Multiple Pathway Assessment

The potential for people to be exposed to COPC through secondary pathways, predictive models were used to estimate the quantity of COPC might accumulate within food, water, soil and dusts, and what concentrations of COPC people would be exposed to. The multiple pathway exposure model was used to:

- Predict concentrations of COPC in environmental media to which people might be exposed (dust, soil, water).
- Determine COPC concentrations in various types of food sources (agricultural foods, traditional plants, fish) that people in the area may eat.
- Estimate exposure from consumption of meat of game animals and birds and agricultural foods (beef, dairy, chicken, eggs).
- Estimate the total exposure to COPC via all relevant pathways.

The multiple exposure pathway model predicts environmental media concentrations of the COPC based on the deposition of the airborne COPC onto land and water. These predicted concentrations are then used to model COPC concentrations in foods. The model also estimates total human exposure, and using exposure limits, generates risk estimates.

The algorithms and input values in the model are based on published information from leading regulatory authorities including the United States Environmental Protection Agency (US EPA OSW 2005) and Health Canada (2012), as well as from several primary literature sources.

This section outlines the various steps and assumptions made within the multiple pathway assessment. A worked example is provided in Appendix D.

3.3.3.1 Physical-Chemical Screening

To assess the potential health risks associated with possible secondary pathways, it was necessary to identify those chemicals released by the selected continuous emission sources (Section 3.2.2) that would be expected to deposit onto land or water and possibly persist or accumulate in the environment in sufficient quantities for people to be exposed via soil, food and water pathways. As a starting point in this identification process, two general categories of chemicals were identified from the list of COPC:

- Gaseous chemicals, which are unlikely to contribute to human exposure via secondary pathways as they will remain airborne for prolonged periods of time and over extended distances (*i.e.*, NO_2, SO_2 and H_2S). In addition, the health effects of these gaseous chemicals are strictly related to inhalation (*i.e.*, these COPC act at their points of physical contact, like the eyes, nose or lungs). Accordingly, the gaseous chemicals were considered only in the inhalation assessment, and were removed from further consideration in the multiple pathway assessment.
- Non-gaseous chemicals, which may deposit in the study area, and persist or accumulate in the environment in sufficient quantities for people to be exposed via secondary pathways. 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 inherent properties of the chemicals that influence their fate in the environment, and subsequently their potential occurrence in the secondary pathways of exposure. This was accomplished via the process outlined below.

Comparison of Physical Chemical Properties with Established Criteria

The purpose of this step is to identify the chemicals emitted by the continuous emission sources that are non-volatile, and have a high likelihood of partitioning to environmental compartments other than air, in accordance with the following criteria from the US EPA (2003c):

- Molecular weight ≥ 200 g/mol (or 2.0×10^{2} g/mol)
- Henry's Law Constant ≤ 0.00001 atm-m³/mol (or 1.0×10^{-05} atm-m³/mol)
- Vapour pressure ≤ 0.001 mmHg (or 1.0×10^{-03} mmHg)

Comparison of Physical Chemical Properties with Established Criteria for Bioaccumulation

The purpose of this step is to identify those chemicals that have the potential to accumulate in living organisms (*e.g.,* fish, plants, invertebrates), in accordance with the following criterion from Environment Canada (2007):

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

Physical-chemical properties (*i.e.*, molecular weight, Henry's Law Constant, vapour pressure, and octanol-water partitioning coefficient) were adopted from Syracuse Research Corp. (SRC 2011) or, if a property was not available from SRC 2011, the EPI Suite program developed by US EPA (2011) was searched.

Fugacity Modelling

Fugacity modelling was completed to determine the potential relative chemical apportionment in environmental compartments other than air. This helps to determine the potential likelihood that people could be exposed via secondary pathways of exposure. Fugacity model results were based on the 'Level III' fugacity model developed by the US EPA (2011) that adheres to methods developed by MacKay *et al*. (1992, 1993). This fugacity screening was conducted on the assumption that if a chemical is expected to partition in soil, water or sediment to an extent greater than 5%, there may be a reasonable opportunity for that chemical to be present in environmental media other than air (Boethling *et al*. 2009; Environment Canada 2003).

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 COPC to deposit in the study area, and persist or accumulate in the environmental media other than air is likely negligible, and only limited opportunity exists for exposure via secondary pathways. Accordingly, these COPC were removed from further consideration in the multiple pathway assessment, and were only evaluated in the inhalation assessment. However, if a COPC met any one of these criteria, the chemical was evaluated in both the inhalation and multiple pathway assessments.

The relevant physical-chemical properties and fugacity model results for each of the COPC, along with the results of the overall physical-chemical screening process are presented in Table 3–11. [10]

The final list of chemicals assessed through multiple pathways of exposure is presented in Table 3–12.

[10] The values within this table are expressed in scientific notation. In this format, values are written are expressed either to the negative power (*i.e.*, E-x) or to the positive power (*i.e.*, E+x).

Table 3–11 Physical Chemical Screening of the Chemicals of Potential Concern for the Multiple Pathway Assessment

Chemical[1,2,3]	Chemical Abstract Service (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	≥5.0E+00	≥5.0E+00	≥5.0E+00	≥5.0E+00	
Volatile Organic Compounds									
1,3-butadiene	106-99-0	5.4E+01	7.4E-02	2.1E+03	2.0E+00	1.1E-02	3.1E-02	1.1E-04	No
Acetaldehyde	75-07-0	4.4E+01	6.7E-05	9.0E+02	-3.4E-01	1.8E+00	**1.0E+01**	2.0E-02	**Yes**
Acrolein	67-64-1	5.8E+01	4.0E-05	2.3E+02	-2.4E-01	3.3E+00	**1.2E+01**	2.4E-02	**Yes**
Benzene	71-43-2	7.81E+01	5.5E-03	9.5E+01	2.1E+00	2.9E-01	5.1E-01	5.0E-03	No
Cyclohexane	110-82-7	8.4E+01	1.5E-01	9.7E+01	3.4E+00	1.5E-02	1.5E-01	1.1E-04	No
Ethylbenzene	100-41-4	1.1E+02	7.9E-03	9.6E+00	3.1E+00	4.7E-01	2.8E-01	5.0E-03	No
Formaldehyde	50-00-0	3.0E+01	**3.4E-07**	3.9E+03	3.5E-01	**6.1E+01**	**2.3E+01**	4.0E-02	**Yes**
Isopropylbenzene	98-82-8	1.2E+02	1.1E-02	4.5E+00	3.7E+00	4.8E-01	1.9E-01	5.0E-03	No
Naphthalene	91-20-3	1.3E+02	4.4E-04	8.5E-02	3.3E+00	4.9E+00	4.8E+00	4.1E-01	No
n-Hexane	110-54-3	8.6E+01	1.8E+00	1.5E+02	3.9E+00	4.0E-03	1.0E-03	6.0E-06	No
n-Pentane	109-66-0	7.0E+01	4.0E-01	6.4E+02	2.7E+00	6.0E-03	5.0E-03	2.0E-05	No
Toluene	108-88-3	9.2E+01	6.6E-03	2.8E+01	2.7E+00	3.0E-01	3.0E-01	3.0E-03	No
1,2,3-trimethylbenzene	526-73-8	1.2E+02	4.4E-03	1.7E+00	3.7E+00	1.2E+00	6.4E-01	2.2E-02	No
1,2,4-trimethylbenzene	95-63-6	1.2E+02	6.2E-03	2.1E+00	3.6E+00	8.7E-01	4.6E-01	1.5E-02	No
1,3,5-trimethylbenzene	108-67-8	1.2E+02	8.8E-03	2.5E+00	3.4E+00	6.0E-01	3.0E-01	1.0E-02	No
m-Xylene	108-38-3	1.1E+02	7.2E-03	8.3E+00	3.2 E+00	4.0E-01	3.0E-01	5.0E-03	No
o-Xylene	95-47-6		5.2E-03	6.6E+00	3.1 E+00	6.0E-01	4.0E-01	7.0E-03	
p-Xylene	106-42-3		6.9E-03	8.8E+00	3.2 E+00	5.0E-01	3.0E-01	5.0E-01	
Polycyclic Aromatic Hydrocarbon Compounds[5]									
Benzo(a)pyrene	50-32-8	**2.5E+02**	**4.6E-07**	**5.5E-09**	**6.1E+00**	**8.2E+01**	6.8E-01	**1.6E+01**	**Yes**
Benzo(a)anthracene	56-55-3	**2.28E+02**	1.20E-05	**2.10E-07**	**5.76E+00**	**8.0E+01**	1.42 E+00	**1.6E+01**	**Yes**
Benzo(b)fluoranthene	205-99-2	**2.52E+02**	**6.57E-07**	**5.00E-07**	**5.78E+00**	**8.0E+01**	7.2E-01	**1.8E+01**	**Yes**
Benzo(k)fluoranthene	207-08-9	**2.52E+02**	**5.84E-07**	**9.65E-10**	**6.11E+00**	**8.2E+01**	7.0E-01	**1.6E+01**	**Yes**
Chrysene	218-01-9	**2.28E+02**	**5.23E-06**	6.23E-09	**5.81E+00**	**8.7E+01**	1.01 E+00	**1.1E+01**	**Yes**
Fluoranthene	206-44-0	**2.0E+02**	**8.9E-06**	**9.2E-06**	**5.2E+00**	**6.4E+01**	4.3E+00	**1.7E+01**	**Yes**
Indeno(1,2,3-c,d)pyrene	193-39-5	**2.76E+02**	**3.48E-07**	**1.25E-10**	**6.70E+00**	**8.0E+01**	5.1E-01	**1.9E+01**	**Yes**
Phenanthrene	85-01-8	1.8E+02	4.2E-05	**1.2E-04**	**4.5E+00**	**3.9E+01**	9.2E+00	**1.2E+01**	**Yes**

Notes:
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.
2 With scientific notation, values are expressed either to the negative power (*i.e.*, E-x) or to the positive power (*i.e.*, E+x).
3 **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.
4 Physical-chemical parameters for all COPC were obtained from the following sources in the order of priority: SRC (2011), US EPA (2011) (*i.e.*, EPISuite). The exception is for aliphatic and aromatic hydrocarbons where physical-chemical parameters were obtained from CCME (2008).
5 The PAHs included in the HHRA were limited to those that were identified as part of the Air Quality Assessment as being associated with an oil and gas emission source.

Table 3–12 Final List of Chemicals of Potential Concern Included in the Multiple Pathway Assessment

COPC Category	Chemicals of Potential Concern
Volatile Organic Compounds	Acetaldehyde
	Acrolein
	Formaldehyde
Polycyclic Aromatic Hydrocarbons	Benzo(a)pyrene
	Benzo(a)anthracene
	Benzo(b)fluoranthene
	Benzo(k)fluoranthene
	Chrysene
	Fluoranthene
	Indeno(123-c,d)pyrene
	Phenanthrene

3.3.4 Ambient Environmental Data

To assist in the interpretation of the predicted air quality and environmental media data, a search was performed of various databases in an effort to identify and evaluate measured ambient or background data for the study area. Information regarding this process and the data selected for use in the detailed HHRA are provided in Appendix B. As a limited amount of germane information was identified, background conditions based only on measured data could not be quantitatively evaluated. Instead, this information was used in an effort to capture potential existing health risks associated with the identified COPC in various environmental media, In order to do so, a comprehensive search of multiple databases was completed. Searches were performed in relation to measured ambient air, surface water, soil, vegetation, fish and animal tissue concentrations. To help ensure that the information was reasonably current and representative of conditions in the NE BC region that people may be exposed to, the focus of the search was on information compiled since 2000.

The databases and resources considered in this search were as follows:

- BC Ministry of Health Drinking Water Quality
- BC Environmental Assessment Office, Project Information Centre
- BC Environmental Monitoring System Database11
- BC EcoCat Ecological Reports Catalogue
- BC MOE Habitat Wizard
- BC MOE Protocol 4 for Contaminated Sites
- BC Soils Information Service
- BC Waterbucket
- BC Water Smart
- BC Water Resources Atlas
- BC MOE Water Quality

[11] The BC EMS database includes the Land Resource Data Warehouse (LRDW), GeoBC Data Discovery, and BC Geographic Warehouse databases.

- BC MOE Terrestrial Ecosystem Information
- Data BC
- Environment Canada GENIE Database
- Environment Canada OpenData
- Environment Canada National Air Pollution Surveillance
- FlowWorks
- Geoscience BC
- Northern Health
- Water Environment Hub

In addition to the above list of resources, the websites for various communities within the study area were evaluated for the presence of drinking water monitoring data. Although drinking water quality data are available for some communities, information on the COPC assessed as part of the HHRA was not available (as it was focused primarily on metals).

Overall, a very limited amount of data was available for the COPC for environmental media in the region. A brief summary of the database findings is presented in Table 3-13 in association with the above list of resources.

Table 3–13 Summary of Database and Resource Search Findings

Database / Resource	Findings
BC Air Quality Archive	Measured air quality data for various locations in province. Limited data for COPC available.
BC Ministry of Health Drinking Water Quality	Primarily information relating to water quality guidelines and regulation. No measured chemical data available.
BC Environmental Assessment Office, Project Information Centre	Focus of database and site information is on water management. No measured chemical data available.
BC Environmental Monitoring System Database[12]	Queries performed for air, soil, vegetation, water, wildlife tissue and fish tissue. Limited amount of PAH data in soils available for Peace region. Some animal and fish tissue concentration data available, but limited to metals. Some air quality information available for a very limited number of COPC.
BC EcoCat Ecological Reports Catalogue	Contains reports from a variety of disciplines, including aquatic species and habitats, terrestrial species and habitats, floodplain mapping, reservoirs, ground water and vegetation. Fish tissue data identified for pre-2000 for metals. No other measured chemical data available.
BC MOE Habitat Wizard	Contains information about fish and fish habitat. No measured chemical data available.
BC MOE Protocol 4 for Contaminated Sites	Under the Contaminated Sites Regulation, BC MOE presents regional 'background' soil quality information. Regional background soil quality estimates are limited to inorganic substances. BC MOE does not plan to expand its background soil quality inventory beyond inorganic substances.
BC Soils Information Service	Soil database from 1960s to 1980s. No comment about more recent data on website.
BC Waterbucket	Information relates to water management and sustainability. No measured chemical data available.
BC Water Smart	Provides links to several references. Focus is on water use and management. No measured chemical data available.

[12] The BC EMS database includes the Land Resource Data Warehouse (LRDW), GeoBC Data Discovery, and BC Geographic Warehouse databases.

Database / Resource	Findings
BC Water Resources Atlas	Provides information related to the water resources BC, such as watersheds, water quantity and quality monitoring sites, aquifers, water wells and flood protection works. No measured chemical data available.
BC MOE Water Quality	Contains links to water quality and sediment reports for various communities in NE BC, but majority of links were dead ends. No measured chemical data could be obtained.
BC MOE Terrestrial Ecosystem Information	Contains several data bases, seems to be focused on GIS and ecosystem mapping
Data BC	Some water quality information available for Fort St. John and Dawson Creek.
Environment Canada GENIE Database	Parameters monitored and reported in database limited to basic water quality monitoring data. No chemical data for BC available.
Environment Canada NAPS database	Some Criteria Air Contaminant (CAC) data available.
Environment Canada OpenData	Contains various air and water quality data. No relevant water quality information identified. Some Province-wide ambient air data, but no region-specific information.
FlowWorks	On line subscription based data sharing services for industry and government. Invitation-basis only. Seems to be related to data submission.
Geoscience BC	Water quality studies, primarily for ground water in shale gas producing areas. Parameters monitored relevant to hydrogeology, but not to human health.
Northern Health	NH regulates water supply systems. Data in EMS. No information for COPC.
Water Environment Hub	Presents a list of links to resources, many of them included in this summary Table. No relevant data.

3.3.5 Exposure Modelling Method

A general summary of the approaches used in the HHRA to estimate the exposure of people in the study area is presented in Table 3–14, for each exposure pathway.

Table 3–14 Summary of Approaches Used in the Exposure Assessment

Media	Description
Air	Air dispersion modelling incorporated meteorological data that represented conditions contributing to maximum predicted ground-level air concentrations of the COPC. Ground-level air concentrations were predicted for the selected continuous airborne emission sources at the various discrete locations, as well as for the entire study area grid. Maximum predicted air concentrations (*e.g.,* the MPOI for the entire study area) were used to represent exposure concentrations to which wildlife and livestock could be exposed, to help ensure that the greatest possible exposures were captured. In addition, the maximum predicted air concentration of the COPC at each community location was considered to permit the prediction of location-specific estimates of exposure and risk.
Soil	Background concentration data for some PAHs were available for soils in the NE BC region. Due to the limited sample size and high number of non-detectable results, this information was not used in the prediction of human exposure estimates for the study area. Additional information is provided in Appendix B. In general, predicted soil concentrations were estimated based on: • the highest predicted annual concentrations of the COPC for the region • 80-years of deposition • chemical losses due to degradation and volatilization All residents were assumed to have direct exposure to soils via incidental ingestion and to dust via inhalation.

Media	Description
Vegetation	No data were identified for the COPC in vegetation from the region. Additional information regarding the databases searched is provided in Appendix B. Concentrations of above-ground plant and browse were predicted based on: • the highest predicted annual concentrations of the COPC for the region • 80-years of deposition • vapour uptake • root uptake from soil Concentrations of below-ground plants (*i.e.*, rat and willow roots, below-ground garden vegetables) were predicted from soil concentrations. The multiple pathway assessment did not make any adjustments for washing or peeling of plant foods.
Water	No data were identified for the COPC in surface water or drinking water from the region. Additional information regarding the databases searched is provided in Appendix B. Alternatively, airborne deposition to surface water was used to predict COPC concentrations in water to which people, terrestrial wildlife, livestock and fish could be exposed. The surface water body within the study area that was selected for the purpose of predicting exposures related to drinking water, swimming and fish consumption for the Aboriginal and Community locations was Charlie Lake. Charlie Lake served as the primary drinking water source for the largest community in the region, Fort St. John until 1997. Currently, Fort St. John obtains water from a groundwater source. This lake was selected, as it represents one of the largest surface water bodies in the study area (the next largest being Moberly Lake), and is located in an area with a relatively high density of continuous oil and gas emission sources and population. This lake was used as a surrogate in the HHRA in the prediction of deposition of the COPC to surface water. Water quality data for the COPC were not available, and measured data were not integrated into the HHRA for this lake as a result. Hydrologic information for this lake was obtained from the BC OGC NEWT database and other literature sources. Additional information is available in Appendix C. For the Agricultural group, it was assumed that both residents and livestock animals would obtain drinking water from a dugout or slough. The parameters for a generic agricultural dug out were obtained from the BC Ministry of Agriculture and other sources as appropriate. The concentrations of the COPC in this dugout were predicted in the multiple exposure assessment through the consideration of deposition from air to the water surface.
Agricultural	No data were identified for the COPC in agricultural animal tissues (beef, milk, chicken, eggs) from the region. Additional information regarding the databases searched is provided in Appendix A. Agricultural animal tissue was based on: • the highest predicted annual average air concentrations of the COPC • ingestion of soil, soil invertebrates (where appropriate) • ingestion of water and aquatic plants (where appropriate) • chemical loss due to elimination of the COPC within animals
Wild game	No data were identified for the COPC in game animal or bird tissues from the region. Additional information regarding the databases searched is provided in Appendix B. Wild game (large and small, mammal and bird) concentrations were predicted in the multiple pathway model based on: • the highest predicted annual average air concentrations of the COPC • ingestion of soil, soil invertebrates (where appropriate) • ingestion of water, and terrestrial or aquatic plants (where appropriate) • chemical loss due to elimination of the COPC within animals
Fish	No data were identified for the COPC in fish tissue from the region. Additional information regarding the databases searched is provided in Appendix B. Fish tissue concentrations were predicted in the multiple pathway model from predicted surface water concentrations.

3.3.6 Development of Exposure Estimates

In recognition of the potentially unique exposure patterns that may result in association with differences in behaviour characteristics, the exposure assessment has taken into consideration

such factors as the practice of traditional lifestyles, reliance on local agricultural foods, urban lifestyles and potential differences in time spent in the area. Descriptions of the various assumptions made for each group of individuals included in the multiple pathway assessment of the HHRA are provided in Section 3.3.6.1 to Section 3.3.6.4.

No distinction beyond community name was made between people in the area in the inhalation assessment, as no lifestyle group adjustments were made either on a short-term or long-term basis. The air inhalation rates within the tables in Section 3.3.6.1 to Section 3.3.6.4 were used to predict dust exposures. In addition, in order to calculate potential human exposures, various physical characteristics were used to represent people of various ages (Table 3-15).

Table 3–15 Assumed Physical Characteristics for All Individuals

Parameter	Life Stage Characteristics (Health Canada 2012)[1]				
	Infant	Toddler	Child	Adolescent	Adult
Body weight [kg]	8.2	16.5	32.9	59.7	70.7
Body surface area [cm²]					
Hands	320	430	590	800	890
Arms	550	890	1,480	2,230	2,500
Legs	910	1,690	3,070	4,970	5,720
Total body	3,620	6,130	10,140	15,470	17,640
Skin adherence factor [g/cm²/day]					
Hands	1.0E-07	1.0E-07	1.0E -07	1.0E-07	1.0E-06
Surfaces other than hands	1.0E-08	1.0E-08	1.0E -08	1.0E-08	1.0E-07

Notes:
1 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).

3.3.6.1 Aboriginal Resident

The study area includes a number of First Nation communities, and groups of individuals who identify themselves as being Aboriginal live within other communities in the study area (Section 3.2.1). While it is not known with certainty what percentage of the population within the entire NE includes traditional food items in their diet, the default assumption in the HHRA was that all Aboriginal Residents consumed a diet high in traditional and local foods.

For the assessment of the Aboriginal Residents, food consumption data, such as the type of traditional foods that are known to be consumed in the study area and the frequency and rate of consumption, were obtained from the First Nations Food Nutrition and Environment Survey (FNFNES) (Chan *et al*. 2011) and from Health Canada (*Guidance on Human Health Preliminary Quantitative Risk Assessment*, Health Canada 2012).

Conducted in 2008 to 2009, the FNFNES study included several First Nations communities within BC, including a number that fall within the HHRA study area. For the purposes of the FNFNES study, the Province was subdivided into five 'ecozones', which represented regions that are physically separated by large features such as oceans, deserts or mountain ranges (Chan *et al*. 2011). The FNFNES ecozones that are of relevance to the HHRA study area are:

- Boreal Plains/Subarctic (ecozone 2). This area includes the Doig River, Blueberry River, Halfway River, and Saulteau/East Moberly Lake First Nations).

- Montane Cordillera/Subarctic (ecozone 4). This area includes the West Moberly Lake First Nation).

In total, over 1,100 interviews of First Nation people were completed. Of the First Nations located within the HHRA study area, the Doig River and Saulteau (East Moberly) First Nations participated in the study. Food consumption information was collected through the use of 24-hour food recall surveys and food frequency questionnaires.

Some of the FNFNES (Chan *et al*. 2011) study findings for ecozones 2 and 4 that are relevant to the HHRA are:

- The percentages of on-reserve participants who reported consuming fruit and vegetables from home or community gardens were 90% (ecozone 2) and 18% (ecozone 4). Traditional plant harvesting was reported by 48% and 38% of participants within ecozones 2 and 4, respectively. Commonly consumed berries in these two ecozones included: blueberries, raspberries, blue huckleberry, wild strawberry, Saskatoon berries, and cranberries. Labrador tea, rat root, and miscellaneous other plants were the most commonly consumed wild plants. Tree-based food use appeared to be higher in ecozone 4, with the bark from balsam and various other tree species being consumed.
- A total of 66% of participants in ecozone 2 and 48% of participants in ecozone 4 reported hunting or setting snares for wildlife. Within these ecozones, the most commonly consumed land mammals included moose, deer and elk. For wild birds, grouse were the most commonly consumed.
- Fishing was practiced by 37% of participants in ecozone 2 and 55% of participants in ecozone 4. The most commonly reported freshwater fish species consumed in both ecozones 2 and 4 were various species of salmon and trout, with less frequent consumption of other species.

Chan *et al*. (2011) presented serving size data, which allowed for the consumption of ecozone-specific consumption rates for various traditional foods. In addition, Chan *et al*. (2011) provides average and 95th percentile consumption rates for all adults (male and female) for all of the First Nations who participated in the study as a whole. Consumption information was collected for the most commonly consumed traditional food items in ecozones 2 and 4 to help ensure that the HHRA is as representative as possible of the traditional diets that might be consumed by First Nations communities within the study area. These traditional food items were divided into the following categories for the purposes of the HHRA:

- Large game mammals (moose meat and kidney, deer, elk)
- Wild birds (grouse)
- Berries (blueberries, raspberries, wild strawberries, huckleberries, soapberries, Saskatoon berries and cranberries)
- Wild aboveground plants (Labrador tea, balsam pitch and bark)
- Fish (salmon and trout)

Although small game mammals and wild belowground plants were not listed among the most frequently consumed wild foods for ecozones 2 and 4, they were still included in the HHRA. This was done based on the study team's experience working with First Nations communities. It also ensures that the exposure assessment was comprehensive.

Health Canada (2012) provides wild game consumption rates for Canadian First Nations populations, based on 24-hour recall data collected in 1971 and 1972 as part of the Nutrition Canada Survey. The 24-hour recall survey was conducted in person by trained interviewers using models of meal portions to determine quantities consumed. More than 2,000 Aboriginal individuals (First Nations and Inuit) were interviewed as part of the Nutrition Canada Survey. Summary data are provided by Health Canada for wild game 'eaters only', which exclude individuals reporting no wild game consumption. Using statistics for eaters only helps to ensure that the consumption rates of the individuals who consume the majority of the wild game and fish harvested are not under estimated. The consumption rate data from Chan *et al*. 2011 was given preference in this HHRA over the Health Canada consumption rates, as it is more recent, and included communities within the study area and nearby areas of the Province. However, the Health Canada First Nations information was used to adjust consumption rate data from Chan *et al*. 2011 for the various age groups.

The consumption rate information for the HHRA was selected for the Aboriginal Residents, for the traditional food items identified as being relevant to ecozones 2 or 4, using the following hierarchy:

- Preference was given to the 95th percentile consumption rates from Chan *et al*. (2011) for all adults and for all ecozones for the traditional food items that were identified by the study team as being relevant to the two ecozones that apply to the HHRA study area.
- In the event that a 95th percentile consumption rate for a relevant food item was not available, a consumption rate was calculated using ecozone-specific consumption frequency data (in grams per day) and serving size (in grams) from Chan *et al*. (2011).

As it was reported that a high proportion of Aboriginal individuals in the study area consume local home-grown or community-grown foods, the consumption of aboveground leafy vegetables and root vegetables were also considered in addition to traditional food exposures.

It was assumed that Aboriginal Residents consumed local surface water as a drinking water source, and inadvertently ingested water while swimming in local water bodies at rates consistent with Health Canada (2012) and US EPA (2003c) guidance. It is also probable that people will consume small amounts of soil and dusts due to inhalation, hand-to-mouth behaviours (*i.e.,* toddler) or through food consumption. Air and soil exposure rates were obtained for the various life stages from Health Canada (2012).

The assumed rates of exposure for the Aboriginal Residents to various environmental media and food items are presented in Table 3–16. For the physical characteristics for the various life stages see Table 3-15.

Table 3–16 Assumed Exposure Rates for Aboriginal Residents

	Life Stage					Units	Reference
	Infant[1]	Toddler	Child	Adolescent	Adult		
Media							
Air inhalation rate	2.2	8.3	14.5	15.6	16.6	m^3/day	Health Canada 2012
Drinking Water	0.3	0.6	0.8	1.0	1.5	L/day	Health Canada 2012
Incidental intake of water (swimming)[2]	0	0.05	0.05	0.025	0.025	L/hour	US EPA 2003c
Soil ingestion[3]	0.02	0.08	0.02	0.02	0.02	g/day	Health Canada 2012
Wild game							
Large game mammals[4] (moose meat and kidney, deer, elk)	0	46	67	94	145	g/day	Chan *et al*. 2011; Health Canada 2012
Small game mammals[4] (rabbits)	0	0.9	1.4	1.9	3	g/day	Chan *et al*. 2011; Health Canada 2012
Wild birds[4] (ruffed grouse)	0	0.31	0.46	0.65	1	g/day	Chan *et al*. 2011
Fish[5] (salmon, trout)	0	21	70	84	84	g/day	Chan *et al*. 2011
Wild berries[6]	2.5	14	25	28	31	g/day	Chan *et al*. 2011
Wild traditional plants							
Above-ground[7] (Labrador tea, balsam pitch and bark)	0	0.23	0.46	1	1	g/day	Chan *et al*. 2011; Health Canada 2012
Below-ground[7] (rat root)	0	0.005	0.01	0.02	0.02	g/day	Chan *et al*. 2011; Health Canada 2012
Garden Produce							
Above-ground leafy vegetables	72	67	98	120	137	g/day	Health Canada 2012
Below-ground root vegetables	83	105	161	227	188	g/day	Health Canada 2012

Notes:
1 An infant's diet was assumed to be supplemented with breast milk using a consumption rate of 664 g/day (O'Connor and Richardson 1997).
2 Assumed that people could swim in surface water 1 hour/day, 90-days/year
3 Assumed that people could be exposed to soils 365 days/year
4 The 95th percentile consumption rate for large and small game animals and birds for all adult Aboriginals from Chan *et al*. 2011 was adjusted according to the ratios presented in Health Canada (2012) for wild game in order to calculate the consumption rates for the other age groups. The adult rate of 145 g/day was multiplied by 0.65 (adolescent), 0.46 (child), toddler (0.31).
5 The fish consumption rates for the other life stages were calculated from the proportions provided in the Food Directorate publication regarding mercury in fish and the health benefits of fish consumption (Health Canada 2007). Health Canada (2007) reported that children and toddlers consume 83% and 50% of the fish consumed by an adult, respectively. No proportion was given for the adolescent (Health Canada 2007); therefore, for this assessment, it is assumed that the teen and adult consume the same amount.
6 The wild berry consumption rates for the other life stages were calculated using the proportions provided in Health Canada (1994) for plums, grapes, cherries, strawberries, blueberries and jams combined, which suggests that adolescents, children, toddlers and infants consume 90%, 82%, 44% and 8% of the wild berries consumed by an adult, respectively.
7 The consumption rates for children and toddlers were calculated based on a body weight ratio *e.g.,* 32.9 kg/70.7 kg or 16.5 kg/70.7 kg, from Health Canada (2012), as no age specific information is available for comparison.

3.3.6.2 Agricultural Resident

Smaller, more rural communities that do not represent First Nation communities were generally assumed to be Agricultural. This assumption was made to help ensure that a conservative exposure estimate for people who live in these smaller communities was determined, as the

Agricultural Resident presumably consumes a much higher proportion of local foods than the Community Resident.

Within the NE BC region, the farming of crops such as wheat, barley, canola, oats, various forage crops and vegetables is prevalent. There are also commercial cattle, game and exotic livestock farming operations in the area (FBC 2012; BC ACF 2008). As a result, it is possible that people living outside the larger communities and First Nations traditional territory in the study area may be exposed to the COPC through the consumption of local agricultural foods (beef, chicken, dairy, fruit, vegetables). To capture potential exposures that might be received by agricultural residents who consume a diet with a high proportion of local foods, the study team assumed that residents in this group consume local foods exclusively.

Food consumption patterns for the Agricultural Residents were obtained from Health Canada's Guidance on Human Health Preliminary Quantitative Risk Assessment for the Canadian general population (Health Canada 2012) and Health Canada's Human Health Risk Assessment for Priority Substances (Health Canada 1994).

For some food items, including beef, poultry, dairy, eggs and fruit, consumption rates were based on 24-hour recall data collected in 1970 and 1972 as part of the NCS (Health Canada 1994). Although more recent 24-hour food recall data are available as part of the Canadian Community Health Survey (CCHS 2004), these data are not readily available. In addition, a recent comprehensive evaluation of the CCHS data by the Public Health Agency of Canada (PHAC 2012) indicated that the data set is not appropriate for the estimation of multi-day exposures based on a statistical analysis that evaluated the extrapolation of the 24-hour recall data over 3-, 5- and 7-day periods. The data was not found to be reliably extrapolated for periods longer than 24-hours. The consumption rates for garden vegetables in this HHRA were based on the above-ground and root vegetable ingestion rates recommended for the Canadian general population published by Health Canada (2012).

Fish consumption values for the Agricultural Residents were obtained from a study completed by Health Canada's Food Directorate (2007) involving fish consumption. In Health Canada (2007), fish consumption rates of 40 g/day, 33 g/day and 20 g/day were used to represent adult, child and toddler fish intakes. These consumption rates were obtained from a Market Facts of Canada (1991) study on national seafood consumption, and a Health Canada Bureau of Chemical Safety evaluation of current intake rates by Canadian consumers (BCS 2004).

The assumed exposure rates for the Agricultural Resident for environmental media and food are presented in Table 3–17. For the assumed physical characteristics for the various life stages see Table 3-15.

Table 3–17 Assumed Exposure Rates for Agricultural Residents

	Life Stage					Units	Reference
	Infant[1]	Toddler	Child	Adolescent	Adult		
Media							
Air inhalation rate	2.2	8.3	14.5	15.6	16.6	m^3/day	Health Canada 2012
Drinking Water	0.3	0.6	0.8	1.0	1.5	L/day	Health Canada 2012
Incidental intake of water (swimming)[2]	0	0.05	0.05	0.025	0.025	L/hour	US EPA 2003c
Soil ingestion[3]	0.02	0.08	0.02	0.02	0.1	g/day	Health Canada 2012

	Life Stage					Units	Reference
	Infant[1]	Toddler	Child	Adolescent	Adult		
Agricultural Foods and Garden Produce							
Fish	0	20	33	40	40	g/day	Health Canada 2007
Beef[4]	0	39	57	95	99	g/day	Health Canada 1994
Chicken[5]	0	13	17	20	21	g/day	Health Canada 1994
Dairy[6]	0	677	622	590	297	g/day	Health Canada 1994
Eggs	0	24	21	22	32	g/day	Health Canada 1994
Fruit[7] (berries)	5	40	69	56	46	g/day	Health Canada 1994
Vegetables							
Above-ground leafy plants	72	67	98	120	137	g/day	Health Canada 2012
Below-ground (roots)	83	105	161	227	188	g/day	Health Canada 2012

Notes:
1 An infant's diet was also assumed to be supplemented with breast milk using a consumption rate of 664 g/day (O'Connor and Richardson 1997).
2 Assumed that people could swim in surface water 1 hour/day, 90-days/year
3 Assumed that people could be exposed to soils 365 days/year
4 Based on the sum of consumption rates for steak, roast and stewing beef, ground beef, pork (fresh and cured), lamb and veal.
5 Based on the consumption rate for poultry, chicken and turkey.
6 Based on the sum of consumption rates for whole milk, 2% milk, skim milk, evaporated milk, cream, ice cream, yogurt, cheese, cottage cheese, processed cheese and butter.
7 Based on the sum of consumption rates for apples, applesauce, cherries, strawberries, blueberries, jams and honey.

3.3.6.3 Community Resident

The larger non-First Nation communities in the study area (having approximately 1,000 residents or more) include:

- Fort St. John
- Dawson Creek

For the purpose of the HHRA, Community Residents were considered to be individuals who reside within these larger communities on a long-term basis.

Food consumption rates for the Community Residents were obtained from Health Canada (2012, 2007).

A comprehensive assessment of the consumption of agricultural and traditional foods is included in the HHRA through the assessment of the Agricultural and Aboriginal Residents. Accordingly, it was assumed that the Community Residents would consume a diet high in supermarket foods, with some local fish, berry and produce consumption (*i.e.*, from a home or community garden).

Like the Agricultural Residents, fish consumption values for the Community Residents were obtained from a study completed by Health Canada's Food Directorate (2007) involving fish consumption.

Based on CCME (2006) guidance, the ingestion rates of leafy and root vegetables recommended for the Canadian general population by Health Canada (2012) were adjusted to reflect the smaller portion of the home-garden produce that a resident living in an urban

environment might obtain locally. Specifically, the CCME (2006) suggests that residents may consume as much as 10% of their produce (fruits and vegetables) from a home-garden. Using this approach, the vegetable consumption rates from Health Canada (2012) were adjusted for all age groups.

Fruit consumption rates assumed for the Community Residents are based on 24-hour recall data collected in 1970 and 1972 as part of the Nutrition Canada Survey (NCS), and presented in a publication by Health Canada (1994). The dietary survey involved a statistically representative sample of the Canadian population, personal interviews conducted by trained interviewers, and models of meal portions to assist in determining food portion sizes for approximately 180 different foods. Food consumption data for the recommended life stages were compiled by the Food Directorate of the Department of National Health and Welfare into 112 individual food composites. For the purpose of this HHRA, these data were grouped to estimate the consumption rates that would be representative of the locally grown or harvested fruits that might be consumed by Community Residents. For example, the consumption rates reported for apples, applesauce, cherries, strawberries, blueberries, jams and honey were summed to estimate the fruit consumption rate. These fruit consumption rates were adjusted to reflect the smaller portion of the home-garden produce (10%) that a resident living in an urban environment might obtain locally.

The exposure rates assumed for the Community Resident for environmental media and food are presented in Table 3–18. For the physical characteristics for the various life stages see Table 3-15

Table 3–18 Assumed Consumption Rates for the Community Resident

	Life Stages					Units	Reference
	Infant[1]	Toddler	Child	Adolescent	Adult		
Media							
Air inhalation rate	2.2	8.3	14.5	15.6	16.6	m^3/day	Health Canada 2012
Drinking water	0.3	0.6	0.8	1.0	1.5	L/day	Health Canada 2012
Incidental intake of water (swimming)[2]	0	0.05	0.05	0.025	0.025	L/hour	US EPA 2003c
Soil ingestion[3]	0.02	0.08	0.02	0.02	0.02	g/day	Health Canada 2012
Fish and Garden Produce							
Fish[4]	0	20	33	40	40	g/day	Health Canada 2007, 2012
Fruit[5] (including berries)	0.1	4	7	6	5	g/day	CCME 2006; Health Canada 1994
Garden Produce[6]							
Above-ground (leafy vegetables)	7	7	10	12	14	g/day	CCME 2006; Health Canada 2012
Below-ground (root vegetables)	8	11	16	23	19	g/day	CCME 2006; Health Canada 2012

Notes:
1 Infant's diet was assumed to be supplemented with breast milk using a consumption rate of 664 g/day (O'Connor and Richardson 1997).
2 Assumed that people could swim in surface water 1 hour/day, 90-days/year
3 Assumed that people could be exposed to soils 365 days/year
4 The fish consumption rates for the other life stages were calculated from the proportions provided in the Food Directorate publication regarding mercury in fish and the health benefits of fish consumption (Health Canada 2007). Health Canada (2007) reported that children and toddlers consume 83% and 50% of the fish consumed by an adult, respectively. No proportion was

given for adolescents (Health Canada 2007). For this HHRA, it is assumed that adolescents and adults consume the same quantities.

5 Based on the sum of consumption rates for apples, applesauce, cherries, strawberries, blueberries, jams and honey provided by Health Canada (1994), but assumed only 10% of the fruit would be obtained from their home-garden, as per CCME (2006) guidance.

6 Based on the consumption rates for root vegetables and other vegetables provided by Health Canada (2012), but assumed only 10% of the vegetables would be obtained from their home-garden, as per CCME (2006) guidance.

3.3.6.4 Maximum Point of Impingement (MPOI)

The inhalation assessment for this group captures potential worst-case exposures to the maximum ground-level concentrations of each of the COPC. The multiple pathway assessment for each the Aboriginal, Agricultural and Community groups included an assessment of the MPOI concentrations for the selected COPC, to provide an estimate of worst-case long-term possible exposures.

3.4 Toxicity Assessment

The Toxicity Assessment involves the identification of potential adverse health effects that may be associated with exposure to each of the identified COPC, and the conditions under which these effects might be observed.

Chemicals may differ not only with respect to the dosage required to cause an adverse effect, but also in the mechanism by which the adverse effect is elicited. For this reason, two general categories were used to evaluate the chemical emissions based upon their mode of action or mechanism of toxicity: threshold and non-threshold.

In the case of threshold chemicals, which are generally non-carcinogenic chemicals, a benchmark or threshold level must be exceeded for toxicity to occur. The degree of toxicity expressed then generally increases with increasing dose. The threshold phenomenon applies to virtually all types of toxic responses and chemicals, with the exception of some carcinogens and some forms of cancer. For these chemicals, a no-observed-adverse-effects level (NOAEL) can often be identified. A NOAEL is the dose or amount of the chemical that results in no obvious response in the most sensitive test species and test endpoint. Depending on the type of data available for an endpoint, a benchmark dose or concentration (BMD or BMC) may be derived, typically from dose-response modelling. A BMD or BMC represent specific response levels (*i.e.,* 5%, 10% of the study population) near the low end of the observable range of the data. The use of BMD/BMC is becoming more of an alternative to the conventional use of NOAELs or lowest-observed-adverse-effect levels (LOAELs) in the assessment of potential non-cancer health effects. The approaches used to calculate an exposure limit by the various agencies was evaluated in the toxicity assessment, with preference being given to values derived from a BMD/BMC approach over a NOAEL- or LOAEL-based approach (US EPA 2012). For the assessment of non-cancer effects, preference was given to BMD/BMC based values, as these values take into account the whole dose-response curve observed for a particular chemical within a study population, where a NOAEL or LOAEL represents only one dose level.

Carcinogens are considered non-threshold chemicals. They are capable of producing cancer through one or more of a number of possible mechanisms (*e.g.*, mutagenicity, cytotoxicity, inhibition of programmed cell death, mitogenesis (uncontrolled cell proliferation) and immune suppression) that, in theory, do not require the exceedance of a threshold (US EPA 2005). In general, tumorigenicity data from animals or human epidemiological studies are evaluated and examined using mathematical models to determine the chemical-specific unit risks or slope

factors, which are in-turn used to develop applicable exposure limits. Regulatory agencies such as Health Canada and the US EPA assume that any level of long-term exposure to carcinogenic chemicals is associated with some 'hypothetical cancer risk'. As a result, Health Canada has specified an incremental lifetime cancer risk (ILCR) (*i.e.*, over and above background) of 1.0 in 100,000 to be acceptable, tolerable or essentially negligible (Health Canada 2012). The regulatory benchmark of an acceptable cancer risk is policy-based and its interpretation by various regulatory agencies differs (CCME 2006).

An assumed incremental cancer risk of 1.0 in 100,000 increases a person's lifetime cancer risk from 0.40000 for women (based on the 40% lifetime probability of developing cancer in Canada) to 0.40001, and 0.45000 for men (based on the 45% lifetime probability of developing cancer in Canada) to 0.45001 (CCS 2010). 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 cancer risks (Wilson 2005).

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

- **Reference Concentration (RfC)** – refers to the safe level of an airborne chemical for which the primary avenue of exposure is inhalation. It is expressed as a concentration of the chemical in air (*e.g.*, µg/m^3) and applies only to threshold chemicals.
- **Reference Dose (RfD)** – refers to the safe level or dose of a chemical for which exposure occurs through secondary pathways (*i.e.*, oral and dermal). It is most commonly expressed in terms of the total intake of the chemical per unit of body weight per day (*e.g.*, µg/kg bw/d). This term applies only to threshold chemicals.

For threshold response chemicals, typically non-carcinogens, a point of departure (POD) is derived (typically a BMD/BMC, NOAEL OR LOAEL). Uncertainty factors are then applied to the POD by up to several thousand-fold, in part to accommodate the need to protect sensitive individuals. Exposure limits derived for threshold-response chemicals are called reference concentrations (RfC), reference doses (RfD), acceptable daily intakes (ADI), tolerable daily intakes (TDI) or permissible daily intakes (PDI). These limits are calculated as follows:

$$\text{Exposure Limit} = \frac{\text{Point of Departure}}{\text{Uncertainty Factor(s)}}$$

For non-threshold Response Chemicals, typically carcinogens, any exposure greater than zero is assumed to have a non-zero probability of causing some type of response or damage. This relationship is typically used for chemicals, which can cause cancer by damaging genetic material. Under a 'non-threshold' assumption, any exposure has some potential to cause damage, so it is necessary to define an 'acceptable' level of risk associated with these types of exposures. The acceptable level of risk is an issue of policy rather than a scientific decision and is set by regulatory agencies. These types of exposure limits are defined in one of several ways:

- **Inhalation unit risk (IUR)**: The US EPA defines a unit risk value as "…the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m³ in air…". A unit risk value of 3.0 x 10^{-5} per µg/m³ would mean that under an upper worst-case estimate, three excess cancer cases are expected to develop per one hundred thousand (100,000) people, if exposed every day for a lifetime to 1 µg of the chemical per m³ of air.
- **Cancer slope factor (SF)**: The US EPA defines a cancer slope factor (SF) as "…[a]n upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg-day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than 1 in 100."
- **Risk-specific Concentration (RsC)** – reserved for non-threshold or carcinogenic chemicals and refers to the level of an air-borne carcinogen for which the primary route of exposure is inhalation that results in a regulatory acceptable incremental increase in cancer (1.0 in 100,000 in Canada). The RSC is expressed as a concentration of the chemical in air (*e.g.*, µg/m³).
- **Risk-specific Dose (RsD)** – reserved for non-threshold or carcinogenic chemicals and refers to the dose of a carcinogen for which exposure occurs through secondary pathways that results in a regulatory acceptable increased incidence of cancer (1.0 in 100,000 in Canada). The RsD is expressed in terms of the total daily intake of the chemical (*e.g.*, µg/kg bw/d).

3.4.1 Identification of Exposure Limits

The term 'exposure limit' refers to the level of exposure or dose of the chemical that is unlikely to produce adverse health effects in humans. Exposure limits are deliberately intended to be protective of even individuals who might be especially vulnerable to chemical exposures. A considerable amount of conservatism is typically incorporated into the derivation of these values. The limits are routinely calculated on the basis of the most sensitive health endpoint in the most sensitive species.

As described, exposure limits typically embrace a high degree of conservatism, in effort to recognize the mandate of most of the authorities to protect public health, including the health of infants and children, the elderly, and individuals who might be especially vulnerable to chemical exposures. As a result, because of the conservatism involved, an exceedance of the exposure limit does not necessarily mean that health effects are certain or imminent.

Exposure limits are often segregated into different categories in recognition of the fact that the appearance and nature of toxic responses are very much dependent on the frequency and duration of exposure. Two general categories of exposure limits were included in the HHRA:

- **Acute Exposure Limit:** refers to the amount, concentration 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:** refers to the dose of a chemical that can be tolerated without evidence of adverse health effects on a long-term basis. These limits are routinely applied to conditions in which exposures extend over several months or years, possibly up to a lifetime.

Both acute and chronic exposure limits were utilized in light of the need to address and differentiate between the potential health effects that could result from short-term and long-term exposures to the COPC in the study area. Reliance was placed on exposure limits developed or recommended by regulatory authorities or reputable scientific authorities as criteria (*e.g.*, objectives, guidelines or standards) for the protection of human health. The use of regulatory limits is a common practice among practitioners of risk assessment.

3.4.2 Process for the Selection of Exposure Limits

Exposure limits for use in the HHRA were selected from a number of regulatory or reputable scientific agencies. Exposure limits were evaluated and selected from various organizations, including:

- BC Ministry of the Environment (BC MOE)
- Health Canada
- United States Environmental Protection Agency (US EPA)
- World Health Organization (WHO)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- Netherlands National Institute of Public Health and the Environment (RIVM)
- California Office of Environmental Health Hazard Assessment (OEHHA)
- Ontario Ministry of the Environment (OMOE)
- Texas Commission on Environmental Quality (TCEQ)
- American Conference of Governmental Industrial Hygienists (ACGIH)

By definition, exposure limits may include standards, guidelines, objectives, reference concentrations or doses, cancer risk estimates, or other limits that have been derived for the protection of human health.

To be selected for use in the HHRA, exposure limits were selected that complied with the following criteria:

- Established or recommended by a reputable scientific or regulatory agency
- Supported by adequate documentation
- Protective of the health of the general public based on current scientific knowledge of the health effects associated with exposure to the chemical
- Protective of sensitive individuals (*i.e.*, children and the elderly) through the incorporation of adequate uncertainty factors

Emphasis was given to those limits that had adequate supporting documentation, as these values could be evaluated independently to ensure that their basis was clear, relevant and sufficient. When these criteria were satisfied by more than one objective, guideline or standard, the most scientifically defensible limit was selected.

Although not identified as COPC in the Problem Formulation, the HHRA also evaluated certain COPC within aromatic and aliphatic groups as recommended by the CCME, based on chemical structure Additional information is in Table 3–19 below. A more comprehensive description of the search process used for exposure limits, as well as the specific limits and associated rationale selected for each COPC, is provided in Appendix C Toxicity Profiles.

A complete list of the exposure limits selected for use in the HHRA is presented in Table 3–19 along with general information regarding the agency from which the limit was selected and the toxicological endpoint.

Benzo(a)pyrene and any other carcinogenic PAHs identified as COPC were evaluated in the chronic inhalation assessment using two different approaches.

In 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 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.

For 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 (TEQ) approach). The toxic equivalency of each PAH was determined using Potency Equivalency Factors (PEFs) that are presented in Health Canada (2012).

Table 3–19 Summary of Exposure Limits Used in the HHRA

Category	COPC	Acute Inhalation Exposure Limits					Chronic Inhalation Exposure Limits					Chronic Oral Exposure Limits			
		Averaging Period	Value (µg/m³)	Type	Agency	Toxicological Endpoint	Averaging Period	Value (µg/m³)	Type	Agency	Toxicological Endpoint	Value (µg/kg-day)	Type	Agency	Toxicological Endpoint
Criteria Air Contaminants	NO_2	1-hour (EPA)	188	NAAQS	US EPA	Respiratory irritation	Annual	100	RfC	US EPA	Respiratory irritation	n/a	n/a	n/a	n/a
	$PM_{2.5}$	24-hour (98th)	25	AAQO	BC MOE	Respiratory and cardiovascular morbidity and mortality	Annual	8	AAQO	BC MOE	Respiratory and cardiovascular morbidity and mortality	n/a	n/a	n/a	n/a
	SO_2	10-minute	500	RfC	WHO	Respiratory irritation	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
		1-hour (EPA)	196	NAAQS	US EPA	Respiratory irritation									
Volatile Organics	1,3-butadiene	24-hour	15	RfC	US EPA	Reproductive/ developmental effects	Annual	0.3	RSC	US EPA	Leukemia	n/a	n/a	n/a	n/a
							Annual	2	RfC	US EPA	Reproductive/ developmental effects				
	Benzene	1-hour	580	RfC	TCEQ	Immunological effects	Annual	1.3	RSC	US EPA	Leukemia	n/a	n/a	n/a	n/a
								9.8	RfC	ATSDR	Immunological and hematological effects				
	Acetaldehyde	1-hour-	470-	RfC-	OEHHA-	Eye, nasal and respiratory irritation	Annual	30	RfC	Health Canada	Nasal irritation	n/a	n/a	n/a	n/a
							Annual	17.2	RSC	Health Canada	Nasal tumours				
	Acrolein	1-hour	2.5	RfC	OEHHA	Eye, nasal and respiratory irritation	Annual	0.35	RfC	OEHHA	Nasal irritation	0.5	RfD	US EPA	Decreased survival
	Cyclohexane	-	-	-	-	-	Annual	6,000	RfC	US EPA	Reproductive/ developmental effects	n/a	n/a	n/a	n/a
	Ethylbenzene	1-hour	21,700	RfC	ATSDR	Neurological	Annual	260	RfC	ATSDR	Kidney effects	n/a	n/a	n/a	n/a
	Formaldehyde	1-hour	50	RfC	ATSDR	Eye and nasal irritation	Annual	0.8	RSC	US EPA	Nasal tumours	n/a	n/a	n/a	n/a
								11	RfC	TCEQ	Eye, nasal and respiratory irritation	150	RfD	Health Canada	Kidney and gastrointestinal effects
	Hydrogen sulphide	1-hour	98	RfC	ATSDR	Respiratory irritation	Annual	2	RfC	US EPA	Nasal irritation	n/a	n/a	n/a	n/a
	Isopropylbenzene	-	-	-	-	-	Annual	400	RfC	US EPA	Kidney effects, adrenal effects	n/a	n/a	n/a	n/a
	Naphthalene	1-hour	2,000	RfC	ACGIH	Eye irritation	Annual	3	RfC	US EPA	Nasal irritation	20	RfD	Health Canada, US EPA	Body weight and thymus lesions
	n-hexane	-	-	-	-	-	Annual	670	RfC	TCEQ	Neurological effects	n/a	n/a	n/a	n/a
	n-pentane	1-hour	200,000	RfC	TCEQ	-	Included in aliphatic C_5-C_8 group					n/a	n/a	n/a	n/a
	Toluene	1-hour	15,000	RfC	TCEQ	Eye and nasal irritation, neurological effects	Annual	5,000	RfC	US EPA	Neurological effects	n/a	n/a	n/a	n/a
	Trimethylbenzenes	1-hour	690,000	RfC	US EPA	Neurological effects	Annual	5	RfC	US EPA	Neurological effects	n/a	n/a	n/a	n/a
	Xylenes	1-hour	7,400	RfC	TCEQ	Respiratory irritation, neurological effects	Annual	610	RfC	TCEQ	Eye and nasal irritation, neurological effects	n/a	n/a	n/a	n/a

Category	COPC	Acute Inhalation Exposure Limits					Chronic Inhalation Exposure Limits					Chronic Oral Exposure Limits			
		Averaging Period	Value (µg/m³)	Type	Agency	Toxicological Endpoint	Averaging Period	Value (µg/m³)	Type	Agency	Toxicological Endpoint	Value (µg/kg-day)	Type	Agency	Toxicological Endpoint
Reduced Sulphur	Hydrogen sulphide	1-hour	98	RfC	ATSDR	Respiratory irritation	Annual	2	RfC	US EPA	Nasal irritation	n/a	n/a	n/a	n/a
PAHs	Benzo(a)pyrene (Approach 1)	Included in the aromatic C_9-C_{16} or aromatic C_{17}-C_{34} groups					Annual	0.00012	RSC	WHO	Respiratory tumours	0.0014	RSD	US EPA	Gastrointestinal tumours
	Benzo(a)pyrene (Approach 2)	Included in the aromatic C_9-C_{16} or aromatic C_{17}-C_{34} groups					Annual	0.32	RSC	Health Canada	Respiratory tumours				
	Fluoranthene	Included in the aromatic C_9-C_{16} or aromatic C_{17}-C_{34} groups					Included in Benzo(a)pyrene Approach 2 group					Included in Benzo(a)pyrene group			
												40	RfC	US EPA	Liver and kidney effects
PHC	Aliphatic C_5-C_8 group	1-hour	200,000	RfC	TCEQ	-	Annual	18,400	RfC	CCME, RIVM, TPHCWG	Neurological effects	n/a	n/a	n/a	n/a
	Aromatic C_9-C_{16} group	1-hour	2,000	RfC	ACGIH	Eye irritation	Annual	50	RfC	MA DEP	Liver and kidney effects	n/a	n/a	n/a	n/a
	Aromatic C_{17}-C_{34} group	-	-	-	-	-	-	-	-	-	-	30	RfD	CCME, MA DEP, RIVM, TPHCWG	Kidney effects

Notes:
n/a = not applicable
– = not available
US EPA: see Appendix C for additional details regarding the statistics associated with the use of the US EPA NAAQS

3.4.3 Chemical Mixtures

Given that chemical exposures rarely occur in isolation, the potential health risks associated with mixtures of the COPC were assessed in the HHRA. The default assumption with respect to how the COPC may interact was that the effects of the COPC were additive. This is consistent with guidance from Health Canada (2012). Additive interactions apply most readily to chemicals that are structurally similar, act toxicologically through similar mechanisms or affect the same target tissue in the body (*i.e.*, share commonality in effect) (Health Canada 2012).

The endpoints of the exposure limits used in the 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.

Chemicals of potential concern that were determined to be carcinogenic were evaluated on an incremental basis using carcinogenic exposure limits. In addition, carcinogenic COPC were evaluated using a different exposure limit (when available), for non-carcinogenic endpoints. For example, as both chronic carcinogenic and non-carcinogenic exposure limits are available for benzene, it was also evaluated as both a carcinogen and non-carcinogen. Details regarding the selected exposure limits and associated toxicological endpoints are provided in the toxicity profiles (Appendix C).

The chemical mixtures identified for the HHRA, based on the identified COPC for the HHRA, are presented in Table 3–20. A number of the toxicological endpoints evaluated in the mixture assessment are related to some of the health effects/diseases of interest that were identified in the Literature Review (Intrinsik 2013).

Table 3–20 Chemical Mixtures for Evaluation in the HHRA

Exposure Duration and Route	Mixtures	COPC in Mixture
Acute Inhalation	Eye irritants	Acetaldehyde, acrolein, aromatic C_9-C_{16} group, formaldehyde, toluene
	Nasal irritants	Acetaldehyde, acrolein, formaldehyde, toluene
	Respiratory irritants	Acetaldehyde, acrolein, H_2S, NO_2, SO_2, xylenes
	Neurotoxicants	Ethylbenzene, toluene, trimethylbenzenes, xylenes
Chronic Inhalation	Eye irritants	Formaldehyde, xylenes
	Nasal irritants	Acetaldehyde, acrolein, formaldehyde, H_2S, naphthalene, xylenes
	Respiratory irritants	Formaldehyde, NO_2, SO_2
	Renal toxicants (kidney)	Aromatic C_9-C_{16} group, ethylbenzene, isopropylbenzene
	Neurotoxicants	Aliphatic C_5-C_8 group, n-hexane, toluene, trimethylbenzenes, xylenes
	Leukemogens (blood cancer)	1,3-butadiene, benzene
	Nasal tumours	Acetaldehyde, formaldehyde
Chronic Multiple Pathway	Renal toxicants (kidney)	Aromatic C_{17}-C_{34} group, fluoranthene, formaldehyde

3.5 Risk Characterization

The Risk Characterization step of the HHRA is concerned with quantifying or otherwise estimating the potential health risks that could be presented to people in the area (*e.g.,* residents) who as a result of exposure to the emissions associated with the selected oil and gas activities and cumulative (regional) sources.

In order to evaluate the potential health risks associated with oil and gas activity in the region, the potential short-term (acute) and long-term (chronic) risks were characterized for the following scenarios:

- **Oil and Gas Scenario.** Emissions from oil and gas activities in the HHRA study area on their own (*i.e.*, continuous emissions from gas plants and production facilities).
- **Cumulative Scenario.** Emissions from other regional sources within the HHRA study area combined with oil and gas emissions.

Sections 3.5.1 to 3.5.3 outline how the risk estimates for the COPC were calculated and interpreted.

3.5.1 Calculation of Risk Quotients for Non-Carcinogens

Risk quotients provide a quantitative measure of the potential health risks that can result from exposure to chemicals, and are calculated by comparing the estimated exposures to the corresponding exposure limits (*i.e.,* the safe levels of exposure), as shown below:

$$RQ = \frac{\text{Exposure Estimate}}{\text{Exposure Limit}}$$

For the HHRA, separate risk quotients were calculated for each of the exposure scenarios, COPC and discrete locations examined. Note that care was taken to match the exposure limits with the appropriate exposure estimates in terms of the duration of exposure. Specifically, the acute and chronic exposure limits were matched to the corresponding estimates of short-term and long-term exposure, respectively.

Interpretation of the risk quotients varied according to the nature of the COPC being assessed, with a different convention followed for non-carcinogens *vs.* carcinogens.

For the inhalation assessment, the interpretation of the risk quotients for non-carcinogens (*i.e.,* the large majority of the COPC) proceeded as follows:

- **Risk Quotients ≤ 1.0**. Signifies that the predicted exposure is less than or equal to the exposure limit, and that no adverse health impacts would be expected. Added assurance of protection is provided by the fact that the determination of both the exposure limit and the estimated exposure embraced a high degree of conservatism.
- **Risk Quotients > 1.0.** Signifies some possibility of health risks, the significance of which must be weighed against the conservatism incorporated into the assessment. Generally, this requires that the degree of protection afforded by the exposure limit and

the degree of conservatism incorporated into the exposure estimate(s) be reviewed to determine to what extent the predicted health risks may have been exaggerated.

For comparison to a target risk quotient of 1.0, the regulatory authorities require that the risk quotient account for background exposures and exposure from multiple media (if applicable). When unable to account for these types of exposures, Health Canada recommends that a target risk quotient of 0.2 (*i.e.*, five possible exposure pathways, each accounting for 20% of exposure) be employed to ensure that the potential health risks not be understated (Health Canada 2010a).

For the multiple pathway assessment, although exposures from multiple media were accommodated, background exposures were not completely due to database limitations. Accordingly, the interpretation of the multiple pathway risks for non-carcinogenic COPC proceeded assuming the target RQ of 0.2 as follows:

- **Risk Quotients ≤ 0.2.** Signifies that the estimated exposure is less than or equal to 1/5th the exposure limit. Given the level of conservatism incorporated in the derivation of both the exposure estimate and the exposure limit, RQs less than or equal to 0.2 are associated with a low health risk and no adverse health effects would be expected.
- **Risk Quotients > 0.2.** Signifies that the estimated exposure exceeds 1/5th the exposure limit. This suggests the possibility of some potential risk, the significance of which must be balanced against the degree of conservatism incorporated into the assessment. Generally this requires that the conservative assumptions used in the exposure assessment and toxicity assessment steps be reviewed to determine to what extent the predicted health risks may have been overstated.

3.5.2 Assessment of Incremental Lifetime Cancer Risks (Carcinogens)

For the purposes of this assessment, incremental lifetime cancer risk (ILCR) estimates have been determined for the incremental contribution of the selected oil and gas activities.

Regulatory agencies such as Health Canada and the US EPA assume that any level of long-term exposure to carcinogenic chemicals is associated with some ‘hypothetical cancer risk’. On this basis, Health Canada has specified an incremental (*i.e.*, over and above an background risk level) LCR of 1.0 in 100,000, which these agencies consider acceptable, tolerable or essentially negligible (AHW 2011; Health Canada 2012). 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 (2012) requires that carcinogens be assessed on an incremental basis, and mandates an ‘acceptable’ ILCR of 1.0 in 100,000. In this HHRA, the potential risks associated with the Oil and Gas Scenario were evaluated incrementally, where defensible cancer-based exposure limits existed. As the Cumulative Scenario includes a number of regional sources, carcinogens were not explicitly evaluated for this scenario.

The ILCR values for the Oil and Gas Scenario were calculated as follows:

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

Interpretation of the ILCR values for the carcinogenic COPC) proceeded as follows:

- **ILCR ≤ 1.0.** Signifies a negligible or *de minimus* incremental lifetime cancer risk (*i.e.,* less than one extra cancer case in a population of 100,000 people).
- **ILCR > 1.0.** Signifies an incremental lifetime cancer risk exceeding 1 x 10^{-5} (*i.e.,* one extra cancer case in a population of 100,000 people). The significance of the risk should be evaluated against the conservative assumptions used in the HHRA.

3.5.3 Risks from Chemical Interactions

To evaluate the potential additive effects of COPC with common toxicological endpoints, the estimated mixture RQ or ILCR values were calculated as follows, using the respiratory irritants mixture as an example:

$$\text{RQ for the respiratory irritation mixture} = \text{RQ for Irritant No. 1} + \text{RQ for Irritant No. 2} \quad \textit{etc.}$$

Similar to the assessment of individual COPC, the mixture RQ values were compared against a benchmark of 1.0, or a carcinogenic risk level of 1 in 100,000 (*i.e.,* 1 x 10^{-5}).

3.5.4 Conservatism and Uncertainty

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

Quantitative HHRA involves assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing chemical concentrations in environmental media, chemical fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk. The US EPA (2005) suggests that the risk characterization process maintain transparency, clarity, consistency, and reasonableness. The goal of the risk characterization component of an HHRA is to clearly communicate the key findings of the assessment and to provide a clear and balanced assessment of the strengths and limitations of the process. Risk characterization involves both

scientific and policy based decision making, thereby resulting in a decision making process that blends both elements.

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

The following section describes uncertainty within the HHRA, and touches on the potential impacts of these limitations on the conclusions drawn from the assessment. Given the tendency for the assumptions described below to overestimate both exposure and toxicity, it is likely that the risk characterization errs on the side of caution and over predicts risk. A summary of the conservative assumptions that were incorporated into the HHRA can be found in Table 3–21, arranged according to the steps of the risk assessment paradigm. Examination of the table shows that conservatism was introduced at virtually every step of the assessment, and extended to both the exposure and toxicity assessment of the HHRA.

Table 3–21 Major Assumptions Applied in the HHRA and Associated Uncertainties

Risk Assessment Step	Assumption	Discussion of Conservatism/Uncertainty
Exposure Assessment	Air dispersion modelling incorporated meteorological data that represented conditions contributing to maximum predicted ground-level air concentrations of the COPC.	Use of the peak (1st highest) predicted ground-level air concentrations for the COPC on an acute basis likely contributed to the overstatement of the actual exposures that might be received by people residing in or visiting the area under most circumstances. The highest predicted annual average concentrations for each community were used to represent chronic exposures. Similar to the acute assessment, the annual concentrations used in the HHRA may overstate the actual risks posed to the area residents.
	The maximum predicted air concentration for each of the communities was used to represent the entire community.	This assumption may have resulted in the overestimation of potential health risks.
	The air quality assessment included continuous emissions from oil and gas facilities such as gas plants and productions facilities. Based on the qualitative ranking in the SLRA, several other air emission sources were excluded from the detailed HHRA.	A number of short-term or more intermittent emission sources in the region associated with oil and gas activities (*e.g.*, well drilling, fluid transportation, flaring, *etc.*) were excluded from further assessment in the SLRA.. Given the limited amount of available monitoring data for the study area particularly with regards to populated areas, it is difficult to estimate how emissions from these sources would impact the findings of the HHRA..

Risk Assessment Step	Assumption	Discussion of Conservatism/Uncertainty
Exposure Assessment (cont'd)	The people with the highest predicted exposures in each receptor group (*i.e.*, Aboriginal Residents, Agricultural Residents, Community Residents) were used to characterize the potential exposures for all people represented by the lifestyle category.	Potential exposure assumed for each lifestyle category represents a reasonable worst-case scenario. For example, it was assumed that all Aboriginal Residents consumed a diet with a high proportion of local and agricultural foods. It is possible that people in these areas consume a diet higher in store-bought foods. In addition, the chronic multiple pathway exposure risk quotients represent the highest value out of all life stage groups (typically the toddler age group).
	Indoor air exposures to the COPC were not considered in this HHRA.	It is possible that people may be exposed to the COPC as a result of indoor air sources. Indoor air was not included in the assessment of either the Oil and Gas or Cumulative Scenarios. As a result, total inhalation exposures to the COPC may be underestimated.
	Maximum annual ground-level air concentrations were used to predict various environmental media concentrations (*e.g.*, soil and garden vegetables).	The maximum annual ground-level air concentrations were used to predict concentrations of the COPC in various environmental media, such as soil, local produce, beef, eggs, dairy milk, and wild game. It is likely that the environmental concentrations (soil, water, plants) and animal tissue concentrations to which people are exposed are variable and lower over the long-term.
	Tissue concentrations from local wild game, such as moose, snowshoe hare, and ruffed grouse, were based on the maximum predicted ground-level air concentrations.	It is unlikely that wild game will forage at one fixed location over their entire lifetime. Assuming that wild game will forage at the location where the maximum concentrations are predicted in air, soil, water and vegetation over their lifetime likely overstates the exposures to people who consume wild game.
	The only local traditional food consumption information that could be identified for Aboriginal populations in BC that was publicly available was the Chan *et al.* 2011 FNFNES study.	Although the 95th percentile of food consumption data for the foods commonly consumed in the study area were selected, some of the consumption rates (such as the wild bird and game rates) are lower than expected. No other data sources were available for the region for comparison purposes. Although the consumption rates are based on the best-available information, it is possible that Aboriginal exposures to wild game have been underestimated.
	It was assumed that Aboriginal and Agricultural Residents obtained 100% of their food from local sources (*e.g.*, berries and plants, wild game, fish and garden produce) and drinking water from local water bodies.	The assumption that people obtain all of their food and water over their lifetime from the area likely contributes to the overstatement of the local exposures that might be received by these people under actual circumstances.
	Residents (Aboriginal, Agricultural, Community) were assumed to be present at their respective locations 24 hours/day, 7 days/week, 52 weeks/year for a lifetime (80 years) when evaluating multi-media (non-inhalation) exposures.	It was assumed that all residents are present in the study area over an entire 80-year lifetime. This may be conservative for individuals who move or travel away from the area for extended durations.

Risk Assessment Step	Assumption	Discussion of Conservatism/Uncertainty
Exposure Assessment (cont'd)	All oral exposures to COPC were assumed to have 100% 'bioavailability'	The magnitude of direct toxicological impact of a COPC is dependent upon that fraction of the ingested quantity of the chemical that is actually absorbed into the blood stream, and thus available for toxicological effect at the target tissue or organ within the body. Complete absorption of a chemical almost never occurs. Some fraction is not absorbed, but is excreted from the body, and not available to produce the relevant health impact. For the current assessment it was assumed that 100% of all ingested/dermally exposed chemical concentrations were absorbed into the blood stream, and would therefore express a toxic potential.
Toxicity Assessment	Exposure limits have been developed by regulatory agencies with sufficient conservatism to ensure protection of the sensitive and more susceptible individuals within the general population (*e.g.*, infants and young children, the elderly, individuals with compromised health).	A considerable amount of conservatism is incorporated in the exposure limits. These benchmarks are deliberately set by regulatory agencies with the protection of sensitive individuals in mind. Typically, the benchmarks used in the current assessment were derived from the most sensitive health-related endpoints, and then adjusted to account for differences in sensitivity to chemicals among individuals. The use of uncertainty factors is directed, in part, toward the protection of sensitive individuals.
	The findings from toxicity studies with laboratory rodents can be used to gauge the types of responses and health effects that the chemicals may cause in humans and the findings from the laboratory rodent studies can be used, in part, to determine exposure limits for the chemicals.	Laboratory rodents have traditionally served as suitable surrogate species for humans. The use of uncertainty factors accounts for the possible differences in responses to chemicals that might be observed between laboratory rodents and other species, such as humans. Recent evidence suggests that rodents might be more sensitive to certain effects than humans because of higher doses reaching the critical target site in rodents (*e.g.*, nasal effects).
	Selection of the most defensible and conservative exposure limits with supporting documentation	A comprehensive search and evaluation of available exposure limits was completed as part of the HHRA. As outlined in Section 3.4 and Appendix C, the values selected generally incorporate a degree of conservatism.
	For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions.	The existence of enzymes and biological pathways that routinely repair damage to genetic material (DNA) is well documented in the scientific literature. The potential adverse health outcomes arising from damage to DNA is usually observed only when the ability of these repair enzymes to 'fix' the damage is blocked or exceeded.
	Large uncertainty factors (*i.e.,* 100-fold or greater) were commonly used in the estimation of the exposure limits for threshold type chemicals.	Uncertainty factors were applied at exposure levels reported in animal or human studies where no adverse effects were observed (*i.e.,* NOAEL). As a result, the exceedance of a health-based exposure limit does not necessarily indicate that adverse health outcomes will occur. Rather, it means that the uncertainty factor beyond the no-effect exposure is somewhat reduced.
	Possible interactions of the COPC present in emissions that might lead to enhanced toxicity were evaluated in the assessment.	Consistent with Health Canada (2012) guidance, potential health risks associated with the COPC were considered to be additive if the exposure limit for the COPC had the same toxicological endpoint. In some instances, it is possible that components of a mixture may have different mechanisms of effect, contributing some uncertainty in the predicted risk estimates for mixtures.

4.0 RESULTS

4.1 Inhalation

4.1.1 Acute Inhalation Assessment Results

Acute inhalation risk estimates, expressed as risk quotients, were based on assumed exposure periods that range from a few minutes (*e.g.*, 10-minute SO_2) to a day (*e.g.*, 24-hour $PM_{2.5}$) and maximum predicted air concentrations (unless other noted below). The predicted risk quotients for the Oil and Gas and Cumulative Scenarios are presented in Table 4-1 for the MPOI and Community locations, Table 4-2 for the Aboriginal locations (as identified in Section 3.2.3.4), and in Table 4-3 for Agricultural locations.

Table 4–1 Acute Inhalation Risk Quotients for the MPOI and Community Locations

Category	Chemical	Case	Averaging Period [1,2]	MPOI	Dawson Creek	Fort St. John
CAC	NO_2	Cumulative	1-hour	**1.558**	0.880	0.923
		Oil and Gas		**1.555**	0.106	0.776
	$PM_{2.5}$	Cumulative	24-hour 98p	**4.506**	0.708	0.815
		Oil and Gas		0.294	0.002	0.024
	SO_2	Cumulative	10-min	**3.505**	0.088	**1.207**
		Oil and Gas		**3.505**	0.057	**1.204**
	SO_2	Cumulative	1-hour 99th	**2.571**	0.120	0.931
		Oil and Gas		**2.571**	0.075	0.924
VOC	Acetaldehyde	Cumulative	1-hour	0.098	0.077	0.098
		Oil and Gas		0.020	<0.001	0.001
	Acrolein	Cumulative	1-hour	**1.705**	0.924	**1.705**
		Oil and Gas		0.313	0.002	0.008
	1,3-Butadiene	Cumulative	24-hour	0.106	0.085	0.106
		Oil and Gas		0.001	<0.001	<0.001
	Benzene	Cumulative	1-hour	0.090	0.084	0.090
		Oil and Gas		0.043	<0.001	0.002
	Ethylbenzene	Cumulative	1-hour	0.001	0.001	0.001
		Oil and Gas		<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	1-hour	**3.805**	0.825	0.669
		Oil and Gas		**3.803**	0.014	0.038
	H_2S	Cumulative	1-hour	0.269	0.002	0.022
		Oil and Gas		0.269	0.002	0.022
	Pentane	Cumulative	1-hour	0.002	0.001	0.001
		Oil and Gas		0.002	<0.001	<0.001
	Toluene	Cumulative	1-hour	0.008	0.007	0.008
		Oil and Gas		0.002	<0.001	<0.001
	Trimethylbenzene	Cumulative	1-hour	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001
	Xylene	Cumulative	1-hour	0.005	0.004	0.005
		Oil and Gas		0.002	<0.001	<0.001
PAH	Naphthalene	Cumulative	1-hour	0.003	0.003	0.003
		Oil and Gas		<0.001	<0.001	<0.001

Category	Chemical	Case	Averaging Period [1,2]	MPOI	Dawson Creek	Fort St. John
PHC	Aliphatic C_5-C_8 group	Cumulative	1-hour	0.002	0.001	0.001
		Oil and Gas		0.002	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	1-hour	0.003	0.003	0.003
		Oil and Gas		<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative		**5.619**	**1.836**	**2.483**
		Oil and Gas		**4.138**	0.016	0.047
	Nasal Irritants	Cumulative		**5.616**	**1.834**	**2.479**
		Oil and Gas		**4.138**	0.016	0.047
	Respiratory Irritants	Cumulative		**7.139**	**2.007**	**3.958**
		Oil and Gas		**5.664**	0.184	**2.010**
	Neurotoxicants	Cumulative		0.014	0.012	0.014
		Oil and Gas		0.004	<0.001	<0.001

Notes:
1 Maximum value unless otherwise noted
2 For the evaluation of hourly NO_2 and SO_2, different statistics have been used in the HHRA as a result of the exposure limits selected. For NO_2, the 3-year average 98th percentile of the annual distribution of daily maximum 1-hour concentrations was assessed, and for SO_2, the 3 year average of the 99th percentile of the daily maximum 1 hour average concentrations was used. Additional information is provided in Appendix C Toxicity Profiles.

Table 4–2 Acute Inhalation Risk Quotients for Aboriginal Locations

Category	Chemical	Case	Averaging Period[1,2]	Blueberry River and Doig River 204	Blueberry River 205	Buick	Doig River 206	East Moberly Lake 169	Halfway River 168	Moberly Lake	West Moberly Lake 168A	Wonowon
CAC	NO_2	Cumulative	1-hour	0.367	0.756	0.618	0.269	0.198	0.227	0.114	0.120	0.190
		Oil and Gas		0.295	0.752	0.264	0.196	0.069	0.185	0.058	0.056	0.115
	$PM_{2.5}$	Cumulative	24-hour 98p	0.014	0.018	0.014	0.016	0.022	0.014	0.011	0.013	0.008
		Oil and Gas		0.006	0.007	0.005	0.007	0.002	0.004	0.002	0.002	0.002
	SO_2	Cumulative	10-min	0.043	0.240	0.060	0.394	0.121	0.157	0.057	0.141	0.060
		Oil and Gas		0.043	0.240	0.058	0.394	0.120	0.157	0.057	0.141	0.059
	SO_2	Cumulative	1-hour	0.057	0.228	0.081	0.085	0.102	0.095	0.068	0.109	0.064
		Oil and Gas		0.057	0.228	0.078	0.085	0.101	0.095	0.068	0.109	0.064
VOC	Acetaldehyde	Cumulative	1-hour	0.003	0.005	0.002	0.002	0.001	0.001	0.001	0.001	0.001
		Oil and Gas		0.002	0.005	0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001
	Acrolein	Cumulative	1-hour	0.040	0.082	0.025	0.035	0.022	0.017	0.022	0.024	0.014
		Oil and Gas		0.037	0.079	0.021	0.022	0.002	0.005	0.002	0.002	0.008
	1,3-Butadiene	Cumulative	24-hour	0.003	0.003	0.004	0.005	0.004	0.003	0.002	0.002	0.002
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Benzene	Cumulative	1-hour	0.003	0.002	0.003	0.002	0.005	0.002	0.001	0.002	0.002
		Oil and Gas		0.002	0.002	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Ethylbenzene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	1-hour	0.017	0.036	0.014	0.012	0.010	0.012	0.010	0.009	0.016
		Oil and Gas		0.013	0.034	0.011	0.007	0.004	0.010	0.003	0.004	0.016
	H2S	Cumulative	1-hour	0.002	0.011	0.003	0.003	0.002	0.002	0.001	0.001	0.001
		Oil and Gas		0.002	0.011	0.003	0.003	0.002	0.002	0.001	0.001	0.001
	Pentane	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Toluene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Trimethylbenzene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Xylene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PAH	Naphthalene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aliphatic C_5-C_8 group	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative	n/a	0.060	0.123	0.041	0.049	0.034	0.029	0.033	0.034	0.031
		Oil and Gas	n/a	0.053	0.118	0.033	0.031	0.005	0.015	0.005	0.005	0.024
	Nasal Irritants	Cumulative	n/a	0.060	0.123	0.041	0.049	0.034	0.029	0.033	0.034	0.031
		Oil and Gas	n/a	0.053	0.118	0.033	0.031	0.005	0.015	0.005	0.005	0.024
	Respiratory Irritants	Cumulative	n/a	0.469	**1.093**	0.729	0.703	0.345	0.403	0.206	0.287	0.271
		Oil and Gas	n/a	0.394	**1.086**	0.367	0.616	0.193	0.349	0.129	0.200	0.189
	Neurotoxicants	Cumulative	n/a	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	n/a	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Notes:
1 Maximum value unless otherwise noted, n/a: not applicable
2 For the evaluation of hourly NO_2 and SO_2, different statistics have been used in the HHRA as a result of the exposure limits selected. For NO_2, the 3-year average 98th percentile of the annual distribution of daily maximum 1-hour concentrations was assessed, and for SO2, the 3 year average of the 99th percentile of the daily maximum 1 hour average concentrations was used.

Table 4–3 Acute Inhalation Risk Quotients for Agricultural Locations

Category	Chemical	Case	Averaging Period[1,2]	Arras	Charlie Lake	Chetwynd	Doe River	East Pine	Goodlow	Hudson's Hope	Kelly Lake	Lone Prairie	Pine Valley	Pine View	Pouce Coupe	Rolla	Rose Prairie	Taylor	Tomslake	Tumbler Ridge
CAC	NO_2	Cumulative	1-hour	0.287	0.401	0.759	0.215	0.435	0.648	0.176	0.075	0.079	0.265	0.410	0.483	0.288	0.498	1.228	0.495	0.047
		Oil and Gas		0.087	0.175	0.052	0.135	0.071	0.607	0.085	0.063	0.046	0.238	0.275	0.130	0.240	0.361	1.209	0.406	0.040
	$PM_{2.5}$	Cumulative	24-hour 98p	0.023	0.075	0.118	0.015	0.027	0.017	0.018	0.002	0.015	0.058	0.039	0.104	0.020	0.028	0.100	0.014	0.004
		Oil and Gas		0.002	0.005	0.002	0.002	0.002	0.014	0.002	0.001	0.001	0.001	0.010	0.001	0.002	0.014	0.023	0.002	<0.001
	SO_2	Cumulative	10-min	0.051	0.122	0.181	0.101	0.073	0.185	0.149	0.036	0.063	0.298	0.261	0.042	0.113	0.071	1.081	0.033	0.023
		Oil and Gas		0.050	0.121	0.177	0.100	0.071	0.185	0.148	0.036	0.062	0.298	0.257	0.041	0.113	0.070	1.079	0.032	0.023
	SO_2	Cumulative	1-hour	0.069	0.162	0.167	0.076	0.071	0.229	0.115	0.048	0.063	0.251	0.260	0.055	0.083	0.095	0.688	0.039	0.028
		Oil and Gas		0.068	0.161	0.166	0.075	0.071	0.228	0.115	0.048	0.062	0.251	0.260	0.052	0.082	0.093	0.684	0.038	0.028
VOC	Acetaldehyde	Cumulative	1-hour	0.004	0.010	0.016	0.001	0.001	0.001	0.001	<0.001	<0.001	<0.001	0.004	0.011	0.002	0.003	0.009	0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001
	Acrolein	Cumulative	1-hour	0.055	0.192	0.196	0.025	0.024	0.037	0.021	0.005	0.011	0.008	0.109	0.158	0.036	0.058	0.421	0.032	0.007
		Oil and Gas		0.002	0.004	0.001	0.002	0.001	0.008	0.002	<0.001	0.001	0.001	0.007	0.001	0.002	0.007	0.032	0.001	0.001
	1,3-Butadiene	Cumulative	24-hour	0.004	0.019	0.013	0.003	0.005	0.003	0.003	<0.001	0.001	0.001	0.007	0.013	0.004	0.007	0.011	0.003	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Benzene	Cumulative	1-hour	0.004	0.011	0.018	0.002	0.003	0.006	0.002	<0.001	0.001	<0.001	0.008	0.011	0.003	0.004	0.013	0.002	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	0.006	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001	0.001	0.003	<0.001	<0.001
	Ethylbenzene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	1-hour	0.032	0.076	0.110	0.025	0.010	0.030	0.009	0.015	0.005	0.004	0.035	0.091	0.058	0.023	0.222	0.031	0.003
		Oil and Gas	1-hour	0.006	0.005	0.002	0.023	0.006	0.028	0.003	0.014	0.002	0.002	0.007	0.013	0.054	0.009	0.210	0.027	0.002
	H_2S	Cumulative	1-hour	0.002	0.004	0.002	0.001	0.001	0.027	0.001	0.003	0.001	0.003	0.005	0.001	0.003	0.004	0.004	0.001	0.001
		Oil and Gas	1-hour	0.002	0.004	0.002	0.001	0.001	0.027	0.001	0.003	0.001	0.003	0.005	0.001	0.003	0.004	0.004	0.001	0.001
	Pentane	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Toluene	Cumulative	1-hour	<0.001	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Trimethylbenzene	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Xylene	Cumulative	1-hour	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PAH	Naphthalene	Cumulative	1-hour	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aliphatic C_5-C_8 group	Cumulative	1-hour	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	1-hour	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas		<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative	n/a	0.091	0.280	0.323	0.052	0.036	0.068	0.031	0.020	0.016	0.012	0.149	0.261	0.096	0.085	0.654	0.065	0.010
		Oil and Gas	n/a	0.008	0.009	0.003	0.025	0.007	0.037	0.005	0.014	0.003	0.002	0.014	0.014	0.056	0.017	0.244	0.028	0.002
	Nasal Irritants	Cumulative	n/a	0.091	0.279	0.323	0.052	0.036	0.068	0.031	0.020	0.016	0.012	0.149	0.260	0.095	0.085	0.653	0.065	0.010
		Oil and Gas	n/a	0.008	0.009	0.003	0.025	0.007	0.037	0.005	0.014	0.003	0.002	0.014	0.014	0.056	0.017	0.244	0.028	0.002
	Respiratory Irritants	Cumulative	n/a	0.417	0.769	**1.154**	0.344	0.534	0.941	0.348	0.130	0.154	0.576	0.789	0.708	0.441	0.658	**2.745**	0.568	0.082
		Oil and Gas	n/a	0.158	0.344	0.233	0.239	0.145	0.870	0.236	0.115	0.110	0.541	0.546	0.185	0.358	0.466	**2.326**	0.446	0.069
	Neurotoxicants	Cumulative	n/a	<0.001	0.001	0.002	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	0.001	<0.001	<0.001	0.002	<0.001	<0.001
		Oil and Gas	n/a	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001

Notes:
1 Maximum value unless otherwise noted, n/a: not applicable
2 For the evaluation of hourly NO_2 and SO_2, different statistics have been used in the HHRA as a result of the exposure limits selected. For NO_2, the 3-year average 98th percentile of the annual distribution of daily maximum 1-hour concentrations was assessed, and for SO2, the 3 year average of the 99th percentile of the daily maximum 1 hour average concentrations was used.

On a short-term basis, acrolein, formaldehyde, NO_2, $PM_{2.5}$ and SO_2 were associated with predicted risk quotients greater than 1.0 for one or both of the Oil and Gas and Cumulative Scenarios. To gain a better understanding of the results with respect to potential human health impacts in the area associated with the elevated short-term exposures to these COPC, consideration was given to:

- The sources of the emissions
- The spatial extent of the exceedances
- The likelihood that people may be exposed
- The degree of conservatism incorporated into the assessment

Additional interpretation for each COPC is presented below.

Acrolein

The risk quotient for the acute MPOI in the Cumulative Scenario was estimated to be 1.7. All other risk quotients, including the MPOI for the Oil and Gas Scenario, were less than 1.0.

A comparison of the predicted hourly concentrations and associated risk quotients at the MPOI locations for the Oil and Gas and Cumulative Scenarios is presented in Table 4-4. Values for Dawson Creek are presented in Table 4-4 as well, as the predicted hourly concentration in the Cumulative Scenario is just below the exposure limit. The predicted maximum hourly acrolein concentration associated with oil and gas activity in the study area is lower than the health-based exposure limit of 2.5 µg/m³, and is not predicted to occur in close proximity to the communities evaluated in the HHRA. In contrast, the predicted air concentration at the MPOI for the Cumulative Scenario is approximately 1.7-times higher than the exposure limit. This Cumulative MPOI is predicted to occur within the City of Fort St. John.

Table 4–4 Summary of Predicted Air Concentrations and Acute Risk Quotients for 1-Hour Acrolein for the Oil and Gas and Cumulative Scenario

	Predicted Concentrations (µg/m³)		Risk Quotients (unitless)	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI	0.8	4.3	0.3	1.7
Dawson Creek	0.005	2.3	0.002	0.9

All isopleths relied upon in this HHRA have been included in Attachment 1. These figures should be interpreted giving consideration to the relative infrequencies with which these concentrations are predicted to occur. No exceedances are predicted for the Oil and Gas Scenario (Isopleth 1). The isopleth for the Cumulative MPOI (Isopleth 2) indicates that the peak concentrations above the exposure limit are anticipated to occur within an area centred over Fort St. John (darker green contour in Isopleth 2). According to the isopleth, much of Fort St. John is predicted to experience hourly acrolein concentrations below the exposure limit of 2.5 µg/m³ (yellow and yellow-green contours in Isopleth 2).

Emission sources that contribute to the Cumulative air concentrations of acrolein in Fort St. John and Dawson Creek include fuel-wood combustion, various non-oil and gas industrial emissions, residential heating, and agriculture. Given that area sources were also included in the Cumulative Scenario, it is not possible to determine exactly what type of emission source is contributing the most risk at either Fort St. John or Dawson Creek.

No publicly available, ambient monitoring data for acrolein were identified for comparison with the HHRA results for any of the communities within the study area. However, some acrolein data were available from a 2013 study of VOCs within residential areas of Prince George. Although not located within the HHRA study area, Prince George is the largest city in NE BC, and measured data from this area is of interest for comparison purposes. The 2013 study completed by the BC MOE (BC MOE 2013) was in response to odour concerns expressed by Prince George residents. A total of 12 hourly acrolein samples were collected and analyzed. Of these samples, five were found to exceed the HHRA exposure limit of 2.5 µg/m³. Although associated with a limited data set, the measured concentrations in Prince George were reported to range from 0.38 µg/m³ to 3.98 µg/m³, and were considered by the BC MOE (2013) to potentially be associated with the effects reported by area residents. This predicted range of concentrations is similar to the predicted concentrations of 4.3 µg/m³ (MPOI – Fort St. John) and 2.3 µg/m³ (Dawson Creek) in the current HHRA. All other predicted hourly acrolein concentrations in the study area were less than 1.0 µg/m³, and thus comparable to the lower end of the reported range in the Prince George dataset.

As discussed in the Methods section of the HHRA, a certain degree of conservatism has been incorporated into the assessment. One example of such conservatism is the degree of uncertainty that has been incorporated into the exposure limit used in the HHRA. Overall, the database of literature regarding human exposure to acrolein is limited. Two short-term studies of human exposure to acrolein are available, and both form the basis of the acute exposure limit of 2.5 µg/m³ used in the assessment. The LOAELs identified from these studies were 140 µg/m³ (Darley *et al.* 1960) and 160 µg/m³ (Weber-Tschopp *et al.* 1977). Mild eye irritation was reported at these concentrations in both studies. At the higher exposure concentrations in these two studies (*i.e.*, above 140 and 160 µg/m³), nasal and respiratory irritation also was reported by the exposed subjects. Based on a comparison with these LOAELs for acute eye, nasal and respiratory irritation, the maximum predicted hourly concentration of 4.3 µg/m³ at the MPOI for the Cumulative Scenario is well below the concentrations at which irritation has been reported in the scientific literature. There is a margin of safety (ratio of potential effect concentration to exposure concentration) of approximately 32 between the LOAEL of 140 µg/m³ and the predicted maximum exposure concentration of 4.3 µg/m³. This suggests that the overall potential for adverse effects in association with acute acrolein exposure is low.

In the interpretation of the results, consideration should also be given to the probability of the predicted hourly acrolein concentrations actually exceeding the acute exposure limit. The results presented in Table 4-5 indicate that for 99.9% of the time, the hourly acrolein concentrations at the Cumulative MPOI, located within the City of Fort St. John, will be less than the health-based exposure limit of 2.5 µg/m³. At Fort St. John, it is estimated that hourly acrolein concentrations would exceed the exposure limit only 1-hour per year. The predicted hourly acrolein concentrations in Dawson Creek do not exceed the exposure limit.

Table 4–5 Frequency Distributions of Predicted 1-hour Concentrations for Acrolein

	Oil and Gas	Cumulative
MPOI (Fort St. John)		
Below 2.5 µg/m³ (Exposure Limit)	100%	99.9%
Below 140 µg/m³ (Lowest Human LOAEL)	100%	100%
Dawson Creek		
Below 2.5 µg/m³ (Exposure Limit)	100%	100%
Below 140 µg/m³ (Lowest Human LOAEL)	100%	100%

In summary, adverse health effects in the study area in relation to short-term acrolein exposure are not anticipated, based on the following:

- For the Oil and Gas Scenario, all predicted air concentrations were lower than the health-based exposure limit of 2.5 µg/m³.
- For the majority of the locations evaluated in the Cumulative Scenario, the predicted hourly concentrations were below the health-based exposure limit of 2.5 µg/m³.
- Although the MPOI for the Cumulative Scenario is anticipated to occur in the City of Fort St. John, analysis of time series data for this location indicates that for 99.9% of the time, the hourly acrolein concentrations will be below the health-based exposure limit of 2.5 µg/m³.
- All predicted hourly acrolein air concentrations for both the Oil and Gas and Cumulative scenarios are well below the threshold above which acute irritation effects have been reported in human studies.

Additionally, it should be noted that the oil and gas sources included in the HHRA do not significantly contribute to potential adverse health risks in relation to short-term acrolein exposures.

Formaldehyde

The predicted risk quotients at the 1-hour MPOI for both the Oil and Gas and Cumulative scenarios were estimated to be greater than 1.0 (risk quotients of 3.8 for each Scenario).

A comparison of the predicted hourly air concentrations and associated risk quotients at the MPOI locations for the Oil and Gas and Cumulative Scenarios is presented in Table 4-6.

Table 4–6 Summary of Predicted Air Concentrations and Acute Risk Quotients for 1-Hour Formaldehyde at the MPOI for the Oil and Gas and Cumulative Scenario

	Predicted Concentrations (µg/m³)		Risk Quotients (unitless)	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI	190	190	3.8	3.8

The locations of the MPOIs were predicted to occur near a remote booster station in a rural forested area in the northwest corner of the study area (near Kobes). Given the isolated nature of the location, it is unlikely that members of the public would be exposed to the maximum predicted concentration.

Predicted hourly formaldehyde air concentrations in the communities included in this HHRA were all less than the acute exposure limit of 50 µg/m³, including the most populated areas of Fort St. John and Dawson Creek. These findings suggest that the majority of individuals in the region would be exposed to short-term formaldehyde concentrations that do not exceed the acute exposure limit.

The predicted hourly formaldehyde concentrations across the study area are illustrated in Isopleths 3 and 4 for the Oil and Gas and Cumulative Scenarios. As shown, both MPOIs occur at the same location and are of the same magnitude, suggesting that the concentrations at the MPOI are primarily attributable to a nearby oil and gas source (booster station), with minimal

impact from regional emission sources. The isopleths suggest that the peak concentrations (shown as the darkest green colouring in the insert) are limited spatially, and that the short-term formaldehyde concentrations drop rapidly with distance from the source. No monitoring data for formaldehyde were available for the HHRA study area or Prince George for comparison with the predicted air concentrations used in from the HHRA.

Consideration also should be given to the degree of conservatism incorporated into the acute exposure limit used in the HHRA. The 1-hour value of 50 µg/m³ was derived by the ATSDR (1999) based on a LOAEL of 500 µg/m³ determined from a 2-hour human inhalation study, where eye and nasal irritation effects were reported. The LOAEL represents the lowest dose at which adverse health effects were actually observed. In 2010, the World Health Organization (WHO) derived a guideline for formaldehyde of 100 µg/m³ that is intended to be protective against short-term sensory irritation (WHO 2010).

The MPOI concentration of 190 µg/m³ is higher than the WHO guideline, signalling the potential that sensory effects may occur. The WHO (2010) guideline was based on a NOAEL of 600 µg/m³ that was adjusted with a factor of 5 to account for variation in sensory irritation thresholds between individuals. The predicted MPOI concentration is lower than the NOAEL of 600 µg/m³ relied on by the WHO. However, the maximum predicted hourly concentration of 190 µg/m³ at the MPOI is approximately 2.6-times lower than the LOAEL of 500 µg/m³ relied upon by the ATSDR in the derivation of the acute exposure limit selected for the HHRA (see Toxicity Profiles in Appendix C). The predicted hourly formaldehyde concentrations at all other locations in the study area are less than 33 µg/m³, which is below the toxicological thresholds of interest discussed above.

The predicted concentrations at the MPOIs represent hourly maximum concentrations based on conservative assumptions made in the air quality dispersion modelling. Analysis of the hourly time series data for the MPOI location reveals that over 99.8% of the time, the concentrations at the MPOI would be below the exposure limit of 50 µg/m³ (see Table 4-7). At the MPOI, it is estimated that the hourly formaldehyde concentration would exceed the exposure limit for 19 hours per year. For 99.9% of the time, the predicted hourly concentrations at the MPOI location would be below the WHO guideline of 100 µg/m³ for the protection against sensory irritation. For 100% of the time, predicted hourly concentrations at the MPOI are below the human LOAEL for irritation effects of 500 µg/m³ identified by the ATSDR (see Appendix C).

Table 4–7 Frequency Distributions of Predicted 1-hour Formaldehyde Concentrations

MPOI	Oil and Gas	Cumulative
Below 50 µg/m³ (Exposure Limit)	99.8%	99.8%
Below 100 µg/m³ (WHO guideline)	99.9%	99.9%
Below 500 µg/m³ (LOAEL)	100%	100%

In summary, adverse health effects in the study area in relation to short-term formaldehyde exposure are not anticipated, based on the following rationale:

- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas and Cumulative scenarios are predicted to occur in a remote area, to which the general population would be unlikely to be exposed.

- No risks associated with short-term formaldehyde exposures were identified for any of the communities evaluated on an individual basis within the study area.
- The predicted hourly formaldehyde concentrations for both the Oil and Gas and Cumulative scenarios are below the threshold above which acute irritation effects have been reported in humans, and are generally below health-based exposure limits.

Based on the findings of the air quality assessment and the HHRA, the overall short-term health risks associated with formaldehyde appear to be low on a regional basis.

Nitrogen Dioxide (NO_2)

Risk quotients greater than 1.0 were identified at MPOI locations as well as for the community of Taylor, for both the Oil and Gas Scenario and Cumulative Scenario. The predicted risk quotients for all other communities evaluated in the HHRA were less than 1.0.

A comparison of the predicted hourly concentrations and associated risk quotients at the locations where the exceedances were noted for the Oil and Gas and Cumulative scenarios is presented in Table 4-8.

Table 4–8 Summary of Predicted Air Concentrations and Acute Risk Quotients for 1-Hour NO_2 at the MPOI for the Oil and Gas and Cumulative Scenario

	Predicted Concentrations (µg/m³)		Risk Quotients (unitless)	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI	293	293	1.6	1.6
Taylor	227	231	1.2	1.2

Both the Oil and Gas and Cumulative hourly MPOI concentrations are predicted to occur in a remote area in the northern portion of the study area, in close proximity to an oil and gas source (a compressor station) (see Isopleths 5 and 6). The MPOIs for both Scenarios are not predicted to occur near any populated areas, resulting in low potential for exposure of the general population to concentrations of this magnitude. In contrast, the maximum predicted concentrations at the community of Taylor (231 µg/m³, equivalent to a risk quotient of 1.2), for both the Oil and Gas and Cumulative scenarios, are predicted to occur within the municipal boundary at approximately the same location. As the location does not appear to be within a facility fenceline, it is possible that people in the area may be exposed to hourly NO_2 air concentrations that exceed the health-based exposure limit (US EPA air standard of 188 µg/m³). The similarity in the magnitude of the concentrations at both the MPOI and Taylor between the two Scenarios indicates that oil and gas sources in the vicinity of these locations contribute the most to the predicted exceedances.

Examination of the isopleth for the Oil and Gas scenario reveals that there are a few areas in addition to the MPOI location and Taylor where exceedances of the exposure limit may occur (see the green contours of Isopleth 5). The areas of exceedance appear to be isolated to locations near oil and gas emission sources. The isopleth contours indicate that the area of exceedance in association with oil and gas activities are spatially limited. However, areas spanning 5 to 25 km around the exceedances are estimated to fall within the range of 94 to 188 µg/m³ (see green contours). Although most concentrations are below the exposure limit, this isopleth indicates that oil and gas activities in the study area may be having an impact on air quality with respect to NO_2 emissions.

Isopleth 6 shows the hourly NO_2 for the Cumulative Scenario. The MPOI for the Scenario is located in the north part of the study area, in the same location as for the Oil and Gas Scenario. Overall, there appear to be similar concentration trends between the isopleths for the Oil and Gas and Cumulative Scenarios with respect to location and concentration ranges, although the NO_2 concentrations and breadth of impacted area are larger for the Cumulative Scenario. The aerial extent of the exceedances has expanded around Taylor, the area around and just outside the community of Goodlow. As indicated on the Cumulative isopleth, larger geographic areas that include communities such as the Blueberry River First Nation, Fort St. John, Goodlow, Chetwynd and Dawson Creek may experience hourly NO_2 concentrations within the range of 94 to 188 µg/m³. As stated previously, while concentrations within this range may not be associated with immediate concern to human health, the results suggest that the regional airshed is being influenced by NO_2 emissions from various sources. It is important to note that only the hourly 98th percentile concentrations are evaluated for NO_2 in this HHRA (consistent with the EPA air standard used in the HHRA); the maximum hourly NO_2 concentrations are predicted to be as high as 535 µg/m³ at the MPOI. However, given that the BC MOE 1-hour maximum objective is under review, the HHRA used only the current US EPA air quality standard of 188 µg/m³.

An exceedance of the exposure limit does not necessarily mean that adverse health effects will occur. A summary of the relationships between short-term exposure to NO_2 and health effects reported in the published scientific literature is provided in Table 4-9. Current literature would suggests that the concentrations at the MPOI (293 µg/m³) and in Taylor (227- 231 µg/m³) are within the range where variable responses have been observed in asthmatics, but not healthy individuals. The isopleths for the Oil and Gas and Cumulative Scenarios indicate that people living in communities in NE BC (other than Taylor, where an exceedance is predicted) could be exposed to concentrations ranging from 18.8 to 188 µg/m³. People in the area for recreational activities may experience similar exposure concentrations on a short-term basis as the residents, depending on their location. Concentrations within this range have not been conclusively associated with adverse health impacts in the scientific literature. Although some studies have reported mild respiratory effects in asthmatics at NO_2 concentrations less than 375 µg/m³ (Cal EPA 2007), because of the absence of a clear dose–response relationship and statistical uncertainty, the findings of these studies are not considered to reflect the acute effects of NO_2 exposure (WHO 2000; Forastiere *et al.* 1996; Cal EPA 2007). A recent meta-analysis of NO_2 exposure and airway hyper-responsiveness in asthmatics suggests that there is no evidence that NO_2 causes clinically relevant effects in asthmatics at concentrations up to 1,100 µg/m³ (Goodman *et al.* 2009).

The likelihood that the concentrations predicted at the MPOI and Taylor would actually occur at levels of concern to human health must also be examined. An analysis of hourly time series data for the MPOI locations and Taylor are presented in Table 4-10. The benchmarks for comparison in this table represent the exposure limit (the US EPA standard) and a concentration of 490 µg/m³ (the concentration above which consistent adverse effects have been observed in asthmatics). At the MPOI for the 98th percentile, it is estimated that the hourly NO_2 concentrations would exceed the US EPA air standard for 103 to 104 hours per year (Oil and Gas Scenario and Cumulative Scenario). For Taylor, it is estimated that the hourly NO_2 concentrations would be greater than the exposure limit for 52 hours per year (both the Oil and Gas and Cumulative Scenarios).

Table 4–9 Potential Acute Health Effects Associated with NO_2

Air Concentration (µg/m³)	Description of the Potential Health Effects[1]
<190	No documented reproducible evidence (consistent and significant) of adverse health effects among healthy individuals or susceptible individuals following short-term exposure. Study results are variable and are indiscernible from background or control groups.
190 to 560	Increased airways responsiveness, detectable via meta-analysis, among asthmatics. Large variability in protocols and responses.
490	Allergen-induced decrements in lung function and increased allergen-induced airways inflammatory response among asthmatics. Most studies used non-specific airways challenges. No NO_2-induced change in lung function. No documented effects among healthy individuals.
560 to 750	Potential effects on lung function indices, including inconsistent changes forced expiratory volume in 1 second (FEV1) and forced vital capacity among patients with chronic obstructive pulmonary disease (COPD) during mild exercise.
1,900 to 3,700	Increased likelihood of inflammatory response and airway responsiveness among healthy individuals during intermittent exercise. Symptoms have not been detected by most investigators among healthy individuals. Asthmatics might experience small decrements in FEV1.
≥3,700	Changes in lung function, such as increased airway resistance, in healthy individuals.

Notes:
1 These descriptions identify the health effects that might be experienced among normal, healthy individuals following acute exposure to NO_2. Also listed are the types of symptoms that might occur among individuals with pre-existing breathing disorders, such as asthma, bronchitis or COPD. The exact nature and severity of responses that might occur among individuals with pre-existing conditions will depend on several factors, including:
- the severity of the person's condition
- the age of the individual
- the level of management of the disorder, including the availability and use of medications
- the person's level of physical activity
- external environmental factors such as temperature and humidity

The symptoms that could be experienced by these individuals could be more or less severe that those described because of these factors.

Sources: Azadniv *et al.* (1998); Beil and Ulmer (1976); Blomberg *et al.* (1997, 1999); Cal EPA (2007); Devlin *et al.* (1999); Gong *et al.* (2005); Goodman *et al.* (2009); Jorres *et al.* (1995); Morrow *et al.* (1992); von Nieding *et al.* (1979, 1980); von Nieding and Wagner (1977); Vagaggini *et al.* (1996); US EPA (2008).

Table 4–10 Frequency Distributions of Predicted Hourly NO_2 Concentrations at the MPOI and Taylor

	Oil and Gas	Cumulative
MPOI (98th percentile)		
Below 188 µg/m³ (US EPA standard)	98.8%	98.8%
Below 490 µg/m³ (Table 4-9)	100%	100%
Taylor		
Below 188 µg/m³ (US EPA standard)	99.4%	99.4%
Below 490 µg/m³ (Table 4-9)	100%	100%

A limited amount of monitoring data for NO_2 was identified for the study area for comparison with the predicted results. The newly established monitoring program for NE BC does not include NO_2 (BC MOE 2014a,b,c) so no recent data from the newly installed stations in the region were available. Examination of other database sources identified two reports from the BC MOE Mobile Air Monitoring Laboratory (MAML) for the communities of Tomslake, Groundbirch, Rolla, Farmington and Kelly Lake (BC MOE 2014a,b,c). Of these, only Tomslake, Rolla and Kelly Lake were explicitly modelled in the HHRA. The available monitoring data for the five rural communities (Table 4-11) is limited to approximately less than one year during 2010 or 2011. No more recent information is available for these or any other locations within the study area.

A comparison of existing measured and predicted NO_2 data is presented in Table 4-11. For the communities of Kelly Lake, Rolla and Tomslake, the monitoring data appears to generally be in agreement with the predicted data from the HHRA. As no monitoring data appeared to be available for NO_2 in Taylor, Fort. St. John or Dawson Creek, a comparison of the predicted data with measured data could not be completed.

In general, it may be concluded from this table that the current monitoring network for NE BC does not appear to capture NO_2 concentrations in areas where elevated concentrations (as per the Isopleths 5 and 6), including communities such as Taylor, were predicted to occur. The BC Air Quality archives indicate that between 2000 and 2002, NO_2 was monitored in Taylor, but it appears that this monitoring was discontinued in 2002. The available information for Taylor post-2000 is presented in Table 4-11.

Table 4–11 Comparison of Measured Ambient NO_2 Concentrations in NE BC with Predicted NO_2 Concentrations from the HHRA

Measured Data	Taylor (2000-2002)	Kelly Lake (2011)	Rolla (2010)	Tomslake (2010)
Maximum measured 1-hour concentrations[1]	272.7	47.2	51.9	19.6
98th percentile of measured 1-hour concentrations[1]	62.1	16.0	31.4	14.0
Predicted HHRA Data	**Taylor**	**Kelly Lake**	**Rolla**	**Tomslake**
Predicted 1-hour maximum concentrations (OG, cumulative)	363 - 368	19.4 – 20.6	124	143 - 146
Predicted 1-hour 98th concentrations (OG, cumulative)	231	12.0 - 14	45 - 54	76 - 93

Notes:
1 Data obtained from BC MOE (2014d) Air Quality Archives

Some data are also available for Prince George for comparison purposes, although it is important to note that this community is outside the HHRA study area. These data have been included as they represent the only current monitoring data for NO_2 in the NE BC region as a whole. In Prince George, the maximum and 98th percentile hourly NO_2 concentrations were reported to be 104.2 µg/m³ and 64.5 µg/m³ for 2010 and 2014, respectively.

Based on the results of this HHRA, the overall potential for adverse effects associated with NO_2 exposures in the region is considered to be low to moderate based on the following:

- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas and Cumulative Scenarios are predicted to occur in a remote area, to which the general population would not likely be exposed.
- An exceedance of the health-based exposure limit was predicted for both Scenarios within the community of Taylor, where people may be regularly exposed. However, the time series analysis suggests that for over 99.4% of the time, hourly NO_2 concentrations would be below Exposure levels of interest to human health.
- No risks associated with hourly NO_2 exposures were identified for any of the other communities in the study area.

- The predicted hourly NO_2 concentrations for the both the Oil and Gas and Cumulative Scenarios are generally below the threshold above which adverse respiratory effects have been reported in human studies.

A number of communities within the HHRA study area are predicted to have 98th percentile hourly NO_2 concentrations at levels slightly below the exposure limit of 188 µg/m³, particularly in the Cumulative Scenario. As discussed in the Problem Formulation (Section 3.2) and Appendix A, the air dispersion modelling was primarily based on long-term, continuous emission sources associated with oil and gas activities. Given the number of short-term or more intermittent emission sources in the region associated with oil and gas activities (*e.g.*, well drilling, fluid transportation, flaring, *etc.*), it is possible that the potential exposures to people in the area would be higher. Given the limited amount of available monitoring data for the study area with respect to NO_2, particularly with regards to populated areas, it is difficult to confirm this conclusively.

Particulate Matter ($PM_{2.5}$)

A risk quotient of 4.5 was predicted at the MPOI for $PM_{2.5}$ on a 24-hour basis in the Cumulative Scenario only. The predicted concentration of 113 µg/m³ (based on the 98th percentile as per the BC MOE objective) is above the BC MOE objective of 25 µg/m³.

No other locations evaluated in the HHRA presented $PM_{2.5}$ concentrations above the objective of 25 µg/m³. The 24-hour $PM_{2.5}$ concentration at the MPOI for the Oil and Gas Scenario was less than the objective (see Table 4-12). This suggests that one or more non-oil and gas sources are the primary contributors to the estimated $PM_{2.5}$ concentration at the MPOI for the Cumulative Scenario. The MPOI is predicted to occur in a rural area, within close proximity (less than 200 m) to a large surface development. The MPOI is not located in or close to residential areas. Analysis of Isopleth 7 reveals that the $PM_{2.5}$ concentrations decrease to less than 25 µg/m³ within approximately 4 km of the MPOI. Given the isolated location of the MPOI, it is unlikely that people would be present and thus exposed to the elevated $PM_{2.5}$ concentration. The closest communities to the MPOI are Lone Prairie (approximately 35 km to the northeast) and Chetwynd (40 km to the north).

Table 4–12 Summary of Predicted Air Concentrations and Acute Risk Quotients for 24-hour $PM_{2.5}$ (98th Percentile) at the MPOI for the Oil and Gas and Cumulative Scenarios

	Predicted Concentrations (µg/m³)		Risk Quotients (unitless)	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI	7.4	113	0.3	4.5

Analysis of 24-hour time series data for the MPOI reveals that, for about 85% of the time, 24-hour concentrations of $PM_{2.5}$ are anticipated to be less than the BC MOE objective of 25 µg/m³. At the MPOI, it is estimated that exceedances of this objective will occur for 76 days per year. The 24-hour $PM_{2.5}$ concentrations in association with the Oil and Gas Scenario (Isopleth 8) are predicted to be less than the BC MOE objective, the Canadian Ambient Air Quality Standard (28 µg/m³) and the US EPA National Air Quality Standard (35 µg/m³) at all locations in the study area.

Table 4–13 Frequency Distributions of Predicted 24-hour Concentrations for $PM_{2.5}$ at the MPOI

MPOI	Oil and Gas	Cumulative
Below 25 µg/m³ (BC AAQO)	100%	79%
Below 28 µg/m³ (CCME AAQS)	100%	85%
Below 35 µg/m³ (US EPA NAQS)	100%	87%

There are a limited amount of ambient monitoring data for $PM_{2.5}$ in the study area. The only available information for the larger communities in the study area (Fort. St. John, Dawson Creek) were obtained from the BC EMS database (Government of BC 2014) along with information for Hudson's Hope. For these locations, 2 to 3 years of data was identified; although in the case of Fort St. John and Hudson's Hope, the data was almost 10 years old and it was not clear for what averaging period the samples were collected. Some additional information was available from reports from the BC MOE MAML unit (BC MOE 2011a,b); although this data appears to be available for a limited number of years (2010, 2011) and a small number of rural locations (Farmington, Ground birch, Kelly Lake, Rolla, Tomslake). The closest monitoring station with $PM_{2.5}$ data to the MPOI location was Hudson's Hope, a community approximately 75 km away from the MPOI. The recently proposed monitoring program for NE BC does not appear to include plans for monitoring $PM_{2.5}$ (BC MOE 2014a,b).

A summary of the available monitoring information for $PM_{2.5}$ is presented in Table 4-14 for comparison with the MPOI for both the Oil and Gas and Cumulative Scenarios. As the limited data from the BC EMS database for Fort St. John and Hudson's Hope appears to have been collected using different monitoring methods than the BC MOE data, and is available only up to 2006, this data is not presented in Table 4-14.

Table 4–14 Comparison of Measured Ambient and Predicted 24-hour 98th Percentile $PM_{2.5}$ Concentrations

Measured Ambient Data	Kelly Lake (2011)	Rolla (2010)	Tomslake (2010)
Maximum daily average concentrations[1]	15.8	27	8.2
98th percentile of measured daily average concentrations[1]	14.2	26.7	7.8
Predicted Data in HHRA	**Kelly Lake**	**Rolla**	**Tomslake**
Predicted 24-hour 98th concentrations, Cumulative Scenario	0.05	0.05	0.35

Notes:
1 Obtained from BC MOE (2014d) Air Quality Data Archives.

The comparison of measured ambient data with the predicted data for the HHRA indicates that the predicted data are lower than the measured data for Kelly Lake, Tomslake and Rolla. No recent monitoring data was available for the areas with the largest population. However, the database for ambient $PM_{2.5}$ concentrations appears to be limited for the region, especially with respect to current data and coverage of monitoring stations.

Although Prince George is outside of the study area, available monitoring data for this NE BC airshed may provide some perspective to the HHRA with respect to 24-hour $PM_{2.5}$ concentrations. The graph in Figure 4-1 obtained from a recent report by the BC MOE (2012b) compares measured 98th percentile of daily $PM_{2.5}$ concentrations (bottom horizontal axis and

red bars on the graph) from two Prince George stations (Plaza and Gladstone) with $PM_{2.5}$ data from other locations in the Province.

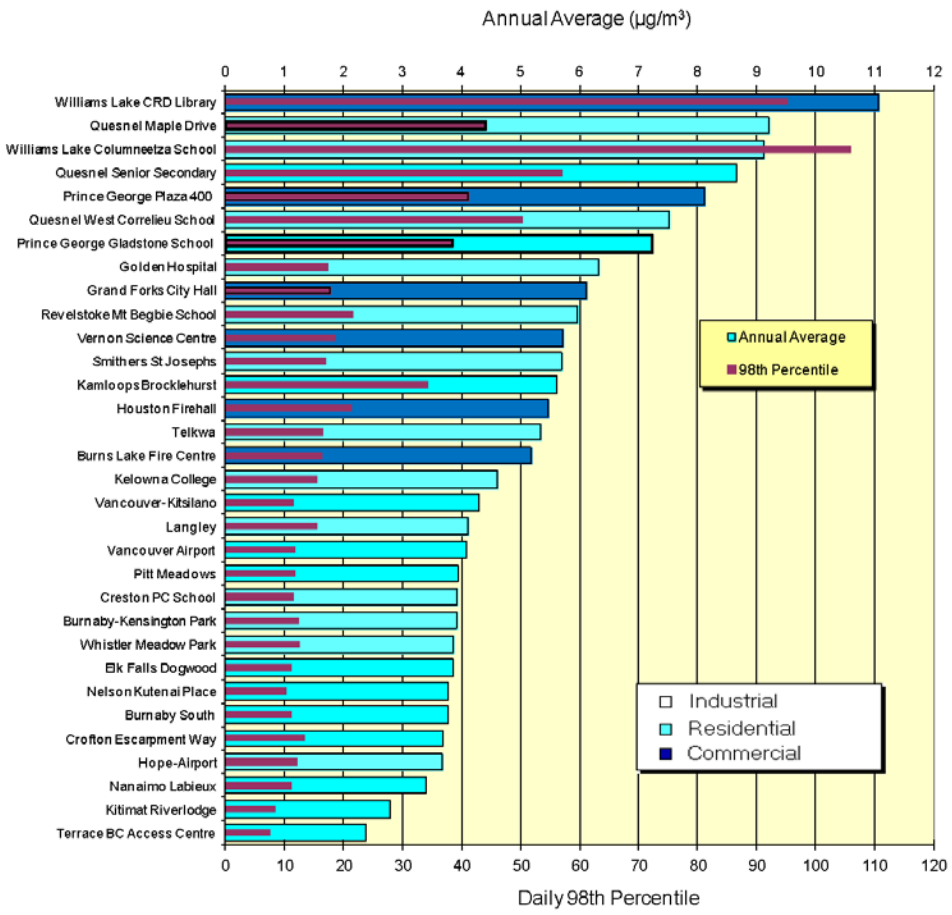


Figure 4-1 Ambient $PM_{2.5}$ Concentrations from Continuous Monitoring Stations in British Columbia (BC MOE 2012b)

Examination of Figure 4-1 reveals that the daily 98th percentile concentrations at the two Prince George stations exceed the BC MOE 24-hour objective of 25 µg/m³ and the CCME (2012b) standard of 28 µg/m³. In other areas of rural, central British Columbia, similar exceedances have been noted. While it is recognized that types and density of emission sources may vary between communities in central and northern BC, the results of the BC MOE (2012b) study suggest that $PM_{2.5}$ may be a COPC of interest to human health in the NE BC region.

The potential human health effects of $PM_{2.5}$ are dependent on both exposure concentrations and the length of exposure. Short-term studies typically only capture a small amount of the overall health effects of PM exposure. In a critical review of health effects of fine particulate air pollution, Pope and Dockery (2006) found that long-term repeated exposures have larger, more persistent cumulative effects than short-term transient exposures. It has also been suggested that daily time series studies only capture a small proportion of the potential health effects of long-term repeated exposure to PM. For this reason, the results of the chronic inhalation

assessment (*i.e.*, comparison of annual average exposure to chronic exposure limits) should also be considered when assessing the potential health risks associated with $PM_{2.5}$.

To summarize:

- For the majority of the locations evaluated, the predicted 24-hour 98th $PM_{2.5}$ concentrations were below criteria from the BC MOE and CCME.
- The MPOI is predicted to occur in an isolated, forested area, and is not in proximity to established communities. It is unlikely that the general population would be exposed to the concentrations that could occur at the MPOI location.
- Based on 24-hour time series data, it is anticipated that 24-hour $PM_{2.5}$ concentrations will be below the BC MOE air quality objective approximately 79% of the time.

The oil and gas sources included in this HHRA do not appear to significantly contribute to overall potential adverse health risks predicted in relation to short-term $PM_{2.5}$ exposures. Based on the findings of the air quality assessment and the HHRA, the overall short-term health risks associated with oil and gas related emissions of $PM_{2.5}$ appear to be low on a regional basis.

Sulphur Dioxide (SO_2)

Risk quotients greater than 1.0 were identified for SO_2 at the MPOI and for the communities of Fort St. John and Taylor in both the Oil and Gas and Cumulative Scenarios.

A summary of these exceedances and associated predicted air concentrations are provided in Table 4-15.

Table 4–15 Summary of Predicted Air Concentrations and Acute Risk Quotients for Locations with Exceedances of Exposure Limits for SO_2

	Predicted Short-term Air Concentrations (µg/m³)		Risk Quotients	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI				
10-minute	1,752	1,752	**3.5**	**3.5**
1-hour (US EPA)	504	504	**2.6**	**2.6**
Fort St. John				
10-minute	602	603	**1.2**	**1.2**
1-hour (US EPA)	181	182	0.9	0.9
Taylor				
10-minute	540	541	**1.1**	**1.1**
1-hour (US EPA)	134	135	0.7	0.7

The MPOI for the study area is predicted to occur in close proximity to a large continuous emission source northwest of Fort St. John, set back several kilometres east from Highway 97. This area is not densely populated or near communities or known recreation areas. Workers related to the facility are the most likely to frequent the area, while members of the public are not.

Isopleths for hourly SO_2 concentrations within the study area for the Oil and Gas and Cumulative Scenarios are provided in Isopleths 9 and 10. The contours around the MPOI indicate that the concentrations will decrease rapidly to levels below the exposure limit within 1 km. Although the hourly concentrations in the isopleths and Table 4-15 are below the US EPA standard of 196 µg/m³ in and surrounding the community of Taylor, the estimated 10-minute concentrations are predicted to exceed the WHO guideline of 500 µg/m³. As indicated by the light green contours in the Isopleths, areas surrounding Taylor are predicted to experience hourly SO_2 concentrations between 98 to 196 µg/m³. The results in Table 4-15 and the isopleths suggest that oil and gas activity is the primary contributor of SO_2 in the region.

Sulphur dioxide can irritate the breathing passages, and as a result, people with breathing difficulties are often at higher risk from exposure. The airways of these individuals might already be susceptible, making them particularly sensitive to the irritant action of SO_2. Asthmatics are known to be especially responsive to SO_2 and might show symptoms at lower concentrations than those affecting most people. The level of sensitivity can vary among individuals depending on the asthmatic condition, the level of physical activity and the pattern of breathing (*i.e.*, oral *vs.* nasal). While at rest, most people breathe mainly through the nose, which acts as a scrubber to remove SO_2 from the air, preventing the gas from penetrating into the deeper airways and lungs where it can cause damage. On the other hand, while exercising, breathing occurs primarily through the mouth, with very little scrubbing, allowing greater amounts of SO_2 to reach the lungs. Typically, the onset of an individual's response to SO_2 is immediate, occurring within the first few minutes of exposure and usually reaching maximum levels within five to 10 minutes. After this time, the response might either stabilize or decline despite continued exposure. Recovery from short-term exposure to SO_2 is generally complete within 1-hour from the time the exposure ends (US EPA 1994).

The highest predicted ground-level concentration of SO_2 on a 10-minute and hourly basis at the MPOI were 1,752 µg/m³ and 504 µg/m³, respectively. These concentrations are within the range of 1,300 to 2,600 µg/m³ (see Table 4-16), where it is possible that both sensitive and healthy individuals may experience adverse respiratory effects. The predicted hourly 99th percentile concentrations at the MPOI were estimated to be approximately 504 µg/m³ in both Scenarios.

The maximum 10-minute concentrations of SO_2 in Fort St. John and Taylor are approximately 602 µg/m³ and 540 µg/m³, respectively.

According to the dose-response literature for short-term SO_2 exposures, at concentrations within the 250 to 1,300 µg/m³ range sensitive individuals may experience adverse respiratory symptoms, including breathing difficulties during physical activity. Effects in normal, healthy individuals are not anticipated.

Table 4–16 Potential Adverse Health Effects Associated with Acute SO_2 Exposure

Concentration in Air (µg/m³)	Description of Potential Health Effects[1]
<250	No documented reproducible evidence of adverse health effects among healthy individuals or susceptible individuals[2] following short-term exposure.
250 to 530	Possible modest, transient changes in lung function indices, detectable by spirometry, among asthmatics during moderate to strenuous exercise. Changes characterized by increased airway resistance and/or reduced air conductance. All changes fully reversible and strictly sub-clinical in nature, with no evidence of wheezing, shortness of breath or other clinical signs. No documented effects among healthy individuals.
530 to 1,300	Increased airway resistance and potential bronchoconstriction in asthmatic or sensitive individuals engaged in moderate exercise. Bronchoconstriction with or without attendant clinical signs depending on severity of asthmatic condition. Typically no effects on lung function in healthy individuals.
1,300 to 2,600	Increased resistance in airways and difficulties breathing may be experienced by healthy individuals (in addition to asthmatics and sensitive individuals). Sore throat and the ability to taste and smell SO2 may also be apparent. Effects in asthmatics and other sensitive individuals may also include wheezing, dyspnea, and bronchoconstriction.
2,600 to 13,000	Odour is detectable. Increased resistance in airways, decreased lung volume, reduced bronchial clearance, and evidence of lung irritation (increased macrophages in lung fluid) were observed at this exposure level. Headache, coughing, throat irritation, nasal congestion, increased salivation may be evident, and some symptoms may persist for several days after exposure. Mucociliary transport in the nasal passages may also be impaired, potentially leading to nasal congestion. Respiratory effects may be more severe in asthmatics and sensitive individuals.
13,000 to 26,000	Increased resistance in airways, decreased respiratory volume, difficulties breathing, and lung irritation were reported at this exposure level. Nasal, throat, and eye irritation, nosebleeds, coughing, potentially accompanied by erythema of trachea and bronchi may occur. Respiratory effects may be more severe in asthmatics and sensitive individuals.
26,000 to 130,000	Symptoms of more severe respiratory irritation may appear, such as burning of nose and throat, sneezing, severe airway obstruction, choking, and dyspnea. Exposure may result in damage to airway epithelium that may progress to epithelial hyperplasia, an increased number of secretory goblet cells, and hypertrophy of the submucosal glands. A condition known as Reactive Airway Dysfunction Syndrome (RADS) may arise in the concentration ranges (as well as above) as a result of bronchial epithelial damage. Chronic respiratory effects may develop. Eye irritation, watery eyes, and skin eruptions (rashes) may be evident. Respiratory effects may be more severe in asthmatics and sensitive individuals.
130,000 to 260,000	Symptoms of severe respiratory irritation may occur, such as bronchitis, intolerable irritation of mucous membranes in addition to other effects described above, such as decreased lung capacity and breathing difficulties, runny nose, eye and skin irritation.
>260,000	Immediately dangerous to life and health. Chemical bronchopneumonia and asphyxia were reported at high levels of exposure. Death may result from severe respiratory depression at concentrations of approximately[2] 600,000 µg/m³.

Notes:
1 Note that the descriptions pertain largely to the types of health effects that might be experienced among normal, healthy individuals following acute exposure to SO_2. Some descriptions refer to the types of symptoms that might occur among individuals with pre-existing eye and/or breathing disorders, such as asthma, bronchitis or COPD. The exact nature and severity of responses that might occur among these latter individuals will depend on several factors, including: i) the severity of the person's condition; ii) the age of the individual; iii) the level of management of the disorder, including the availability and use of medications; iv) the person's level of physical activity; and/or, v) external environmental factors such as temperature and humidity. The symptoms that could be experienced by these individuals could be more or less severe that those described because of these factors.
2 Includes individuals suffering from respiratory disorders, such as asthma, bronchitis, and chronic obstructive pulmonary disease (COPD).
Sources: NIOSH (1974), WHO (1979), ATSDR (1998), HSDB (2010), Cal EPA (1999), WHO (2000).

Consideration must be given to how often these maximum SO_2 concentrations would actually occur, and how often the health-based thresholds of interested would be exceeded. As no

exceedances were identified for other communities in the study area, this analysis was only completed in association with the MPOI, Fort St. John and Taylor. Time series analysis was conducted on the predicted 1-hour and 10-minute concentrations, and a frequency analysis is presented in Table 4-17.

Table 4–17 Frequency Distributions of Predicted 10-minute SO_2 Concentrations in Relation to Acute SO_2 Health Benchmarks

	Oil and Gas	Cumulative
MPOI (location 14541)		
Below 500 µg/m³	99.9 %	99.9%
Below 530 µg/m³	99.9 %	99.9%
Below 1,300 µg/m³	99.9 %	99.9 %
Below 2,600 µg/m³	100 %	100%
Fort St. John		
Below 500 µg/m³	99.9%	99.9%
Below 530 µg/m³	99.9%	99.9%
Below 1,300 µg/m³	100%	100%
Below 2,600 µg/m³	100%	100%
Taylor		
Below 500 µg/m³	99.9%	99.9%
Below 530 µg/m³	99.9%	99.9%
Below 1,300 µg/m³	100%	100%
Below 2,600 µg/m³	100%	100%

Table 4–18 Frequency Distributions of Predicted Hourly SO_2 Concentrations in Relation to Acute SO_2 Health Benchmarks

	Oil and Gas	Cumulative
MPOI (location 14541)		
Below 196µg/m³	99.8%	99.9%
Below 250 µg/m³	99.8%	99.9%
Below 530 µg/m³	99.9%	99.9%
Below 1,300 µg/m³	99.9%	99.9%
Below 2,600 µg/m³	100%	100%
Fort St. John		
Below 196µg/m³	99.8%	99.9%
Below 250 µg/m³	99.9%	99.9%
Below 530 µg/m³	100%	100%
Below 1,300 µg/m³	100%	100%
Below 2,600 µg/m³	100%	100%
Taylor		
Below 196 µg/m³	99.8%	99.9%
Below 250 µg/m³	99.9%	99.9%
Below 530 µg/m³	100%	100%
Below 1,300 µg/m³	100%	100%
Below 2,600 µg/m³	100%	100%

The analysis within Table 4-17 and Table 4-18 indicate that over 99.8% of the time the 10-minute and hourly SO_2 concentrations at the MPOI, Fort St. John and Taylor are predicted to be less than the health-based guidelines (*e.g.,* 500 µg/m³ and 196 µg/m³). The likelihood that the concentrations in these locations would exceed concentration thresholds above which adverse effects might be expected in sensitive or healthy individuals is low (< 0.01% of the time). On a 10-minute basis for both the Oil and Gas and Cumulative scenarios, the predicted concentrations are estimated to exceed the exposure limit 10 hours per year at the MPOI, and 1 hour per year at Fort St. John and Taylor. The hourly SO_2 concentrations are estimated to exceed the exposure limit for 20 hours per year at the MPOI and 1 hour per year at Fort St. John and Taylor.

The predicted concentrations and exceedance frequencies for the Oil and Gas and Cumulative Scenarios were similar for the three locations evaluated in detail (MPOI, Fort St. John, Taylor). This suggests that oil and gas activities in these areas contribute the most to acute SO_2 air concentration and potential health effects. However, as discussed earlier, the locations where the maxima may occur are in areas that are unlikely to be frequented by people on a regular basis (MPOI). The majority of the communities included in this HHRA were associated with predicted short-term SO_2 concentrations below health-based guidelines.

Ambient monitoring data are available for a number of locations in the HHRA study area. A comparison of these measured concentrations with the predicted hourly concentrations in the HHRA for the communities and the MPOI is presented in Table 4-19.

Table 4–19 Comparison of Ambient and Predicted SO_2 Concentrations for HHRA Study Area

	Chetwynd Pine River (2010-2012)[1]	Taylor Townsite (2010-2012)[1]	Taylor South (2010-2012)[1]	Kelly Lake (2010-2014)	Tomslake (2010-2014)	Rolla (2010-2014))
Maximum measured 1-hour concentrations	765	317	204	15.6	6.81	6.02
99th percentile of measured 1-hour concentrations	75.4	59.5	17.3	10.5	3.14	3.67
Predicted 1-hour maximum concentrations(OG, Cumulative)	53.7 – 54.8	327 – 328	–	11.0	9.8 – 9.9	34.1 – 34.2
Predicted 1-hour 99th concentrations (OG, Cumulative)	32 - 33	134 - 135	–	9.4	7.5 – 7.6	16

Notes:
1 Data obtained from Environment Canada (2014) NAPS database
2 Data obtained from BC MOE (2014c) Air Quality Archives
– not available, OG: Oil and Gas Scenario

The results presented in the table reveals that the predicted hourly concentration at the MPOI is higher than the majority of the measured concentrations at the communities for which monitoring data was available. Although the 99th percentiles of the predicted concentrations are generally higher than the measured data, the two data sets convey a consistent message with respect to SO_2 – that concentrations within these communities are generally low. Measured

hourly SO_2 concentrations at monitoring stations near Chetwynd and Taylor are higher than the other small communities displayed in Table 4-19; however, it is noted that these areas are larger and have more industrial activity (including oil and gas). Data for Prince George are available, but was not included due to the number and locations of monitoring stations in the study area for SO_2.

To summarize:

- For the majority of the locations evaluated, for both the Oil and Gas and Cumulative scenarios, the predicted hourly concentrations were below the health-based exposure limits of 500 µg/m³ and 196 µg/m³.
- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas and Cumulative scenarios are predicted to occur in a remote area, where the general population is not likely to be exposed. As such, these exceedances are of limited regional relevance.
- Slight exceedances were predicted for 10-minute exposures for both the City of Fort St. John, and the community of Taylor. However, the time series data analysis indicates that over 99.8% of the time, the 10-minute SO_2 concentrations would be lower than the health-based guideline of 500 µg/m³.

Based on the findings of the air quality assessment and the HHRA, the overall short-term health risks associated with SO_2 appear to be low on a regional basis.

4.1.2 Acute Inhalation Mixtures Results

Acute mixtures were evaluated according to the methods outlined in Section 3.4.3. The exceedances identified in the acute inhalation mixture assessment are described below.

Eye Irritants

The predicted risk quotients for the eye irritants mixture were above 1.0 (RQ values 1.8 to 5.6) at the MPOI in both the Oil and Gas Scenario and Cumulative Scenario, and at two community locations (Dawson Creek and Fort St. John) in the Cumulative Scenario. A summary of the predicted exceedances is presented in Table 4-20.

The predicted acute risk quotients did not exceed 1.0 for any of the Aboriginal or Agricultural community locations in either scenario. This suggests that potential health risks associated with short-term exposure to the eye irritants are considered to be low and adverse health effects are not predicted to occur at these locations.

Table 4–20 Summary of Eye Irritant Mixture Risk Quotients Greater than 1.0

Scenario	MPOI	Dawson Creek	Fort St. John
Cumulative	**5.6**	**1.8**	**2.5**
Oil and Gas	**4.1**	0.02	0.05

The constituents of the acute eye irritants mixture include:

- Acetaldehyde
- Acrolein

- Aromatic C_9-C_{16} group
- Formaldehyde
- Toluene

Of these, the primary contributors are acrolein and formaldehyde, as presented in Figure 4-2 for the MPOI. These two COPC were also the primary contributors to the eye mixture risk quotients for Dawson Creek and Fort St. John. Together, acrolein and formaldehyde represent approximately 96 to 99% of the risk quotients associated with the eye irritants mixture at the MPOI, Dawson Creek and Fort St. John. Given that the predicted risk quotient is highest at the MPOI and that the primary contributors to the eye irritant risks are equivalent at the MPOI and Community locations, further discussion will focus on the exceedances predicted at the MPOI.

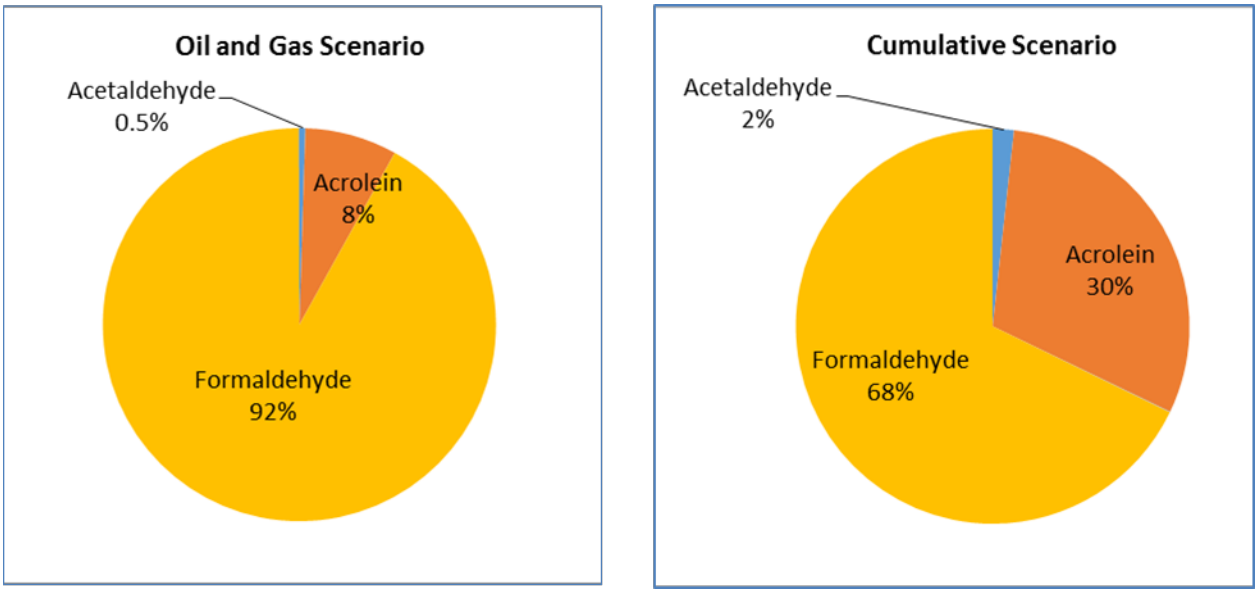


Figure 4-2 Relative COPC Contributions to the Acute Eye Irritants Mixture at the MPOI

Since acrolein and formaldehyde are the principal contributors to the eye irritant risks, the interpretation of the predicted risks focuses on these two COPC. Both of these COPC were associated with exceedances at their respective MPOIs when evaluated individually. The degree of conservatism incorporated in the acute inhalation risk quotients for acrolein and formaldehyde has previously been discussed.

As discussed in Section 4.1.1, the potential acute risks for acrolein were likely overstated in the HHRA. A high degree of conservatism was incorporated into the exposure limit used in the assessment of acrolein. The acute limit of 2.5 µg/m³ was based on a LOAEL of 140 µg/m³, at which mild eye irritation was reported in humans. This LOAEL is several times higher than the maximum predicted hourly acrolein concentrations of 4.3 µg/m³ at the MPOI (predicted to occur in Fort St. John) and 2.3 µg/m³ for Dawson Creek. As such, there is low potential for adverse health effects in association with acute acrolein exposures at these locations.

Similarly, the exposure limit used in the assessment of formaldehyde was based on a LOAEL of 500 µg/m³, a level at which eye and nasal irritation has been reported in humans. The LOAEL is approximately 2.6-times greater than the maximum predicted hourly concentration of 190 µg/m³

at the MPOI for the Oil and Gas Scenario and Cumulative Scenario. All other locations were predicted to have concentrations less than 33 µg/m³, which is lower than the exposure limit for formaldehyde itself (50 µg/m³). Thus, there is a low likelihood that exposures to formaldehyde will cause adverse health effects.

The probability that concentrations of acrolein and formaldehyde will occur at levels above health-based exposure limits must also be considered. As discussed in Section 4.1.1, although exceedances were predicted for acrolein at the Cumulative MPOI (Fort St. John), it is expected that predicted hourly acrolein concentrations will be below the health-based exposure limit of 2.5 µg/m³ approximately 99.9% of the time. Similarly, predicted hourly formaldehyde concentrations at the MPOI (Oil and Gas and Cumulative Scenarios) are expected to be below its health based exposure limit 99.8% of the time. Both formaldehyde MPOIs were predicted to occur in a remote location southeast of Wonowon, near a booster station. Neither of the acrolein MPOI concentrations (Cumulative or Oil and Gas) is predicted to occur in close proximity to this location.

Therefore, due to the low likelihood that acrolein and formaldehyde exceedances will occur simultaneously and at the same location (due to the distance between the predicted MPOI locations for each COPC), the predicted eye irritants risk quotients overstate the actual risks.

In summary, adverse health effects in the study area in relation to short-term exposure to the eye irritants mixture are not anticipated based on the following rationale:

- The low likelihood that predicted maximum acrolein and formaldehyde concentrations would exceed the health-based exposure limits.
- The maximum hourly concentrations of both acrolein and formaldehyde predicted in the HHRA are lower than the level at which responses have been observed in humans.

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

Nasal Irritants

The nasal irritants RQs were predicted to be above 1.0 (RQ values 1.8 to 5.6) at the MPOI in both the Oil and Gas Scenario and Cumulative Scenario, and in the communities of Dawson Creek and Fort St. John in the Cumulative Scenario. The predicted RQ values for these locations are presented in Table 4-21.

Predicted acute RQs did not exceed 1.0 for any of the Aboriginal or Agricultural communities in either the Cumulative or Oil and Gas scenarios. This suggests that the potential health risks associated with short-term exposure to the nasal irritants are considered low and adverse health effects are not predicted to occur at these locations.

Table 4–21 Summary of Acute Nasal Irritant Risk Quotients Greater than 1.0

Scenario	MPOI	Dawson Creek	Fort St. John
Cumulative	5.6	1.8	2.5
Oil and Gas	4.1	0.02	0.05

The components of the acute nasal irritants mixture include:

- Acetaldehyde
- Acrolein
- Formaldehyde
- Toluene

Risk quotients of 4.1 and 5.6 are predicted for the Oil and Gas MPOI and Cumulative MPOI, respectively. At the Community locations, an RQ value of 1.8 is predicted for Dawson Creek and 2.5 for Fort St. John under the Cumulative Scenario. Exceedances were not predicted under the Oil and Gas Scenario at any of the Community locations. Therefore, it is not expected that Oil and Gas activities will cause risks of nasal irritation at Community locations.

The principal contributors to the nasal irritants risks are acrolein and formaldehyde. Together, acrolein and formaldehyde represent approximately 96 to 99% of the risks associated with the nasal irritants mixture at the MPOI and Community locations where risk quotients above 1.0 are predicted. Given that the predicted risk is greatest at the MPOI and that the relative contributions to the nasal irritant risks are similar at the MPOI and Community locations, further discussion will focus on health-based exposure limit exceedances at the MPOI. Figure 4-3 displays the relative contributions of the COPC constituents to the nasal irritants mixture at the Oil and Gas MPOI and the Cumulative MPOI, respectively.

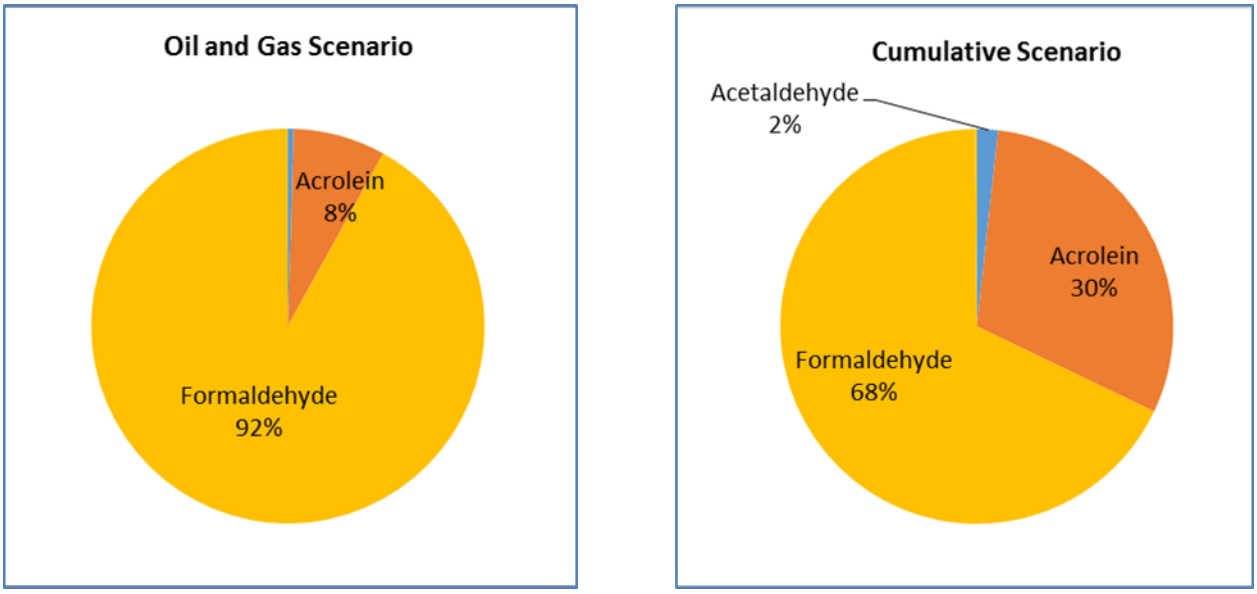


Figure 4-3 Relative COPC Contributions to the Acute Nasal Irritants Mixture at the MPOI

Since acrolein and formaldehyde are the principal contributors to the eye irritant risks, the interpretation of the predicted risks focuses on these two compounds. These two COPC are the same primary drivers of the eye irritant risks. Thus, the interpretation of the predicted risks follows the same discussion as that of the eye irritants mixture.

From these discussions, adverse health effects due to short-term exposure to the nasal irritants mixture are not anticipated based on the following rationale:

- The low likelihood that predicted maximum acrolein and formaldehyde concentrations would exceed the health-based exposure limits.
- The maximum predicted hourly concentrations of both acrolein and formaldehyde at the MPOI and the two community locations are lower than the level at which responses have been observed in humans.

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

Respiratory Irritants

The predicted respiratory irritants RQs were above 1.0 (RQ values 2.0 to 7.2) at the MPOI, Agricultural and Aboriginal locations in both the Oil and Gas Scenario and Cumulative Scenario.

Table 4–22 Summary of Acute Respiratory Irritant Risk Quotients Greater than 1.0

Scenario	MPOI	Dawson Creek	Fort St. John	Chetwynd	Taylor	Blueberry River No. 205
Cumulative	**7.2**	2.0	**4.0**	**1.1**	**2.7**	1.1
Oil and Gas	**5.7**	0.18	**2.0**	0.2	**2.3**	1.1

The constituents of the acute respiratory irritants mixture included:

- Acetaldehyde
- Acrolein
- H_2S
- NO_2
- SO_2
- Xylenes

Acrolein, NO_2 and SO_2 are the primary contributors to the respiratory irritants risks, contributing approximately 95 to 99% of the total mixture risk.

Given that the predicted risk is greatest at the MPOI and that the primary contributors to the respiratory irritant risks are similar at the MPOI and the communities listed in Table 4-22, further discussion will focus on the exceedances at the MPOI. Figure 4-4 displays the relative contributions of the COPC constituents to the respiratory irritants mixture at the Oil and Gas MPOI and the Cumulative MPOI, respectively.

As acrolein, NO_2 and SO_2 are the principal contributors to the respiratory irritant risks, the interpretation of the predicted risks focuses on these three compounds. Interpretation of the predicted risks requires an examination of the acute inhalation assessment of acrolein, NO_2 and SO_2.

The degree of conservatism incorporated in the acute inhalation risk quotients for acrolein, NO_2 and SO_2 has previously been discussed in the acute inhalation section (Section 4.1.1).

As stated previously, the predicted risks for acrolein are likely overstated and the potential for respiratory irritation to occur is thus low. This conclusion is based on the following observations:

- The overall frequency with which exceedances of the acute acrolein exposure limit may occur is generally low, occurring less than 2% of the time at the MPOI.
- There is a margin of safety incorporated into the acute acrolein exposure limit. Thus the exceedance of the limit does not necessarily indicate that people's health will be adversely affected. All predicted concentrations are well below the reported LOAEL of 140 µg/m³ in humans.

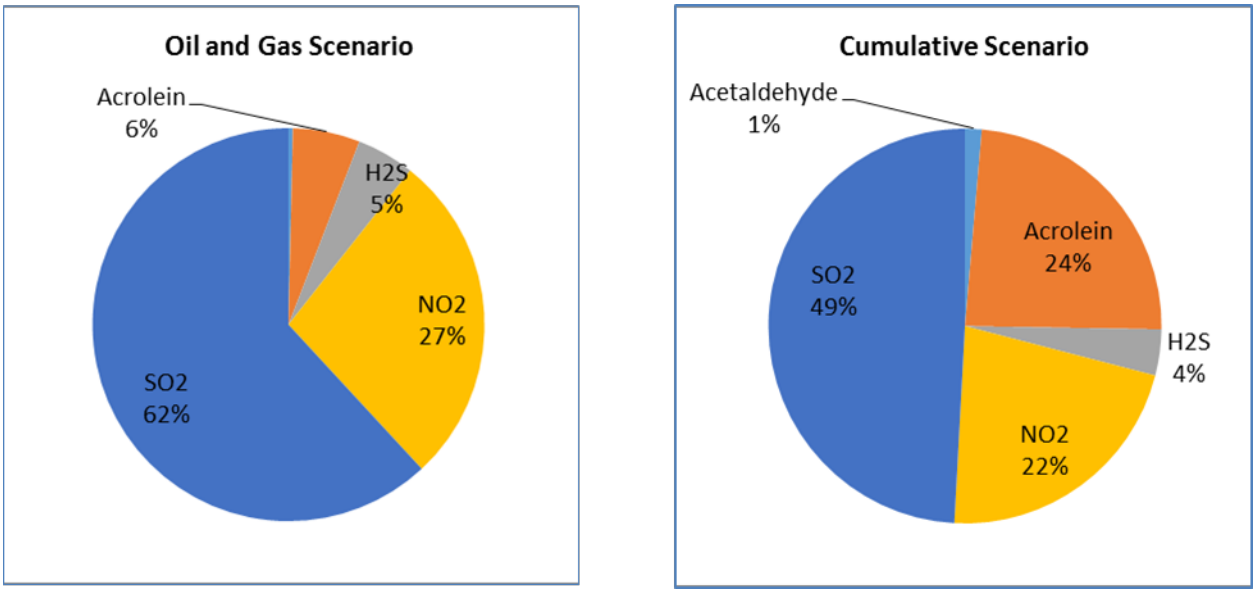


Figure 4-4 Relative COPC Contributions to the Acute Respiratory Irritants Mixture at the MPOI

For NO_2, maximum predicted hourly concentrations at MPOI locations (293 µg/m³) and the community of Taylor (227 to 231 µg/m³) exceeded the HHRA exposure limit of 188 µg/m³ for both the Oil and Gas Scenario and the Cumulative Scenario. No exceedances for acute NO_2 were determined for Chetwynd, Dawson Creek or the Blueberry First Nation, indicating that the predicted maximum hourly NO_2 concentrations for these communities were less than the NO_2 exposure limit. The predicted concentrations at the MPOI for the Cumulative Scenario are within the range where variable responses have been observed (although inconsistently) in asthmatics, though no documented effects have been observed among healthy individuals. Analysis of available time series data for the MPOI locations and Taylor revealed that over 98.8% of the time, hourly NO_2 concentrations are predicted to be below 188 µg/m³ for both the Cumulative and Oil and Gas Scenarios.

For SO_2, maximum predicted 10-minute concentrations at the MPOI (1,752 µg/m³) and the communities of Fort St. John (673 µg/m³) and Taylor (541 µg/m³) exceeded the WHO guideline of 500 µg/m³ for both the Oil and Gas Scenario and Cumulative Scenario. Maximum predicted hourly concentrations at the MPOI (1,602 µg/m³) also exceeded the US EPA standard of 196 µg/m³. MPOI concentrations are within the range where there is potential for both sensitive and healthy individuals to experience respiratory effects. In contrast, the predicted hourly

concentrations at the communities of Fort St. John and Taylor are within the range that only sensitive individuals may experience adverse respiratory effects. However, when consideration is given to how often exceedances of the SO_2 exposure limits could occur, time series analysis indicates that for over 99.8% of the time, 10-minute and hourly SO_2 concentrations are expected to be less than their health-based guidelines at the MPOI, Fort St. John and Taylor.

Consideration must also be given to where the maximum concentrations of acrolein, NO_2 and SO_2 are predicted to occur. For example, the MPOI for acrolein is predicted to occur in Fort St. John (Cumulative Scenario), while the predicted MPOI for SO_2 is estimated to occur in a remote location near a gas plant (Oil and Gas Scenario and Cumulative Scenario), and the MPOI for NO_2 is predicted to occur at a rural location in the northern portion of the study area, between the Blueberry and Doig First Nations. Thus, it is highly unlikely that a person could be exposed to the MPOI concentrations of all three of the key mixture constituents at the same time. Within the other communities where mixture exceedances were noted, the time series data suggests the probability that maximum concentrations of the mixture COPC would occur is generally low, based on the time series analysis completed for acrolein, NO_2 and SO_2.

Overall, adverse health effects due to short-term exposure to the respiratory irritants mixture are not anticipated based on: the low likelihood that predicted maximum acrolein, NO_2 and SO_2 concentrations would exceed their health-based exposure limits; and, the low likelihood that the maximum concentrations would occur at precisely the same time and at precisely the same location.

4.1.3 Chronic Inhalation Assessment Results

Chronic inhalation risk estimates, expressed as risk quotients or ILCR values, were based on chronic exposure periods that range from days to years.

It must be noted that the assumption that people could be exposed to concentrations at the MPOI over an extended time is very conservative and in all likelihood unrealistic. Many of the MPOI concentrations appear to occur within facility boundaries or in remote areas where people are unlikely to spend appreciable amounts of time over the long-term. The MPOI results have been presented to provide worst-case estimates for the study area and for comparison purposes against the chronic inhalation results for the discrete receptor locations.

Due to the differences in the methods used and in how the results are interpreted, the chronic inhalation assessment is presented and discussed in separate sections: i) the non-carcinogenic inhalation assessment; and, ii) the carcinogenic inhalation assessment.

To gain a better understanding of these results with respect to potential human health impacts in the area, consideration was given to:

- The sources of the emissions
- The spatial extent of the exceedances
- The likelihood that people may be exposed
- The degree of conservatism incorporated into the assessment

4.1.3.1 Chronic Risk Estimates for Non-Carcinogenic COPC

The predicted risk quotients for the non-carcinogenic inhalation assessment for the Oil and Gas and Cumulative Scenarios are presented in Table 4–23 for the MPOI and Community Locations, Table 4–24 for the Aboriginal locations (as identified in Section 3.2.3.4), and in Table 4-25 for the Agricultural locations.

Table 4–23 Chronic Inhalation Risk Quotients for the MPOI and Community Locations

Category	Chemical	Case	MPOI	Dawson Creek	Fort St. John
CAC	NO_2	Cumulative	0.478	0.398	0.439
		Oil and Gas	0.475	0.012	0.166
	$PM_{2.5}$	Cumulative	3.612	0.606	0.753
		Oil and Gas	0.239	0.001	0.023
VOC	Acetaldehyde	Cumulative	0.066	0.055	0.066
		Oil and Gas	0.006	<0.001	<0.001
	Acrolein	Cumulative	0.588	0.442	0.588
		Oil and Gas	0.043	<0.001	0.003
	1,3-Butadiene	Cumulative	0.150	0.120	0.150
		Oil and Gas	0.001	<0.001	<0.001
	Benzene	Cumulative	0.466	0.370	0.466
		Oil and Gas	0.090	<0.001	0.003
	Isopropylbenzene	Cumulative	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001
	Cyclohexane	Cumulative	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001
	Ethylbenzene	Cumulative	0.005	0.004	0.005
		Oil and Gas	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	0.734	0.120	0.144
		Oil and Gas	0.733	0.001	0.002
	Hexane	Cumulative	0.011	0.005	0.006
		Oil and Gas	0.011	<0.001	0.001
	H_2S	Cumulative	0.513	0.001	0.004
		Oil and Gas	0.513	0.001	0.004
	Toluene	Cumulative	0.002	0.002	0.002
		Oil and Gas	<0.001	<0.001	<0.001
	Trimethylbenzene	Cumulative	0.295	0.192	0.239
		Oil and Gas	0.284	<0.001	0.002
	Xylene	Cumulative	0.005	0.003	0.005
		Oil and Gas	0.001	<0.001	<0.001
PAH	Naphthalene	Cumulative	0.104	0.087	0.104
		Oil and Gas	0.001	<0.001	<0.001
PHC	Aliphatic C_5-C_8 group	Cumulative	0.001	0.001	0.001
		Oil and Gas	0.001	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	0.006	0.005	0.006
		Oil and Gas	<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative	0.739	0.124	0.148
		Oil and Gas	0.734	0.001	0.002
	Nasal Irritants	Cumulative	2.009	0.709	0.910
		Oil and Gas	1.297	0.002	0.009
	Respiratory Irritants	Cumulative	1.212	0.518	0.583
		Oil and Gas	1.208	0.013	0.168
	Renal toxicants	Cumulative	0.011	0.009	0.011
		Oil and Gas	<0.001	<0.001	<0.001
	Neurotoxicants	Cumulative	0.314	0.203	0.253
		Oil and Gas	0.297	<0.001	0.003

Table 4–24 Chronic Inhalation Risk Quotients for the Aboriginal Group

Category	Chemical	Case	Blueberry River and Doig River 204	Blueberry River 205	Buick	Doig River 206	East Moberly Lake 169	Halfway River 168	Moberly Lake	West Moberly Lake 168A	Wonowon
CAC	NO_2	Cumulative	0.053	0.171	0.110	0.074	0.057	0.060	0.013	0.015	0.028
		Oil and Gas	0.044	0.141	0.034	0.037	0.008	0.018	0.007	0.007	0.017
	$PM_{2.5}$	Cumulative	0.008	0.015	0.014	0.016	0.022	0.015	0.007	0.009	0.005
		Oil and Gas	0.005	0.005	0.003	0.007	0.001	0.005	0.001	0.001	0.002
VOC	Acetaldehyde	Cumulative	0.001	0.002	0.002	0.002	0.001	0.001	<0.001	<0.001	0.001
		Oil and Gas	0.001	0.001	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Acrolein	Cumulative	0.008	0.021	0.012	0.013	0.012	0.008	0.004	0.005	0.004
		Oil and Gas	0.005	0.010	0.004	0.005	<0.001	0.001	<0.001	<0.001	0.002
	1,3-Butadiene	Cumulative	0.001	0.003	0.008	0.006	0.008	0.006	0.001	0.001	0.002
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Benzene	Cumulative	0.005	0.010	0.015	0.014	0.016	0.014	0.002	0.003	0.004
		Oil and Gas	0.003	0.005	0.001	0.002	<0.001	0.001	<0.001	<0.001	0.001
	Isopropyl-benzene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Cyclohexane	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Ethylbenzene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	0.002	0.005	0.004	0.004	0.004	0.004	0.001	0.001	0.002
		Oil and Gas	0.001	0.003	0.001	0.001	<0.001	0.001	<0.001	<0.001	0.001
	Hexane	Cumulative	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	H_2S	Cumulative	0.002	0.004	0.002	0.005	0.001	0.002	0.001	0.001	0.001
		Oil and Gas	0.002	0.004	0.002	0.005	0.001	0.002	0.001	0.001	0.001
	Toluene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Trimethylbenzene	Cumulative	0.002	0.004	0.014	0.011	0.014	0.012	0.001	0.002	0.003
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Xylene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PAH	Naphthalene	Cumulative	<0.001	0.002	0.001	0.001	0.001	0.001	<0.001	0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aliphatic C_5-C_8 group	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative	0.002	0.005	0.004	0.004	0.004	0.004	0.001	0.001	0.002
		Oil and Gas	0.001	0.003	0.001	0.001	<0.001	0.001	<0.001	<0.001	0.001
	Nasal Irritants	Cumulative	0.014	0.034	0.021	0.025	0.021	0.016	0.007	0.009	0.008
		Oil and Gas	0.009	0.019	0.009	0.012	0.002	0.004	0.002	0.002	0.005
	Respiratory Irritants	Cumulative	0.055	0.176	0.114	0.079	0.061	0.063	0.014	0.017	0.030
		Oil and Gas	0.046	0.144	0.036	0.039	0.008	0.019	0.007	0.007	0.018
	Renal toxicants	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Neurotoxicants	Cumulative	0.003	0.006	0.014	0.012	0.014	0.012	0.002	0.002	0.003
		Oil and Gas	0.001	0.002	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Table 4–25 Chronic Inhalation Risk Quotients for the Agricultural Locations

Category	Chemical	Case	Arras	Charlie Lake	Chetwynd	Doe River	East Pine	Goodlow	Hudson's Hope	Kelly Lake	Lone Prairie	Pine Valley	Pine View	Pouce Coupe	Rolla	Rose Prairie	Taylor	Tomslake	Tumbler Ridge
CAC	NO_2	Cumulative	0.065	0.106	0.218	0.039	0.111	0.085	0.043	0.007	0.013	0.018	0.078	0.132	0.041	0.101	0.429	0.072	0.004
		Oil and Gas	0.012	0.026	0.008	0.020	0.010	0.045	0.009	0.006	0.007	0.008	0.047	0.012	0.015	0.059	0.422	0.024	0.003
	$PM_{2.5}$	Cumulative	0.019	0.061	0.130	0.011	0.027	0.017	0.015	0.001	0.014	0.037	0.029	0.091	0.013	0.028	0.107	0.013	0.002
		Oil and Gas	0.001	0.003	0.001	0.002	0.001	0.007	0.001	<0.001	0.001	0.001	0.009	0.001	0.001	0.015	0.018	0.001	<0.001
VOC	Acetaldehyde	Cumulative	0.001	0.005	0.011	0.001	0.001	0.001	0.001	<0.001	<0.001	<0.001	0.002	0.008	0.001	0.002	0.009	0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	0.002	<0.001	<0.001
	Acrolein	Cumulative	0.019	0.055	0.095	0.012	0.015	0.015	0.010	0.001	0.003	0.003	0.028	0.081	0.013	0.014	0.104	0.017	0.001
		Oil and Gas	<0.001	0.001	<0.001	0.001	<0.001	0.002	<0.001	<0.001	<0.001	<0.001	0.003	<0.001	<0.001	0.004	0.011	<0.001	<0.001
	1,3-Butadiene	Cumulative	0.007	0.016	0.027	0.003	0.009	0.006	0.005	<0.001	0.001	0.001	0.005	0.021	0.004	0.006	0.021	0.005	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Benzene	Cumulative	0.013	0.041	0.077	0.005	0.018	0.018	0.011	<0.001	0.002	0.002	0.017	0.056	0.007	0.015	0.072	0.009	<0.001
		Oil and Gas	<0.001	0.001	<0.001	<0.001	<0.001	0.006	<0.001	<0.001	<0.001	<0.001	0.004	<0.001	<0.001	0.003	0.019	<0.001	<0.001
	Isopropylbenzene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Cyclohexane	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Ethylbenzene	Cumulative	<0.001	0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	0.005	0.013	0.024	0.004	0.005	0.006	0.003	0.001	0.001	0.001	0.005	0.019	0.004	0.004	0.066	0.004	<0.001
		Oil and Gas	0.001	0.001	<0.001	0.002	0.001	0.002	<0.001	0.001	<0.001	<0.001	0.001	0.001	0.001	0.001	0.058	0.001	<0.001
	Hexane	Cumulative	<0.001	0.001	0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001	0.001	<0.001	0.001	0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001
	H_2S	Cumulative	0.001	0.003	0.002	0.002	0.001	0.013	0.001	0.001	0.001	0.001	0.004	0.001	0.002	0.003	0.003	0.001	<0.001
		Oil and Gas	0.001	0.003	0.002	0.002	0.001	0.013	0.001	0.001	0.001	0.001	0.004	0.001	0.002	0.003	0.003	0.001	<0.001
	Toluene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Trimethylbenzene	Cumulative	0.010	0.026	0.042	0.004	0.016	0.012	0.009	<0.001	0.001	0.001	0.008	0.030	0.005	0.011	0.295	0.006	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	0.284	<0.001	<0.001
	Xylene	Cumulative	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PAH	Naphthalene	Cumulative	0.001	0.007	0.016	0.001	0.001	0.001	0.001	<0.001	<0.001	<0.001	0.002	0.012	0.001	0.001	0.012	0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aliphatic C_5-C_8 group	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Aromatic C_9-C_{16} group	Cumulative	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixture	Eye Irritants	Cumulative	0.005	0.013	0.025	0.004	0.005	0.006	0.003	0.001	0.001	0.001	0.005	0.020	0.004	0.004	0.066	0.004	<0.001
		Oil and Gas	0.001	0.001	<0.001	0.002	0.001	0.003	<0.001	0.001	<0.001	<0.001	0.001	0.001	0.001	0.001	0.058	0.001	<0.001
	Nasal Irritants	Cumulative	0.028	0.084	0.149	0.019	0.023	0.036	0.017	0.002	0.006	0.005	0.041	0.120	0.021	0.024	0.193	0.024	0.002
		Oil and Gas	0.002	0.006	0.003	0.005	0.002	0.018	0.002	0.001	0.002	0.002	0.008	0.002	0.003	0.009	0.073	0.002	0.001
	Respiratory Irritants	Cumulative	0.069	0.119	0.243	0.043	0.115	0.090	0.047	0.008	0.014	0.019	0.084	0.151	0.045	0.106	0.495	0.077	0.005
		Oil and Gas	0.012	0.027	0.008	0.022	0.010	0.047	0.009	0.006	0.007	0.008	0.048	0.013	0.017	0.060	0.480	0.025	0.003
	Renal toxicants	Cumulative	<0.001	0.001	0.002	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Neurotoxicants	Cumulative	0.010	0.027	0.044	0.005	0.016	0.012	0.010	<0.001	0.001	0.001	0.009	0.031	0.006	0.012	0.297	0.006	<0.001
		Oil and Gas	<0.001	0.001	<0.001	0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	0.001	0.001	0.285	<0.001	<0.001

The only COPC that presented an exceedance of a non-carcinogenic exposure limit on a chronic basis was $PM_{2.5}$. Additional discussion of the $PM_{2.5}$ exceedance is provided below. All other chronic RQ values were less than 1.0. This indicates that the predicted long-term exposure COPC concentrations at the community locations included in the HHRA were below their respective exposure limits and that the associated health risks are low.

Particulate Matter ($PM_{2.5}$)

A chronic RQ value greater than 1.0 (RQ of 3.6) was predicted for the annual MPOI for $PM_{2.5}$ in the Cumulative Scenario. The RQ values for all communities within the study area were less than 1.0, as was the MPOI for the Oil and Gas Scenario.

A summary of the predicted MPOI concentrations and RQ values for the Oil and Gas and Cumulative scenarios are provided in Table 4–26 below.

Table 4–26 Summary of Predicted Air Concentrations and Risk Quotients for Annual $PM_{2.5}$ at the MPOI for the Oil and Gas and Cumulative Scenarios

	Predicted Concentrations (µg/m³)		Risk Quotients (unitless)	
	Oil and Gas	Cumulative	Oil and Gas	Cumulative
MPOI	1.9	29	0.2	3.6

According to Isopleth 11 oil and gas activity appears to have a minimal, localized impact on annual $PM_{2.5}$ concentrations in the HHRA study area. The MPOI for the Cumulative Scenario is located in an isolated and remote location near what appears to be a surface mine site. The nearest community (Pine Valley) is approximately 6 km away. Isopleth 12 indicates that the spatial extent of the exceedance of the BC MOE objective of 8 µg/m³ is localized, with concentrations decreasing rapidly to levels below the BC MOE air quality objective of 8 µg/m³ within less than 1 km. Given the location of the MPOI, it is unlikely that members of the public would be at this location for extended periods of time.

Another area with an exceedance was identified to the southeast of Pine Valley. Like the MPOI, this is also geographically removed from any of the community locations assessed in the HHRA. The exceedance of the BC MOE objective appears to be isolated and localized, and in an area not likely to be frequented by the general population.

There are a limited amount of ambient air monitoring data for $PM_{2.5}$ in the study area, limiting the opportunity for comparison with the predicted data. The only available information was obtained from the BC EMS database for Fort St. John, Dawson Creek and Hudson's Hope (Government of BC 2014). In the study area, the closest monitoring station with $PM_{2.5}$ data to the MPOI location is Hudson's Hope, a community approximately 50 km away from the MPOI. These data are available only for the period 2003 to 2006. Limited data for Fort St. John (2001 to 2003) are available for $PM_{2.5}$. Data from two stations in Dawson Creek was available for the years 2011 to 2014. The information from Dawson Creek is presented in Table 4–27 for comparison with the MPOI for each the Oil and Gas and Cumulative Scenarios, and the communities included in the HHRA for which data are available. Given that the data for Fort St. John and Hudson's Hope are 8 to 10 years out of date, they were not included for comparison purposes. These results suggest that the concentrations predicted for the communities are comparable to available measured data, and that these concentrations are several times lower than what has been predicted at the MPOI. However, the Kelly Lake, Rolla and Tomslake

locations are not located within the areas identified in the SLRA (Intrinsik 2014a) as having the highest population density. The study team is aware that BC Hydro has installed two PM monitors in the NE BC region since 2011– one along the Peace River valley between Hudson's Hope and Fort St. John, and the other along the Peace River just south of Fort St. John (BC Hydro 2012). These two monitors measure both $PM_{2.5}$ and PM_{10}, however, the data are not publicly available and thus cannot be included in this report. Other than for Dawson Creek, there is no available recent information for $PM_{2.5}$ in the most populated areas.

Table 4–27 Comparison of Measured Ambient and Predicted Annual $PM_{2.5}$ Concentrations

	Dawson Creek (2011-2014)	Kelly Lake (2011)	Rolla (2010)	Tomslake (2010)
	Concentration (µg/m³)			
Average Measured Concentrations	4.9	4.6	7.5	4.6
Predicted Annual Concentrations, Cumulative Scenario	4.9	0.011	0.11	0.11

A BC MOE (2012b) report regarding Air Quality in the Prince George Air shed is available. Although Prince George is outside of the study area, the data offers some perspective to the HHRA with respect to annual $PM_{2.5}$ concentrations. The graphs in Figure 4-5 and Figure 4-6 obtained from the BC MOE (2012b) report compares measured annual $PM_{2.5}$ concentrations from two Prince George stations (Plaza and Gladstone) with $PM_{2.5}$ data from other locations in the Province. Some data for the Tumbler Ridge Industrial Park is presented in this figure (although the study team could not access the original monitoring data). The graphs suggests that annual $PM_{2.5}$ concentrations associated with various emission sources exceeded the BC MOE objective of 8 µg/m³ in the year 2010, with locations in northern BC having concentrations of the highest magnitude. BC MOE (2012b) reports that compared to data from past years for the air shed, there appears to be an upward trend in annual $PM_{2.5}$ concentrations. However, it is noted that forest fire activity in the area in 2010 had an impact on $PM_{2.5}$ levels in the Prince George air shed. At the Tumbler Ridge Industrial Park location, the measured annual average concentration was reported to be less than 6 µg/m^3.

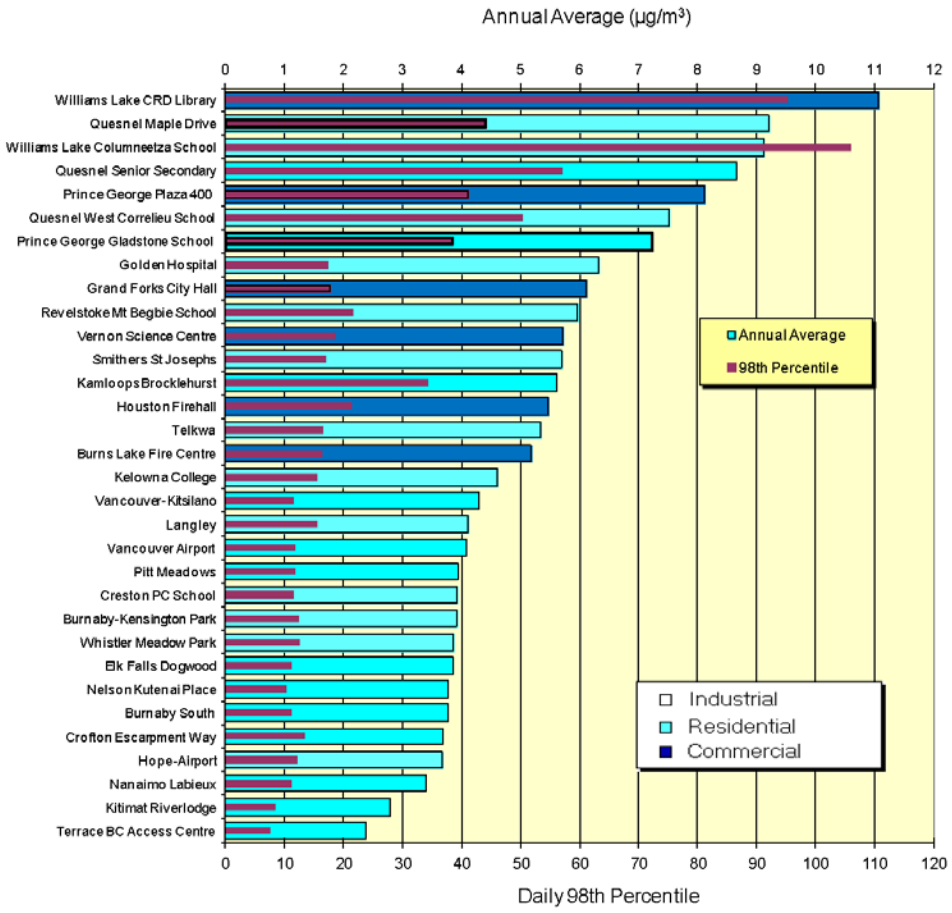


Figure 4-5 Ambient Annual and Daily 98th Percentile $PM_{2.5}$ Concentrations from Continuous Monitoring Stations in British Columbia (BC MOE 2012b)

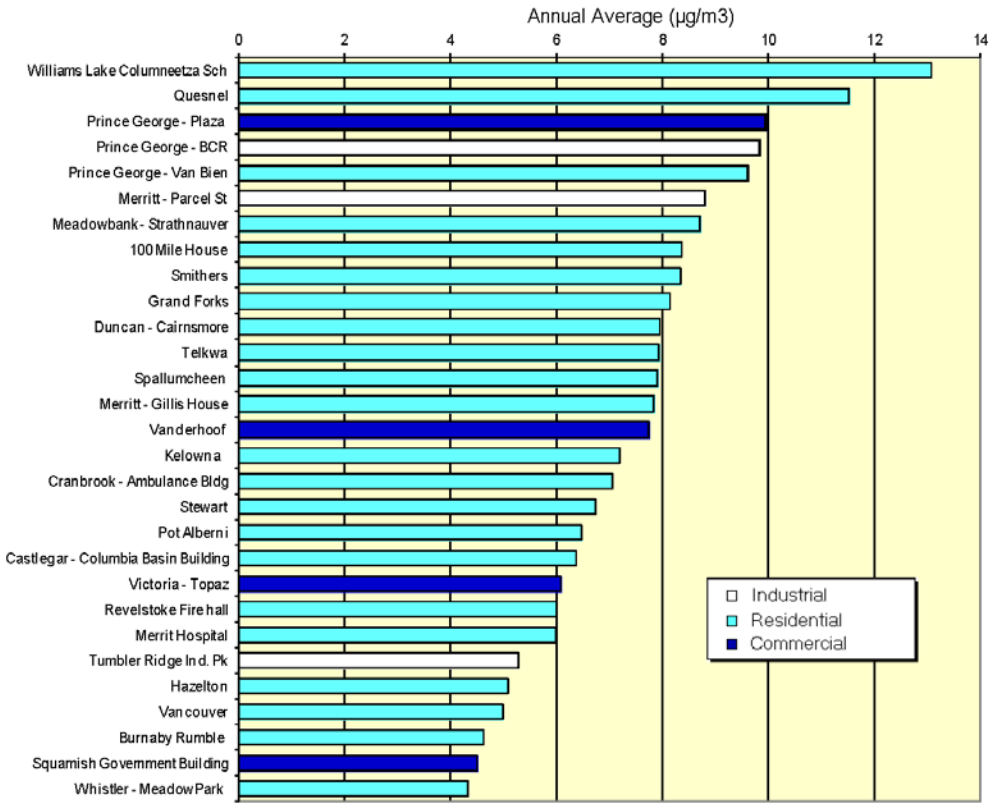


Figure 4-6 Ambient Annual $PM_{2.5}$ Concentrations from Non-Continuous Monitoring Stations in British Columbia (BC MOE 2012b)

Data presented in Figure 4-5 and Figure 4-6 seem to suggest that higher $PM_{2.5}$ concentrations, including some that are in exceedance of the BC MOE objective, have been reported not only in the NE BC City of Prince George, but also in a number of smaller, rural communities in various parts of the Province, including the area to the south and southwest of the study area. It is reasonable to assume that site-specific industrial activities and differences in population density in these areas are different than those that take place in the study area. However, given the limited degree of current monitoring data for $PM_{2.5}$ in the study area, it is difficult to compare the predictions from this HHRA with other areas.

The MPOI for the Oil and Gas scenario was associated with an RQ value that was approximately 18-times lower than Cumulative MPOI. The annual $PM_{2.5}$ concentrations predicted for both scenarios in all of the communities included in the HHRA were all less than 1.0, indicating that the predicted $PM_{2.5}$ concentrations were below the BC MOE objective of 8 µg/m³.

Based on the results of this HHRA, the overall potential health risks associated with chronic $PM_{2.5}$ exposure from oil and gas activity in the study area are anticipated to be low.

- The locations where the exceedances are predicted are isolated and in remote areas not likely to be frequented by the general population on a regular basis.

- The extent of the exceedances are anticipated to be highly localized. No exceedances of the BC MOE objective were predicted for any of the communities in the study area, for either the Oil and Gas or Cumulative scenarios.

The World Health Organization states that "research has not identified thresholds below which adverse effects do not occur" (WHO 2005). The World Health Organization chose an annual average concentration of 10 µg/m3 as its long-term air quality guideline as this represents the lower end of the range over which significant effects have been observed in epidemiological studies (Pope *et al.* 2002). The BC MOE objective is below WHO's annual guideline for $PM_{2.5}$.

Based on the "no threshold of effect" concept described by the World Health Organization (and a number of other organizations; *e.g.* US EPA), any increase in regional $PM_{2.5}$ concentrations could theoretically result in adverse health effects. The degree to which health would be affected would depend on the extent, frequency and magnitude of the predicted $PM_{2.5}$ concentrations and where these concentrations would occur. Combined with a monitoring program for $PM_{2.5}$, an emissions management plan for both the primary and secondary contributors to $PM_{2.5}$ formation would help mitigate any potential PM-related health risks in the area.

4.1.3.2 Chronic Risk Estimates for Carcinogenic COPC

Carcinogenic risks were predicted only for the Oil and Gas Scenario such that the incremental risks associated with the industry could be considered above and beyond what is contributed by ambient or regional sources. The predicted ILCR values for the COPC evaluated as carcinogens in the inhalation assessment are presented in Table 4–28, Table 4–29 and Table 4–30 for the MPOI and all communities included in the HHRA.

Table 4–28 Chronic Inhalation ILCR for the MPOI and Community Locations (risks expressed per 100,000)

Category	Chemical	Case	Incremental Lifetime Cancer Risk (per 100,000)		
			MPOI	Dawson Creek	Fort St. John
VOC	Acetaldehyde	Oil and Gas	0.011	<0.001	0.001
	1,3-butadiene	Oil and Gas	0.005	<0.001	<0.001
	Benzene	Oil and Gas	0.677	0.001	0.023
	Formaldehyde	Oil and Gas	10.1	0.011	0.021
PAH	Benzo(a)pyrene (Approach 1)	Oil and Gas	0.143	<0.001	0.005
	Benzo(a)pyrene (Approach 2)	Oil and Gas	<0.001	<0.001	<0.001
Mixtures	Nasal Tumours	Oil and Gas	10.092	0.011	0.022
	Leukemogens	Oil and Gas	0.681	0.001	0.023

Table 4–29 Chronic Inhalation ILCR for the Aboriginal Locations (risks expressed per 100,000)

Category	Chemical	Case	Incremental Lifetime Cancer Risk (per 100,000)								
			Blueberry River and Doig River 204	Blueberry River 205	Buick	Doig River 206	East Moberly Lake 169	Halfway River 168	Moberly Lake	West Moberly Lake 168A	Wonowon
VOC	Acetaldehyde	Oil and Gas	0.001	0.003	0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001
	1,3-butadiene	Oil and Gas	<0.001	0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Benzene	Oil and Gas	0.022	0.038	0.009	0.011	0.001	0.007	0.001	0.001	0.005
	Formaldehyde	Oil and Gas	0.020	0.042	0.020	0.020	0.006	0.011	0.005	0.004	0.012
PAH	Benzo(a)pyrene (Approach 1)	Oil and Gas	0.005	0.011	0.002	0.005	0.001	0.004	<0.001	<0.001	0.002
	Benzo(a)pyrene (Approach 2)	Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixtures	Nasal Tumours	Oil and Gas	0.021	0.045	0.021	0.022	0.006	0.012	0.005	0.005	0.013
	Leukemogens	Oil and Gas	0.023	0.040	0.010	0.012	0.001	0.007	0.001	0.001	0.005

Table 4–30 Chronic Inhalation ILCR for the Agricultural Locations (risks expressed per 100,000)

Category	Chemical	Case	Incremental Lifetime Cancer Risk (per 100,000)															
			Arras	Charlie Lake (Community)	Chetwynd	Doe River	East Pine	Goodlow	Hudson's Hope	Kelly Lake	Lone Prairie	Pine Valley	Pine View	Pouce Coupe	Rolla	Rose Prairie	Taylor	Tomslake	Tumbler Ridge
VOC	Acetaldehyde	Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	<0.001	<0.001	0.001	<0.001	<0.001	0.001	0.003	<0.001	<0.001
	1,3-butadiene	Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001
	Benzene	Oil and Gas	0.001	0.006	0.001	0.002	0.001	0.043	0.001	<0.001	0.001	0.001	0.031	0.001	0.001	0.021	0.145	<0.001	<0.001
	Formaldehyde	Oil and Gas	0.010	0.011	0.006	0.025	0.009	0.034	0.005	0.008	0.005	0.003	0.014	0.013	0.018	0.017	0.795	0.020	0.002
PAH	Benzo(a)pyrene (Approach 1)	Oil and Gas	0.001	0.002	0.001	0.001	0.001	0.002	0.001	<0.001	<0.001	<0.001	0.009	<0.001	0.001	0.013	0.002	<0.001	<0.001
	Benzo(a)pyrene (Approach 2)	Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixtures	Nasal Tumours	Oil and Gas	0.010	0.011	0.006	0.025	0.009	0.035	0.006	0.008	0.005	0.003	0.015	0.013	0.018	0.018	0.798	0.020	0.002
	Leukemogens	Oil and Gas	0.001	0.007	0.001	0.002	0.001	0.044	0.002	<0.001	0.001	0.001	0.031	0.001	0.001	0.021	0.147	<0.001	<0.001

The only COPC for which an ILCR greater than 1 in 100,000 was predicted was formaldehyde. This ILCR value was associated with the annual MPOI concentration for the Oil and Gas Scenario. The predicted ILCR value and the associated annual air concentration for formaldehyde are presented in Table 4-28 for the MPOI. Due to the elevated formaldehyde concentrations at the MPOI, the ILCR for the chronic nasal tumours mixture was also greater than 1 in 100,000. Additional discussion regarding the potential risks associated with formaldehyde and the nasal tumour mixture is provided in the sections below.

Formaldehyde

Consideration was given to several factors in the interpretation of this ILCR with respect to human health.

For the Oil and Gas Scenario, an ILCR of 10 in 100,000 (or 1 in 10,000) was identified at the MPOI. The MPOI is predicted to occur in what appears to be a remote area southeast of Wonowon, within close proximity of a booster station, in a relatively unpopulated area in the northwest corner of the study area. There appears to be a community named Kobes nearby, but it is not clear how many, if any, people reside here. Given the location of the MPOI, it is unlikely that people would be exposed over an extended period to the predicted maximum concentrations.

The predicted annual formaldehyde concentrations in the Oil and Gas Scenario for all community locations included in the HHRA (Agricultural, Aboriginal and the larger Communities) were all associated with ILCRs less than 1 in 100,000, indicating that the degree of excess cancer risk is essentially negligible.

No monitoring data for formaldehyde were available on a regional basis for the study area or other centres such as Prince George for comparison purposes.

Isopleth 13 indicates that the spatial extent of formaldehyde concentrations in exceedance of the US EPA RSC of 0.8 µg/m³ is limited. The peak concentrations are anticipated to be limited to a distance of approximately 1 km, and then rapidly decrease with increasing distance from the emission source. Within 2 km of the source, the annual concentrations are estimated to be less than 0.4 µg/m³. A second location that does not represent a community location in the HHRA, but appears in Isopleth 13, is also associated with an exceedance of the carcinogenic exposure limit of 0.8 µg/m³. The emission source at this second location is a gas plant that is situated within a remote location near Highway 97. Similar to the MPOI, it does not represent a location where the general population is likely to be exposed on a regular basis. The spatial extent of this exceedance is also isolated, and the annual formaldehyde concentrations rapidly diminish with distance from the source.

The US EPA exposure limit used in the assessment of formaldehyde (RSC of 0.8 µg/m³) is based on the incidence of tumours (squamous cell carcinomas and polyploidy adenomas) in the nasal cavities of rodents. Of all the chronic carcinogenic exposure limits reviewed as part of this HHRA, this value was the most conservative (*i.e.,* the lowest). Although this value is currently being reviewed by the US EPA, the RSC of 0.8 µg/m³ represents their currently recommended value until a final assessment is published. The US EPA has produced at draft re-assessment of formaldehyde and has proposed RSC values, the methodologies used by the EPA are currently

under peer-review and there is much debate over mechanisms of toxicity and target tissues (NAS 2011).

Since this RSC value was first derived by the US EPA (1991), the scientific database regarding formaldehyde carcinogenicity has evolved. The 1991 RSC was based on the assumption that formaldehyde acted via a directly genotoxic mechanism of action, and its carcinogenic effects followed a linear trend in the low-dose range.

Based on the database of scientific literature in rodents, non-human primates and humans, the mechanism of action for the formation of nasal tumours with long-term exposure involves both genotoxic and non-genotoxic mechanisms such as increased cell proliferation and cytotoxicity in tissues that first come into contact with inhaled formaldehyde (Bolt and Morfeld 2013; Swenberg *et al*. 2013). Formaldehyde is endogenously produced in humans and animals and is present in exhaled breath. Its natural presence in animal tissues presents significant uncertainty that impacts the risk assessment associated with formaldehyde exposures, in particular the assessment of additional exposures from inhalation (NAS 2011).

Formaldehyde is suspected of having a non-linear, bi-phasic dose-response relationship with a threshold effect (*e.g.*, nasal tumours) around 2,500 µg/m³. This is in contrast with the basis of the US EPA RSC that was used to characterize the long-term formaldehyde cancer risks in the current study. The US EPA RSC is based on the assumption that formaldehyde's carcinogenic potency follows a linear trend. A recent review of formaldehyde carcinogenicity by the World Health Organization (WHO 2010) concluded that long-term human exposure to average concentrations below 1,250 µg/m³ have not been associated with nasopharyngeal cancers. In contrast, the predicted annual MPOI concentration of 8.1 µg/m³ is approximately 154-times lower than this value, and approximately 310-times lower than the suspected threshold of 2,500 µg/m³.

Although the WHO (2010) derived an indoor air guideline of 100 µg formaldehyde/m³ to be protective against acute sensory irritation following 30-minute exposures, based on their literature review, the WHO concluded that the guideline of 100 µg/m³ also would be adequately protective against carcinogenic effects in humans, including the general population. The predicted annual MPOI concentration of 8.1 µg/m³ is approximately 12-times lower than this guideline.

Overall, based on the information currently available, the potential carcinogenic impacts of formaldehyde emissions from oil and gas and cumulative sources are anticipated to be minimal.

- The two locations with exceedances of the US EPA RSC are both located in fairly remote areas, near oil and gas facilities.
- These predicted maximum concentrations are anticipated to be spatially limited, with concentrations decreasing rapidly with increasing distance from the emission source.
- The predicted maximum annual formaldehyde concentrations are well below levels at which carcinogenic effects have been identified.
- No ILCR values for any of the communities included in the HHRA were predicted to be above the regulatory acceptable level of 1 in 100,000.

4.1.4 Chronic Inhalation Mixture Results

Separate assessments of non-carcinogenic and carcinogenic effects were conducted due to the differences in calculating and interpreting the risk estimates. Chronic mixture effects were evaluated using the methods outlined in Section 3.4.3. The results for the three mixtures associated with predicted risk quotients greater than 1.0 are presented in the Sections below.

4.1.4.1 Nasal Irritants Mixture

The chronic risk quotients for the nasal irritants mixture were predicted to exceed 1.0 for the MPOI in both the Oil and Gas Scenario (risk quotient of 1.3) and Cumulative Scenario (risk quotient of 2.0). The nasal irritant risks for all of the community locations (Aboriginal, Agricultural, and Community) were predicted to be less than 1.0, indicating that, on a regional basis, the potential health risks that might be associated with long –term exposure to the nasal irritants are considered to be low.

The analysis of the potential mixture risks involves an examination of the mixture components, and their relative contributions to the overall predicted risk for the mixture in the HHRA. The components of the chronic nasal irritants mixture include:

- Acetaldehyde
- Acrolein
- Formaldehyde
- H_2S
- Naphthalene
- Xylenes

The relative contribution of these COPC to the Oil and Gas and Cumulative MPOI mixture risk quotients are described graphically in Figure 4-7.

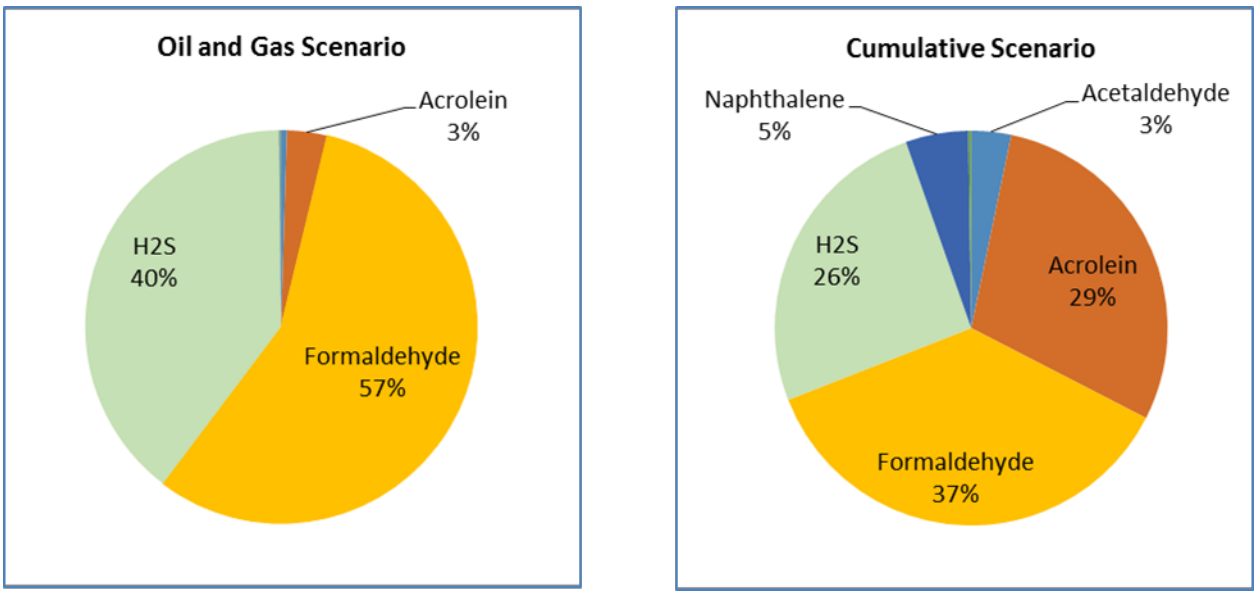


Figure 4-7 Relative COPC Contributions to the Chronic Nasal Irritants Mixture at the MPOI

In both the Oil and Gas Scenario and the Cumulative Scenario, formaldehyde (37 to 57%), hydrogen sulphide (26 to 40%), and to a lesser degree, acrolein (3 to 29%) are the COPC that contribute the most to the nasal mixture risks. None of these three COPC presented exceedances when evaluated on their own, indicating that the predicted MPOI concentrations for all three COPC are estimated to below their respective health-based exposure limits.

The probability that a person could be realistically exposed to the maximum predicted concentrations at the MPOI for each COPC, as has been assumed in this assessment, must be put into context. The chronic MPOI for formaldehyde in both scenarios is predicted to occur in a remote location in the northwest part of the study area, while the MPOI for acrolein in both scenarios are estimated to occur in Fort. St. John. The MPOI for H_2S in both the Oil and Gas and Cumulative scenarios are both predicted to occur in a remote location near a gas plant, not in proximity to either the formaldehyde or acrolein MPOIs. Thus, the assumption that the MPOIs for the COPCs in the mixture could occur simultaneously at the same location is very conservative, and in all likelihood unrealistic.

Overall, the potential adverse health impacts associated with chronic nasal irritants in the region is anticipated to be low:

- The only mixture risk quotient greater than 1.0 was predicted for the MPOI. All nasal irritant risk estimates for the individual communities were low (*i.e.*, less than 1.0).
- None of the predicted risk quotients for the individual COPC in the mixture were greater than 1.0.
- The annual MPOIs for the two COPC that contribute the most risk to the mixture are estimated to occur at different locations.

4.1.4.2 Respiratory Irritants

The predicted risk quotients for the chronic respiratory irritants mixture were predicted to be approximately 1.2 for the MPOI in both the Oil and Gas Scenario and the Cumulative Scenarios. The predicted chronic respiratory risks at all of the individual community locations were determined to be less than 1.0, suggesting that long-term exposure to the respiratory irritants at the concentrations evaluated in this HHRA are not anticipated to result in adverse health effects.

The constituents of the chronic respiratory irritants mixture include:

- Formaldehyde
- NO_2

Neither one of these COPC were associated with exceedances when evaluated on an individual basis, indicating that the predicted annual air concentrations of these COPC are expected to occur at levels below their respective health-based exposure limits.

The respective contributions of formaldehyde and NO_2 to the mixture risks are presented in Figure 4-8 for the Oil and Gas Scenario and Cumulative Scenario for the MPOI. In both scenarios, formaldehyde contributes 61% of the risk to the overall mixture, while NO_2 contributes 39%.

The formaldehyde MPOI for both scenarios are predicted to occur at a remote location in the northwest portion of the study area, and is not close to a populated areas. The NO_2 MPOI is predicted to occur in the northeastern part of the study area close to a gas plant (approximately 100 km away). Thus, it is unlikely that a person would be exposed to either MPOI, as both are in locations where the public aren't expected to spend appreciable amounts of time. Further, these two MPOI are not anticipated to occur in proximity to each other, thus preventing a person from being exposed to both simultaneously.

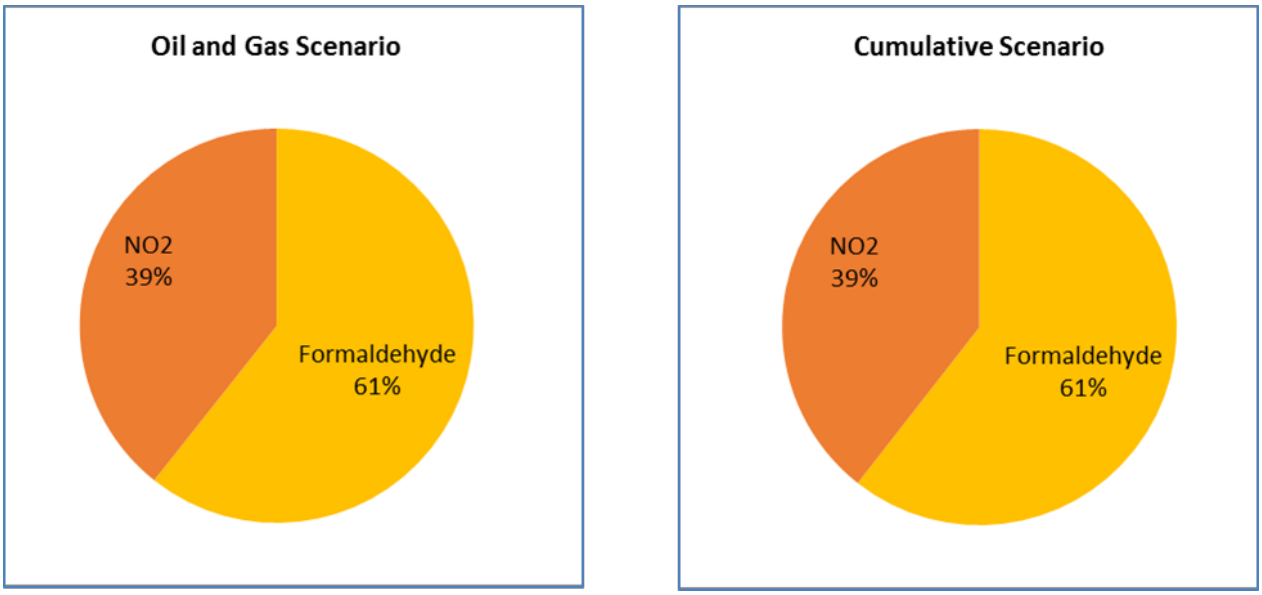


Figure 4-8 Relative COPC Contributions to the Chronic Respiratory Irritants Mixture at the MPOI

4.1.4.3 Nasal Tumours Mixture

The summed ILCR for the chronic nasal tumours mixture was predicted to be 10 in 100,000 (or 1 in 10,000) for the Oil and Gas Scenario at the MPOI (Figure 4-9). The predicted cancer risks at all of the individual community locations were determined to be less than 1.0 (in 100,000), suggesting that long-term exposure to the COPC that have the potential to cause nasal tumours are not anticipated to result in adverse health effects.

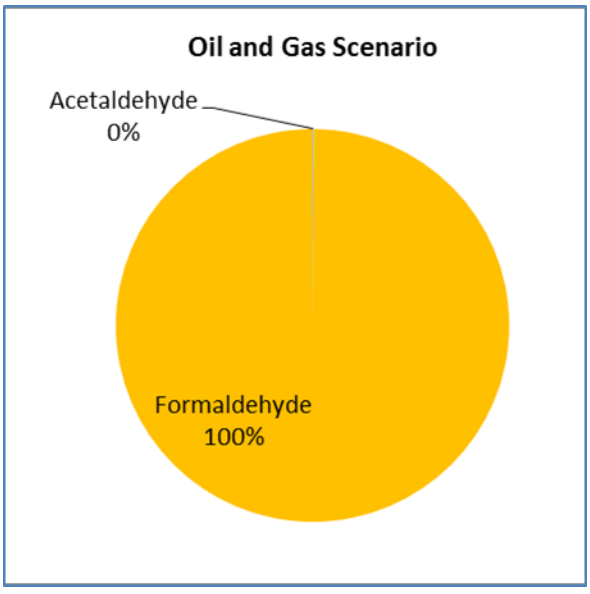


Figure 4-9 Relative COPC Contributions to the Incremental Lifetime Cancer Risks for the Nasal Tumours Mixture at the MPOI

The two COPC within this mixture include:

- Formaldehyde
- Acetaldehyde

Over 99.9% of the mixture risk is attributable to the formaldehyde MPOI. Isopleth 13 indicate that the spatial extent of formaldehyde concentrations in exceedance of the US EPA RSC of 0.8 µg/m³ is limited. The MPOI is predicted to occur in what appears to be a remote area southeast of Wonowon, within close proximity of a booster station, in a relatively unpopulated area in the northwest corner of the study area. The peak concentrations are anticipated to be limited to a distance of approximately 1 km, and then rapidly decrease with increasing distance from the emission source. Given the location of the MPOI, it is unlikely that people would be exposed over an extended period to the predicted maximum concentrations.

As discussed in Section 4.1.3.2, the predicted annual average concentration of formaldehyde at the Oil and Gas MPOI is below relevant health thresholds described by the WHO (2010).

Overall, the potential for adverse health effects in the study area as a result of exposure to chronic nasal carcinogens is minimal due to:

- The location of the MPOI in relation to the population in the region
- The degree of conservatism incorporated into the exposure limit used in the assessment

4.1.5 Other Considerations - Ozone

Oil and gas activity in the region has the potential to affect photochemical ozone formation through the release of ozone precursor emissions (*e.g.*, NO_x and VOCs). Both NO_x and VOCs are emitted from anthropogenic sources (including oil and gas activities) and natural sources. Whereas some meteorological conditions lead to the reactions that produce ozone, other conditions lead to the destruction of ozone. In fact, reactions that create ozone can occur at the same time as those that destroy ozone. Due to the inherent complexity of ozone formation and ozone destruction, photochemical ozone modelling was not undertaken for the HHRA.

In its Integrated Science Assessment (ISA) document for ozone, the US EPA (2013) concludes that, based on a weight of evidence, there is no clear health effects threshold for ozone. The US EPA acknowledges that there is some uncertainty in the lower end of the dose-response evaluations for ozone due to data limitations. No ambient air quality guideline or standard is recommended in the ISA document.

The current Canadian Ambient Air Quality Standard (CAAQS) from the Canadian Council of Ministers of the Environment (CCME 2012a) for ozone is 63 ppb (equivalent to 123 µg/m³) for comparison with the 3-year average of the annual 4th highest daily maximum 8-hour average concentrations. The Province has adopted the CAAQS as its ambient air quality objective for ozone.

Limited information is available for ozone concentrations in NE BC. The BC EMS Web Reporting database returned no results for ozone, while the NAPS database does not monitor ozone in the HHRA study area. A search of the BC Air Data Archive revealed that the Taylor Townsite monitoring location reported hourly ozone concentrations prior to mid-January 2002.

Data between 2000 and 2002 for this site indicates that hourly ozone concentrations were less than 50 ppb approximately 97% of the time (*i.e.*, exceeded 50 ppb 530 hours out of a total 16,247 hours measured). The maximum hourly concentration was 67 ppb.

Ozone levels have been reported for five different MAML sites in the HHRA study area. Results from the MAML sites were recorded in 2010 and 2011 and ozone concentrations were found to average between 17.9 and 39.6 ppb; with hourly maximums between 40.1 and 70.1 ppb. Again, ozone readings from the Taylor Townsite station, within the RSA, are only available prior to early 2002.

More detailed results of the MAML sites are as follows:

- Farmington MAML recorded ozone concentrations between August and September 2010, with a mean hourly concentration of 17.9 ppb and a maximum hourly concentration of 40.1 ppb.
- Groundbirch MAML recorded a mean hourly concentration of 22.5 ppb and a maximum hourly concentration of 40.4 ppb between June and July 2010.
- Kelly Lake MAML recorded a mean hourly concentration of 39.6 ppb and a maximum hourly concentration of 57.9 ppb between February and April 2011.
- Rolla MAML recorded a mean hourly concentration of 24.5 ppb and a maximum hourly concentration of 70.1 ppb between July and August 2010.
- Tomslake MAML recorded a mean hourly concentration of 26.7 ppb and a maximum hourly concentration of 51.3 ppb between May and June 2010.

CCME's lowest threshold value for ozone has been set at 50 ppb (*i.e.*, "green-yellow" threshold). According to the CCME (2012a), the lowest threshold value:

> *"… corresponds to "baseline" concentrations. The term "baseline" refers to air quality data that is least influenced by local and regional anthropogenic sources, as far as possible reflecting natural source emissions and very long range (intercontinental) anthropogenic emissions… The lowest Threshold Value represents the cleanest sites across Canada."*

The majority of the measured hourly air concentrations in the HHRA study area were below the CCME lowest threshold value for ozone (50 ppb).

Based on a study of ozone concentrations in British Columbia that was prepared for the BC MOE, mean background concentrations of ozone are estimated to be in the range of 20 to 35 ppb (McKendry 2006). Due to short-term ozone variability typical of airsheds, ozone concentrations are expected to occasionally exceed the BC air quality objective (and CAAQS) due to either "background sources alone, or the additive effect of local anthropogenically generated ozone and background levels" (McKendry 2006). The same study offered the recommendation that an ozone monitoring site be established in NE BC.

As ozone emissions from oil and gas activity in NE BC depend, in part, on precursor emissions (NO_x and VOCs), and regional concentrations of ozone may vary due to various conditions, the management of ozone in relation to potential human health effects associated with oil and gas activity should be focused on monitoring, in accordance with current Provincial and Federal guidance; and, on precursor emissions management (*e.g.*, eventual adherence to Canada's Base Level Industrial Emission Requirements or "BLIERs").

Similar to $PM_{2.5}$, based on the "no threshold of effect" concept described by the US EPA (2013), any increase in regional ozone concentrations could result in adverse health effects. The degree to which health would be affected would then depend on the extent, frequency and magnitude of the predicted ozone concentrations. Combined with a monitoring program for ozone, an emissions management plan for the precursor compounds would help mitigate any potential ozone-related health risks in the area.

4.2 Chronic Multiple Pathway Exposure Results

The multiple pathway assessment was completed based on the assumption that people living in the area (*i.e.*, Aboriginal Group, Agricultural Group and Community Group) could be exposed to COPC via multiple exposure pathways over their entire lifetime (80 years). As indicated previously, the multiple pathway assessment focused only on COPC that satisfied the environmental fate and persistence criteria that were used to determine the likelihood that exposure might occur through secondary pathways (including food and water consumption).

Predicted health risks are expressed as risk quotients for the non-carcinogenic COPC and as ILCR values for the carcinogenic COPC. Separate assessments were completed for non-carcinogenic and carcinogenic exposures, reflecting the different approaches used in calculating and interpreting the risk estimates.

The predicted risk quotients are presented for the Oil and Gas Scenario and the Cumulative Scenario for the non-carcinogenic COPC in Section 4.2.1. As discussed in Section 3.5 (Risk Characterization), carcinogenic COPC were only evaluated for the Oil and Gas Scenario. The results of the carcinogenic assessment are presented in Section 4.2.2.

Potential multiple pathway exposures were also assessed as the MPOI locations, even though it is unlikely that people would be exposed at the locations where the MPOIs were predicted to occur. The maximum predicted chronic air concentrations within each community were used in the multiple pathway assessment to predict human dust, soil, and plant-based foods concentrations. For the prediction of game and agricultural tissue concentrations, the MPOI concentrations for the COPC were used to account for the potential for animals to be exposed in different locations within the study area.

A worked example for the multiple pathway assessment has been included in this report as Appendix D.

4.2.1 Non-carcinogens

The estimated risk quotients for the non-carcinogenic COPC and associated mixtures are presented in Table 4-31 to Table 4-33 for each of the locations assessed within the Community, Aboriginal and Agricultural Groups, respectively. Chronic mixture effects were evaluated using the methodology outlined in Section 3.4.3. Renal toxicants were identified as the only chemical mixture of relevance for the selected COPC.

The chronic multiple pathway risk quotients are predicted to be below 0.2 for all COPC, at all locations within the Aboriginal, Agricultural, and Community Groups for both the Oil and Gas and Cumulative scenarios, including at the MPOI locations. This indicates that predicted long-term exposures to COPC are less than their health-based exposure limits, even when differences in local food consumption patterns are accounted for. All of the mixture risk

quotients in the multiple pathway assessment were also less than 0.2, indicating that the renal toxicants will contribute negligible risk.

Overall, the long-term emissions from the continuous oil and gas activities accounted for in this HHRA are not anticipated to have an adverse impact on the chronic multiple pathway health risks in the region.

Table 4–31 Chronic Multiple Pathway Risk Quotient (RQ) Values for the Community Group

Category	Chemical	Case	MPOI	Dawson Creek	Fort St. John
VOC	Acrolein	Cumulative	0.001	0.001	0.001
		Oil and Gas	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001
PAH	Fluoranthene	Cumulative	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001
PHC	Aromatic C_{17}-C_{34} group	Cumulative	0.003	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001
Mixture	Renal Toxicants	Cumulative	0.003	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001

Table 4–32 Chronic Multiple Pathway Risk Quotient (RQ) Values for the Aboriginal Group

Category	Chemical	Case	MPOI	Blueberry River and Doig River 204	Blueberry River 205	Buick	Doig River 206	East Moberly Lake 169	Halfway River 168	Kelly Lake	Moberly Lake	West Moberly Lake 168A	Wonowon
VOC	Acrolein	Cumulative	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
	Formaldehyde	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PAH	Fluoranthene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aromatic C_{17}-C_{34} group	Cumulative	0.003	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixtures	Renal Toxicants	Cumulative	0.003	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

Table 4–33 Chronic Multiple Pathway Risk Quotient (RQ) Values for the Agricultural Group

Category	Chemical	Case	MPOI	Arras	Charlie Lake	Chetwynd	Doe River	East Pine	Goodlow	Hudson's Hope	Kelly Lake	Lone Prairie	Pine Valley	Pine View	Pouce Coupe	Rolla	Rose Prairie	Taylor	Tomslake	Tumbler Ridge
VOC	Acrolein	Cumulative	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055	0.055
		Oil and Gas	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004
	Formaldehyde	Cumulative	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
		Oil and Gas	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
PAH	Fluoranthene	Cumulative	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
PHC	Aromatic C_{17}-C_{34} group	Cumulative	0.004	0.002	0.002	0.002	0.001	0.002	0.002	0.002	0.001	0.001	0.001	0.002	0.002	0.002	0.002	0.002	0.002	0.001
		Oil and Gas	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mixture	Renal toxicants	Cumulative	0.006	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004	0.004
		Oil and Gas	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002

4.2.2 Carcinogens

The estimated carcinogenic ILCR values for the locations assessed within the Aboriginal, Agricultural, and Community Groups are presented in Table 4-34, Table 4-35 and Table 4-36. As discussed in the Risk Characterization section, ILCR values were calculated only for the Oil and Gas Scenario. The ILCR values represent incremental lifetime cancer risks per 100,000 individuals, and are compared against a negligible level of risk of 1.0 in 100,000, as recommended by Health Canada (2012).

All ILCR values were predicted to be less than 1.0 in 100,000 at all locations within the Aboriginal, Agricultural, and Community Groups for the Oil and Gas Scenario. As such, when considering all relevant pathways of exposure, the incremental lifetime cancer risks associated with oil and gas activity in NE BC were associated with a negligible level of cancer risk.

Table 4–34 Chronic Incremental Lifetime Cancer Risk (ILCR) Values for the Community Group for the Oil and Gas Scenario

Category	Chemical	Incremental Lifetime Cancer Risks (per 100,000)		
		MPOI	Dawson Creek	Fort St. John
PAH	Benzo(a)pyrene Equivalent	0.080	0.001	0.001

Table 4–35 Summary of Incremental Lifetime Cancer Risk (ILCR) Values for the Aboriginal Group for the Oil and Gas Scenario

Category	Chemical	Incremental Lifetime Cancer Risks (per 100,000)										
		MPOI	Blueberry River and Doig River 204	Blueberry River 205	Buick	Doig River 206	East Moberly Lake 169	Halfway River 168	Kelly Lake	Moberly Lake	West Moberly Lake 168A	Wonowon
PAH	Benzo(a)pyrene Equivalent	0.094	0.017	0.021	0.015	0.017	0.014	0.015	0.013	0.013	0.013	0.014

Table 4–36 Summary of Incremental Lifetime Cancer Risk (ILCR) Values for the Agricultural Group for the Oil and Gas Scenario

Category	Chemical	Incremental Lifetime Cancer Risks (per 100,000)																
		MPOI	Arras	Charlie Lake	Chetwynd	Doe River	East Pine	Goodlow	Hudson's Hope	Kelly Lake	Lone Prairie	Pine Valley	Pine-view	Pouce Coupe	Rolla	Rose Prairie	Taylor	Tomslake	Tumbler Ridge
PAH	Benzo(a)pyrene Equivalent	0.181	0.097	0.098	0.097	0.098	0.097	0.098	0.097	0.097	0.097	0.097	0.102	0.097	0.097	0.104	0.104	0.097	0.097

5.0 SUMMARY AND CONCLUSIONS OF FINDINGS

This report presents the results of the detailed HHRA, the objectives of which were to understand what potential health risks exist for people who live in proximity to oil and gas activities in NE BC. The HHRA is intended to investigate the potential impact of oil and gas development on human health in Local Health Areas 59, 60 and 81 (the Region). Based on the decisions made in the Screening Level Risk Assessment (SLRA) (Intrinsik 2014a), the HHRA involved a comprehensive and focused assessment of the potential adverse health risks in relation to oil and gas activity in NE BC. Two air emission scenarios were evaluated:

1. Continuous emissions from gas processing plants.
2. Continuous emissions from production facilities.

These two scenarios were considered together to represent continuous emissions from oil and gas activity within this detailed HHRA. In addition to consideration of the combined Oil and Gas Scenario described above, a Cumulative Scenario was evaluated that considered emissions for non-oil and gas sources in order to understand the potential for air quality (and consequently human health) impacts on a cumulative basis.

The HHRA used a widely accepted approach for assessing environmental risks that has been endorsed in the past by regulatory agencies throughout Canada and internationally. The HHRA was performed step-wise following a conventional paradigm and involved the following main steps:

- Problem formulation
- Exposure assessment
- Toxicity assessment
- Risk characterization

Results were presented and described for inhalation on a short-term and long-term basis, and for all possible routes of exposure on a long-term basis.

5.1 Short-term (Acute) Inhalation Assessment Results

Short-term inhalation health risks were evaluated by comparing maximum predicted short-term air concentrations against health-based regulatory guidelines. With the exceptions of acrolein, formaldehyde, NO_2, SO_2, $PM_{2.5}$ and a number of the irritant mixtures, predicted acute risk quotients did not exceed 1.0. This demonstrates that, on a regional basis, predicted COPC air concentrations generally were less than their health-based guidelines and that the additive interactions of the COPC are not predicted to result in adverse health effects.

5.1.1 Acrolein

Although the assessment indicated the potential for some exceedances, adverse health effects in the study area in relation to short-term acrolein exposure are not anticipated, based on the following:

- For the Oil and Gas Scenario, all predicted hourly concentrations were lower than the health-based exposure limit of 2.5 µg/m³.

- For the majority of the locations evaluated, the predicted hourly concentrations were below the health-based exposure limit of 2.5 µg/m³ in the Cumulative Scenario.
- Although the MPOI for the Cumulative Scenario is anticipated to occur in the City of Fort St. John, analysis of time series data for this location indicated that for 99.9% of the time, the hourly acrolein concentrations will be below the health-based exposure limit of 2.5 µg/m³.
- All predicted hourly acrolein concentrations for both the Oil and Gas Scenario and Cumulative Scenario are well below the threshold above which acute irritation effects have been reported in human studies.

Additionally, it should be noted that the oil and gas sources included in the HHRA do not significantly contribute to potential adverse health risks in relation to short-term acrolein exposures.

5.1.2 Formaldehyde

Although an exceedance of the health-based exposure limit for formaldehyde was predicted, adverse health effects in the study area in relation to short-term formaldehyde exposure are not anticipated, based on the following rationale:

- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas Scenario and Cumulative Scenario are predicted to occur in a remote area where members of the public are unlikely to be.
- No risks associated with short-term formaldehyde exposures were identified for any of the communities evaluated on an individual basis within the study area.
- The predicted hourly formaldehyde concentrations for both the Oil and Gas Scenario and Cumulative Scenario are below the threshold above which acute irritation effects have been reported in humans, and are generally below health-based exposure limits.

5.1.3 Nitrogen Dioxide (NO_2)

Based on the results of the HHRA, the overall potential for adverse effects associated with NO_2 exposures in the region is minimal based on the following:

- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas Scenario and Cumulative Scenario are predicted to occur in a remote area.
- An exceedance of the health-based exposure limit was predicted for both Scenarios within the community of Taylor, where people may be regularly exposed. However, the time series analysis suggests that for over 99.4% of the time, hourly NO_2 concentrations would be below the exposure limit of 188 µg/m³.
- No risks associated with hourly NO_2 exposures were identified for any of the other communities evaluated within the study area.
- The predicted hourly NO_2 concentrations for the both the Oil and Gas Scenario and Cumulative Scenario are generally below the threshold above which adverse respiratory effects have been reported in human studies.

A number of communities within the HHRA study area are predicted to experience hourly NO_2 concentrations at levels slightly below the health based criteria, particularly for the Cumulative Scenario. Given the number of short-term or more intermittent emission sources in the region associated with oil and gas activities (*e.g.*, well drilling, fluid transportation, flaring, *etc.*), it is possible that the potential exposures to people in the area would be higher. Given the limited amount of available monitoring data for the study area with respect to NO_2, particularly with regards to populated areas, it is difficult to confirm this conclusively.

5.1.4 Particulate Matter ($PM_{2.5}$)

For the majority of the locations evaluated, the predicted 24-hour 98th $PM_{2.5}$ concentrations were below criteria from the BC MOE and CCME. The MPOI is predicted to occur in an isolated, forested area, and is not in proximity to established communities. It is unlikely that the general population would be exposed to the concentrations that could occur at the MPOI location. Based on the 24-hour time series data, $PM_{2.5}$ concentrations are predicted to be below the criteria about 85% of the time. Finally, the oil and gas sources included in this HHRA do not appear to significantly contribute to the overall potential adverse health risks associated with short-term $PM_{2.5}$ exposures in the region.

5.1.5 Sulphur Dioxide (SO_2)

Overall, the potential that SO_2 emissions from oil and gas activities will impact human health in the area are considered to be low.

- For the majority of the locations evaluated, for both the Oil and Gas and Cumulative Scenarios, the predicted hourly concentrations were below the exposure limits of 500 µg/m³ and 196 µg/m³ available from the WHO and US EPA respectively.
- The peak concentrations identified at the MPOI appear to be isolated and infrequent, with a limited spatial extent. The MPOIs for both the Oil and Gas and Cumulative Scenarios are predicted to occur in a remote area, where members of the general public are not likely to be.
- Exceedances were predicted for 10-minute exposures at both the City of Fort St. John and the community of Taylor. However, the time series data analysis indicates that over 99.8% of the time, the 10-minute SO_2 concentrations would be lower than the health-based guideline of 500 µg/m³.

5.1.6 Acute Irritants Mixtures

Adverse health effects in relation to short-term exposure to the irritants mixtures are not anticipated based on the following rationale:

- The low likelihood that the predicted maximum air concentrations of the “member” COPC (*i.e.*, the mixture constituents) would exceed their health-based exposure limits.
- The maximum hourly concentrations of the mixture constituents are generally lower than the levels at which responses have been observed in humans.
- The low likelihood that the maximum concentrations of the mixture constituents would occur at precisely the same time and at the exact same location.

The weight of evidence indicates a low potential for adverse health effects as a result of combined exposure to the different irritants.

5.2 Chronic Inhalation Assessment Results

Chronic inhalation risk estimates, expressed as risk quotients or ILCR values, were based on long-term exposure periods. The assumption that people could be exposed to concentrations at the MPOI over an extended period of time is conservative. Many of the MPOI concentrations appear to occur within facility boundaries or in remote areas where people are unlikely to spend appreciable amounts of their time over the long-term. The analysis of the results has been separated into two different sections, due to the differences in the assessment methods and in the interpretation of the results: (i) the non-carcinogenic inhalation assessment; and, (ii) the carcinogenic inhalation assessment.

5.2.1 Non-Carcinogens

The only COPC that presented an exceedance of a non-carcinogenic exposure limit on a chronic basis was $PM_{2.5}$. The exceedance was noted only for the Cumulative Scenario. All other chronic risk quotients values were less than 1.0. This indicates that the predicted long-term COPC concentrations at the community locations included in the HHRA were below their respective exposure limits and that the associated health risks are low. For the chemical mixtures, exceedances were predicted for the nasal and respiratory irritants.

Particulate Matter ($PM_{2.5}$)

Based on the results of this HHRA, the overall potential health risks associated with chronic $PM_{2.5}$ exposure in the study area are anticipated to be low, based on the following rationale:

- The locations where the exceedances are predicted to occur are isolated and in remote areas not likely to be frequented by the general population on a regular basis.
- No exceedances of the BC MOE objective were predicted for any of the communities in the study area, for either the Oil and Gas Scenario or Cumulative Scenario.

Chronic Irritants Mixture

Overall, the potential adverse health risks associated with chronic nasal and respiratory irritants in the region are anticipated to be low. The exceedances for the mixture risk quotients are predicted to be limited to the MPOI. All nasal and respiratory irritant risks at the individual communities were low. As well, none of the risk quotients for the chronic mixture constituents on their own were predicted to exceed 1.0. In addition, the annual MPOIs for the different mixture constituents are predicted to occur at different locations.

5.2.2 Carcinogens

Carcinogenic risks were only modelled for the Oil and Gas Scenario such that the incremental risks associated with the industry could be considered above and beyond what is contributed by ambient or regional sources. The only COPC for which an ILCR greater than 1 in 100,000 was predicted was formaldehyde. This ILCR value was associated with the annual MPOI concentration for the Oil and Gas Scenario. The ILCR values for formaldehyde at all of the other

locations evaluated in the HHRA were less than 1.0 in 100,000 for the Oil and Gas Scenario. ILCR values for all other COPC were less than 1 in 100,000.

Due to the incremental lifetime cancer risks for formaldehyde, exceedances were also predicted for the nasal carcinogenic mixture.

Formaldehyde

Overall, the potential carcinogenic impacts of formaldehyde emissions from oil and gas sources are anticipated to be minimal for the following reasons:

- The two locations with exceedances of the exposure limit are both located in fairly remote areas, near oil and gas facilities.
- These predicted maximum concentrations are anticipated to be spatially limited, with concentrations decreasing rapidly with increasing distance from the emission source.
- No ILCR values for any of the communities included in the HHRA were predicted to be above the generally acceptable level of 1 in 100,000.

Nasal Carcinogens

The summed ILCR for the chronic nasal carcinogen mixture was predicted to exceed 1.0 in 100,000 for the Oil and Gas Scenario at the MPOI. The predicted cancer risks at all of the individual community locations were determined to be less than 1.0 (in 100,000), suggesting that long-term exposure to the COPC that have the potential to cause nasal cancer are not anticipated to result in adverse health effects.

Over 99.9% of the mixture risk is due to formaldehyde at the MPOI. Overall, the potential for adverse health effects in the study area as a result of exposure to chronic nasal carcinogens is minimal because the exceedances are expected to occur in a remote area. On a regional basis, the risks are considered to be low.

5.3 Chronic Multiple Pathway Assessment Results

The multiple pathway assessment was completed based on the assumption that people living in the area (*i.e.*, Aboriginal Group, Agricultural Group and Community Group) could be exposed to COPC via multiple exposure pathways over their entire lifetime. The multiple pathway assessment focused only on COPC that satisfied the environmental fate and persistence criteria that were used to determine the likelihood that exposure might occur through secondary pathways. Predicted health risks are expressed as risk quotients for the non-carcinogenic COPC and as ILCR values for the carcinogenic COPC. Separate assessments were completed for non-carcinogenic and carcinogenic exposures, reflecting the different approaches used in calculating and interpreting the risk estimates.

For non-carcinogens, the chronic multiple pathway risk quotients are predicted to be below 0.2 for all COPC and COPC mixtures, at all locations within the Aboriginal, Agricultural, and Community Groups for both the Oil and Gas and Cumulative Scenarios. This indicates that predicted long-term exposures to COPC are less than their exposure limits, even when differences in local food consumption patterns are accounted for. For the carcinogens, all ILCR values were predicted to be less than 1 in 100,000 at all locations within the Aboriginal,

Agricultural, and Community Groups for the Oil and Gas Scenario, indicating that emissions from the oil and gas activities included in this HHRA are associated with a negligible cancer risk via secondary exposure pathways.

Overall, health risks in the region associated with multiple pathways of exposure to the COPC and mixtures are considered low.

6.0 NEXT STEPS

The next steps of the Phase 2 HHRA project are described below.

6.1 Review of British Columbia Regulatory Framework

The Phase 2 HHRA study also includes a review of the existing BC regulatory framework. Information will be collected and reviewed in relation to applicable statutes, legislation and policy frameworks relevant to the oil and gas activities in NE BC. A number of scenarios discussed within the SLRA (Intrinsik 2014a) were recommended for further evaluation within the regulatory review. These scenarios are summarized in Table 6–1.

Table 6–1 Summary of Scenarios from the SLRA to be Evaluated in the Regulatory Review

Potential Emission Source	Scenario
Air Emission Scenarios	
Emergency Flaring	1-3 Gas Processing Plants (Emergency Flaring) 2-3 Production Facilities (Emergency Flaring)
Routine Flaring	1-2 Gas Processing Plants (Flaring) 2-1 Production Facilities (Flaring) 3-1 Wells Drilling (Clean up and Testing)
Fugitive Leaks	Fugitive leaks were not explicitly evaluated as separate scenarios in the air matrix. Small fugitive gas leaks from wells, pipelines, batteries and metering buildings emit very small amounts of hydrocarbon products from seals, pumps and flanges due to wear, damage, manufacturing flaws, poor design or installation.
Emissions from Well Site Activities	When wells are drilled, put into service and connected to flow lines or pipelines, there are a number of necessary activities that could result in emissions to the environment.
Uncontrolled Releases	Well drilling scenarios (3-3 to 3-6) Producing well scenarios (4-1 to 4-4) Non-producing well scenarios (5-1 to 5-4) Pipeline leaks and ruptures (Scenarios 6-1 to 6-10)
Water Emission Scenarios	
Operational Sites	Exploration
	Construction
	Conventional drilling
	Hydraulic fracturing
	Production
	Processing
Historical Sites	In-ground fluid pits and flare system overflows
	Unlined storage tank areas
	Onsite disposal facilities
Transportation and Storage of Products and Wastes	Pipeline leaks Spills

6.2 Project Recommendations

The final task to be completed by the Phase 2 HHRA study team will be to develop a series of recommendations. Based on the outcomes of the earlier tasks (such as this HHRA), recommendations may be made regarding changes in air quality monitoring, water resource monitoring and even human health monitoring that could be provided to ensure that the health of residents in Local Health Areas 59, 60 and 81 is protected. This task may provide additional recommendations or protocols to be employed to assess the potential for future human health impacts in NE BC as a result of historical, continued or future oil and gas activities, and for information management and sharing.

7.0 REFERENCES

AHW (Alberta Health and Wellness). 2011. Guidance on Human Health Risk Assessment for Environmental Impact Assessments in Alberta. August 2011.

Ana, G.R.E.E., Sridhar, M.K.C. and Emerole, G.O. 2012. Polycyclic aromatic hydrocarbon burden in ambient air in selected Niger Delta communities in Nigeria. Journal of the Air & Waste Management Association 62:18-25.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Sulphur Dioxide. Atlanta, GA: US Department of Health and Human Services, Public Health Service. July 1999.

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

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Toluene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. September 2000.

ATSDR (Agency for Toxic Substances and Disease Registry). 2006. Toxicological Profile for Hydrogen Sulfide. Atlanta, GA: US Department of Health and Human Services, Public Health Service. July 2006.

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

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

Azadniv, M., Utell, M.J., Morrow, P.E., Gibb, F.R., Nichols, J., Roberts, N.J. Jr., Speers, D.M., Torres, A., Tsai, Y., Abraham, M.K., Voter, K.Z. and Frampton, M.W. 1998. Effects of nitrogen dioxide exposure on human host defense. Inhal Toxicol 10(6): 585-601.

BC ACF (British Columbia Agriculture in the Classroom Foundation). 2008. Grow BC: The Regions. Peace River North East Agriculture Profile. Accessed at: http://www.aitc.ca/bc/uploads/growbc/5_regions.pdf.

BC MOE (British Columbia Ministry of the Environment). 2008. Guidelines for Air Quality Dispersion Modelling in British Columbia. Victoria, BC. 152 pp.

BC MOE (British Columbia Ministry of the Environment). 2011a. Peace Region Air Quality Survey: Data Summary Report. Mobile Air Monitoring Laboratory (MAML). N.E. BC – Kelly Lake. July 26, 2011.

BC MOE (British Columbia Ministry of the Environment). 2011b. Peace Region Air Quality Survey: Data Summary Report. Mobile Air Monitoring Laboratory (MAML). N.E. BC. Farmington, Groundbirch, Rolla, Tomslake.

BC MOE (British Columbia Ministry of the Environment). 2012a. Technical Guidance on Contaminated Sites. October 2012. Accessed at: http://www.env.gov.bc.ca/epd/remediation/guidance/technical/pdf/tg07-v3.pdf

BC MOE (British Columbia Ministry of the Environment). 2012b. 2010 Annual Air Quality Report. Prince George Airshed. Environmental Quality Section, Environmental Protection Division. Omineca and Peace Regions. Prince George, British Columbia. June 2012.

BC MOE (British Columbia Ministry of the Environment). 2013. 2011-2012 Prince George Neighbourhood Volatile Organic Compound (VOC) Sampling Program.

BC MOE (British Columbia Ministry of the Environment). 2014a. Report on Initial Network Design – NE BC Air Quality Network. Report to the SCEK Fund, British Columbia Oil and Gas Commission. January 31, 2014.

BC MOE (British Columbia Ministry of the Environment). 2014b. Northeast Monitoring Project (MOE Phase 1). Final Report for Recipient Agreement HS-2013-01-01. Report to the SCEK Fund, British Columbia Oil and Gas Commission. January 31, 2014.

BC MOE (British Columbia Ministry of the Environment). 2014c. British Columbia Air Data Archive Database. Available at: http://envistaweb.env.gov.bc.ca/. Accessed: May 2014.

BC MOE (British Columbia Ministry of the Environment). 2014d. BC Air Quality – Emissions Inventories. May 2014. Accessed at: http://www.bcairquality.ca/assessment/emissions-inventories.html

BC OGC (British Columbia Oil and Gas Commission). 2013. Hydrocarbon and By-Product Reserves in British Columbia 2011. February 21, 2013. Accessed at: http://www.bcogc.ca/node/8214/download

BC Hydro. 2012. Volume 2 Appendix L. Air Quality Technical Data Report. Environmental Impact Assessment. Site C Clean Energy Project. November 2012.

BC Stats, 2013 Sub-Provincial Population Estimates. Released: January 31, 2014

BCS (Bureau of Chemical Safety), Health Canada. 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

Beil, M. and Ulmer, W.T. 1976. Wirkung von NO_2 im MAK-Bereich auf Atemmechanik und bronchiale Acetylcholinempfindlichkeit bei Normalpersonen [Effect of NO_2 in workroom concentrations on respiratory mechanics and bronchial susceptibility to acetylcholine in normal persons]. Int. Arch. Occup. Environ. Health 38: 31-44.

Blomberg, A., Krishna, M.T., Bocchino, V., Biscione, G.L., Shute, J.K., Kelly, F.J., Frew, A.J., Holgate S.T. and Sandstrom. T. 1997. The inflammatory effects of 2 ppm NO_2 on the airways of healthy subjects. Am J Respir Crit Care Med 156: 418-424.

Blomberg, A., Krishna, M.T., Helleday, R., Söderberg, M., Ledin, M.C., Kelly, F.J., Frew, A.J., Holgate, S.T. and Sandström. T. 1999. Persistent airway inflammation but accommodated antioxidant and lung function responses after repeated daily exposure to nitrogen dioxide. Am. J. Respir. Crit. Care Med. 159: 536-543.

Boethling, R.S., K. Fenner, P. Howard, G. Klecka, T. Madsen, J.R. Snape and M.J. Whelan. 2009. Environmental persistence of organic pollutants: guidance for development and review of POP risk profiles. Integr Environ Assess Manag 5(4):539-556.

Bolt, H.M. and P. Morfeld. 2013. New results on formaldehyde: the 2nd International Formaldehyde Science Conference (Madrid, 19-20 April 2012). Arch Toxicol 87: 217-222.

British Columbia Vital Statistics Agency. 2011. Selected Vital Statistics and Health Status Indicators. One Hundred and Fortieth Annual Report 2011.

Burstyn, I., You, X., Cherry, N. and Senthilselvan, A. 2007. Determinants of airborne benzene concentrations in rural areas of western Canada. Atmospheric Environment 41:7778-7787.

Canadian Community Health Survey. 2004. A Guide to Accessing and Interpreting the Data.

California Environmental Protection Agency. 1999. Air Toxics Hot Spots Program Risk Assessment Guidelines Part I: Determination of Acute Reference Exposure Levels for Airborne Toxicants. Air Toxicology and Epidemiology Section, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency. March 1999.

California Environmental Protection Agency. 2007. Review of the California Ambient Air Quality Standard For Nitrogen Dioxide: Technical Support Document. California Environmental Protection Agency Air Resources Board and Office of Environmental Health and Hazard Assessment. Available at: http://www.arb.ca.gov/research/aaqs/no2-rs/no2tech.pdf

CIHI (Canadian Institute for Health Information). 2006. How Healthy are Rural Canadians? An Assessment of their Health Status and Health Determinants. Canadian Population Health Initiative. Ottawa, ON.

CARB (California Air Resources Board). 2005. Particulate Matter - Overview. Accessed at: http://www.arb.ca.gov/research/aaqs/caaqs/pm/pm.htm. Webpage updated April, 2005.

CCME (Canadian Council of Ministers of the Environment). 2000. Canada-Wide Standards for Particulate Matter (PM) and Ozone. Accepted November 29, 1999, for endorsement in May 2000.

CCME (Canadian Council of Ministers of the Environment). 2006. A protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. ISBN-10 1-896997-45-7 PDF.

CCME (Canadian Council of Ministers of the Environment). 2008. Canada Wide Standards for Petroleum Hydrocarbons (PHC) in Soil: Scientific Rationale. Supporting Technical Document. January, 2008. ISBN 978 1 896997 77

CCME (Canadian Council of Ministers of the Environment). 2012a. Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone.

CCME (Canadian Council of Ministers of the Environment). 2012b. Guidance Document on Air Zone Management. PN 1481 978-1-896997-89-6-PDF.

CCS (Canadian Cancer Society). 2010. General cancer statistics for 2010. Accessed at: http://www.cancer.ca/Canada wide/about%20cancer/cancer%20statistics/stats%20at%20a%20glance/general%20cancer%20stats.aspx

Center for Energy 2013. Statistics. Accessed at: www.centerforenergy.com

Chan, L., Receveur, O., Sharp, D., Schwartz, H., Ing, A. 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.

Cochrane Handbook for Systematic Reviews of Interventions. 2008. Version 5.0.2. Editors: J.P.T Higgins and S. Green. Accessed at: www.cochrane-handbook.org.

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.

Devlin, R.B., Horstman, D.P., Gerrity, T.R., Becker S. and Madden, M.C. 1999. Inflammatory response in humans exposed to 2.0 ppm nitrogen dioxide. Inhalation Toxicol. 11: 89-109.

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

Environment Canada. 2014. National Air Pollutant Surveillance Database. Available at: http://www.ec.gc.ca/rnspa-naps/default.asp?lang=En&n=8BA86647-1.

Fang, R., Kmetic, A., and McCarney, J. 2010. Summary Report on Health for British Columbia from Regional, Longitudinal and Gender Perspectives. Prepared for J. Millar and L. Drasic. Provincial Health Services Authority, Vancouver, B.C.

FBC (Fraser Basin Council). 2012. Identifying Health Concerns Relating to Oil and Gas Development in Northeastern British Columbia. Human Health Risk Assessment Phase 1 Report. A Report of the Fraser Basin Council to the British Columbia Ministry of Health. March 30, 2012.

Forastiere, F., Corbo, G.M., Dell'Orco, V., Pistelli, R., Agabiti, N. and Kriebel, D. 1996. A longitudinal evaluation of bronchial responsiveness to methacholine in children: role of baseline lung function, gender and change in atopic status. Am J Respir Crit Care Med. Mar; 153(3):1098-104.

Gong, H., Jr., Linn, W.S., Clark, K.W., Anderson, K.R., Geller, M.D. and Sioutas, C. 2005. Respiratory responses to exposures with fine particulates and nitrogen dioxide in the elderly with and without COPD. Inhalation Toxicol. 17: 123-132.

Goodman, J.E., Chandalia, J.K., Thakali, S. and Seeley, M. 2009. Meta-analysis of nitrogen dioxide exposure and airway hyper-responsiveness in asthmatics. Critical Reviews in Toxicology 39(9):719-742.

Government of BC (Government of British Columbia). 2014. British Columbia Environmental Monitoring System (EMS). Available at: http://www.env.gov.bc.ca/emswr/ Accessed: May 2014.

Health Canada. 1994. Human Health Risk Assessment for Priority Substances. Canadian Communication Group Publishing. Ottawa, ON.

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2007. Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption. Bureau of Chemical Safety. Food Directorate. Health Products and Food Branch. March 2007.

Health Canada. 2009. Supplemental Guidance on Human Health Risk Assessment on Air Quality ($HHRA_{Air}$). Version 1.0. Draft. Federal Contaminated Site Risk Assessment in Canada. Contaminated Sites Division. Government of Canada.

Health Canada. 2010. Part V: Guidance on Human Health Detailed Quantitative Risk Assessment. Federal Contaminated Site Risk Assessment in Canada. Contaminated Sites Division. Government of Canada. ISBN: 978-1-100-17926-1

Health Canada. 2012. Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). Version 2.0. Federal Contaminated Site Risk Assessment in Canada. Contaminated Sites Division. Government of Canada.

HSDB (Hazardous Substances Data Bank). 2010. Database on-line search for Sulfur dioxide. Available at http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?./temp/~ME7p1C:1.

Hytonen *et al*., 2009, Gas-Particle Distribution of PAHs in Wood combustion Emission Determined with Annular Denuders, Filters, and Polyurethane Foam Adsorbent, Aerosol Science and Technology, 43:5, 442-454.

IHS 2013a. BC Well Data [Database]. Calgary, Alberta.

IHS 2013b. GDM Midstream Data for BC [Database]. Calgary, Alberta.

IHS Energy. (n.d.). Energy Information, Software & Solutions (EGIS 8) database. Available: http://www.ihs.com/products/oil-gas-information/data-access/egis.aspx

Intrinsik (Intrinsik Environmental Sciences Inc.). 2014a. Final Screening Level Risk Assessment. Phase 2 Human Health Risk Assessment of Oil and Gas Activity in Northeastern British Columbia.

Intrinsik (Intrinsik Environmental Sciences Inc.). 2014b. Draft Review of Regulatory Frameworks. Phase 2 Human Health Risk Assessment of Oil and Gas Activity in Northeastern British Columbia.

Intrinsik Environmental Sciences Inc. 2013. Phase 2 Human Health Risk Assessment – Task 3: Literature Review. Prepared by: McDaniel Lambert for Intrinsik.

Jorres, R., Nowak, D., Grimminger, F., Seeger, W., Oldigs, M. and Magnussen, H. 1995. The effect of 1 ppm nitrogen dioxide on bronchoalveolar lavage cells and inflammatory mediators in normal and asthmatic subjects. Eur. Respir. J. 8: 416-424.

Krzyzanowski, J. 2012. Environmental pathways of potential impacts to human health from oil and gas development in northeast British Columbia, Canada. Environ Rev 20: 122-134.

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

Mackay, D., W.Y. Shiu and S. Paterson. 1992. Generic Models for evaluating the regional fate of chemicals. Chemosphere. 24. pp. 695–717.

Market Facts of Canada. 1991. Research Report: National Seafood Consumption Study. Conducted for: Health and Welfare Canada. #C388/JdeB. Cited in: Health Canada 2007.

McKendry, I.G. 2006. Background Concentrations of $PM_{2.5}$ and Ozone in British Columbia, Canada. Prepared for the British Columbia Ministry of the Environment. Geography/Atmospheric Science, The University of British Columbia. March 2006.

McKenzie, L.M., Witter, R.Z. Newman, L.S. and Adgate, J.L. 2012. Human health risk assessment of air emissions from development of unconventional natural gas resources. Science of the Total Environment. (Article in Press).

Moran, Mike, Environment Canada. Personal Communication with Martin Gauthier RWDI AIR Inc. December 2013.

Morrow, P.E., Utell, M.J., Bauer, M.A., Smeglin, A.M., Frampton, M.W., Cox, C., Speers D.M. and Gibb, F.R. 1992. Pulmonary performance of elderly normal subjects and subjects with chronic obstructive pulmonary disease exposed to 0.3 ppm nitrogen dioxide. Am Rev Respir Dis 145: 291-300.

Mossop, G. and I. Shetsen. 1994. Geological Atlas of the Western Canadian Sedimentary Basin. Canadian Society of Petroleum Geologists, Calgary, Alberta.

NAS (National Academies of Science). Review of the Environmental Protection Agency's Draft IRIS Assessment of Formaldehyde. Committee to Review EPA's Draft IRIS Assessment of Formaldehyde. National Research Council. ISBN: 0 309 21194 8.

Nieding, G. von and Wagner, H.M. 1977. Experimental studies on the short-term effect of air pollutants on pulmonary function in man: two-hour exposure to NO_2, O_3 and SO_2 alone and in combination. In: Kasuga, S., N. Suzuki, T. Yamada, G. Kimura, K. Inagaki and K. Onoe (eds.). Proceedings of the fourth international clean air congress, May, Tokyo, Japan. Tokyo, Japan: Japanese Union of Air Pollution Prevention Associations. P.p. 5-8.

Nieding, G. von, H.M. Wagner, H. Casper, A. Beuthan and U. Smidt. 1980. Effect of experimental and occupational exposure to NO_2 in sensitive and normal subjects. In: Lee, S. D. (ed.). Nitrogen oxides and their effects on health. Ann Arbor, MI: Ann Arbor Science Publishers, Inc. P.p. 315-331.

Nieding, G. von, Wagner, H.M., Krekeler, H., Loellgen, H., Fries, W. and Beuthan, A. 1979. Controlled studies of human exposure to single and combined action of NO_2, O_3 and SO_2. Int. Arch. Occup. Environ. Health 43: 195-210.

NIOSH (National Institute of Occupational Safety and Health). 1974. Criteria for a Recommended Standard - Occupational Exposure to Sulphur Dioxide. US Department of Health, Education and Welfare, Public Health Service, Center for Disease Control, National Institute of Occupational Safety and Health. DHHS (NIOSH) Publication No. 74-111.

Northern Health. 2012. Community Health Synopsis- Fort St. John, LHA-60- Peace River North.

Northern Health. 2013a. Community Health Atlas. Accessed at: http://www.phsa.ca/communityhealthatlas

Northern Health. 2013b. Healthy Northern Communities 2013. Fort St. John. Revised September 24, 2013.

Northern Health. 2013c. Healthy Northern Communities 2013. Dawson Creek. Revised September 24, 2013.

O'Connor Associates Environmental Inc. and Richardson, G.M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. 1155-2720 Queensview Dr., Ottawa, Ontario.

OEHHA (California Office of Environmental Health Hazard Assessment). 2008a. TSD for Noncancer RELs. Appendix D. Individual acute, 8 hour, and chronic reference exposure levels. Acrolein (page 42). December 2008. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Accessed at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf

OEHHA (California Office of Environmental Health Hazard Assessment). 2008b. TSD for Noncancer RELs. Acetaldehyde Reference Exposure Levels. Appendix D1. December 2008. Accessed at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=5

Pope, C.A. III, Burnett, R.T., Thun. M.J., Calle, E.E., Krewski, D., Ito, K. and Thurston, G.D. 2002. Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate air pollution. JAMA 287 287(9): 1132-1141.

Pope, C.A. III and Dockery, D.W. 2006. Health effects of fine particulate air pollution: lines that connect. J Air & Waste Manage Assoc 56:709-742

Public Health Agency of Canada. 2012. National Single Day Food Consumption Report: Analysis of the 24-hour dietary recall data from the Canadian Community Health Survey (CCHS). Cycle 2.2, Nutrition (2004) and assessment for food consumption frequency among Canadians.

SAG (Stakeholder Advisory Group). 2013. Stakeholder Advisory Group Meeting Notes. March 26 2013, Fort St. John, British Columbia.

Sassi, Mourrand. Air Quality Modelling and Applications Section of Environment Canada, Dorval, QC Personal Communication with Martin Gauthier RWDI AIR Inc. January 2014.

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

Statistics Canada. 2006. 2006 Aboriginal Community Data Initiative. Doig River First Nation. Available at: http://www.bcstats.gov.bc.ca/StatisticsBySubject/AboriginalPeoples/AboriginalReports.aspx.

Statistics Canada. 2011. Health Profile October 2011. Northeast Health Service Delivery Area 5953-E. British Columbia (Health Region).

Statistics Canada. 2013. National Household Survey Profile. 2011 National Household Survey. Statistics Canada Catalogue no. 99-004-XWE. Ottawa. Released June 26 2013. http://www12.statcan.gc.ca/nhs-enm/2011/dp-pd/prof/index.cfm?Lang=E

Strang, Al. BC Hydro. Personal Communication with David Chadder RWDI AIR Inc. February 2014.

Strosher. 1996. Investigations of Flare Gas Emissions in Alberta. Prepared for Environment Canada Conservative and Protection, Alberta Energy and Utilities Board and the Canadian Association of Petroleum Producers. 158 pp. Website: http://www.ags.gov.ab.ca/publications/SPE/PDF/SPE_005.PDF. Accessed: March 2014.

Swenberg J.A., Moeller, B.C., Lu, K., Rager, J.E., Fry, R. and T.B. Staff. 2013. Formaldehyde Carcinogenicity Research: 30 years and Counting for Mode of Action, Epidemiology, and Cancer Risk Assessment.

TCEQ (Texas Commission on Environmental Quality). 2007a. Benzene (CAS Registry Number: 71 43 2). Developmental Support Document, Final, October 2007. Prepared by: Haney, Joseph T. Jr. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2007b. n Hexane (CAS Registry Number: 100 54 3). Developmental Support Document, Final, October 2007. Prepared by: Kadlubar, Bernard J. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality: Austin Texas.

TCEQ (Texas Commission on Environmental Quality). 2008. Formaldehyde (CAS Registry Number: 50-00-0). Developmental Support Document, Final, August 2008. Prepared by: Haney, Joseph T. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2009. Xylenes. (CAS Registry Numbers: Xylenes mixture: 1330 20 7; m xylene: 108 38 3; o Xylene: 95 47 6; p Xylene: 106 42 3). Development Support Document, Final, February 2009. Prepared by: Manuel Reyna, Toxicology Division, Chief Engineer's Office, Texas Commission on Environmental Quality. Accessed at: http://www.tceq.state.tx.us/assets/public/implementation/tox/dsd/final/xylenes_all_isomers_final_2 13 09.pdf

TCEQ (Texas Commission on Environmental Quality). 2011. Pentane, All Isomers (CAS Registry Numbers: n Pentane: 109 66 0, Isopentane: 78 78 4, Neopentane: 463 82 1. Developmental Support Document, Final, July 29, 2011. Prepared by: Jong Song Lee, Ph.D. Toxicology Division, Chief Engineer's Office, Texas Commission on Environmental Quality.

US EPA (United States Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part A). OERR. Washington, D.C. OERR 9200 6-303-894.

US EPA (United States Environmental Protection Agency). 1991. IRIS Summary of Formaldehyde (CASRN 50-00-0). Carcinogenicity Assessment for Lifetime Exposure. Accessed at: http://www.epa.gov/ncea/iris/subst/0419.htm#carc

US EPA (United States Environmental Protection Agency). 1994. Review of the National Ambient Air Quality Standards for Sulfur Oxides: Assessment of Scientific and Technical Information. Supplement to the 1986 OAQPS Staff Paper Addendum. Research Triangle Park, NC: Air Quality Management Division, Office of Air Quality Planning and Standards, US Environmental Protection Agency. EPA/452/R-94/013. September 1994.

US EPA (United States Environmental Protection Agency). 1996. AP-42, Fifth Edition Compilation of Air Pollutant Emission Factors. Volume I: Stationary Point and Area Sources. Section 3.3: Gasoline and Diesel Industrial Engines. Research Triangle Park, NC. 74 pp.

US EPA (United States Environmental Protection Agency). 1998. Toxicological Review of Naphthalene (CAS No. 91 20 3) In Support of Summary Information on the IRIS. August 1998. US EPA Washington DC. Accessed at: http://www.epa.gov/ncea/iris/toxreviews/0436tr.pdf

US EPA (United States Environmental Protection Agency). 2000. IRIS Summary of Benzene (CASRN 71 43 2). Carcinogenicity Assessment for Lifetime Exposure. Accessed at: http://www.epa.gov/ncea/iris/subst/0276.htm#carc.

US EPA (United States Environmental Protection Agency). 2002. Health Assessment of 1,3 butadiene. National Center for Environmental Assessment Washington Office. Office of Research and Development. Washington, DC. October 2002. EPA/600/P 98/001F

US EPA (United States Environmental Protection Agency). 2003a. Cyclohexane. Chronic Inhalation Reference Concentration. United States Environmental Protection Agency. Accessed at: http://www.epa.gov/ncea/iris/subst/1005.htm

US EPA (United States Environmental Protection Agency). 2003b. Toxicological Review of Hydrogen Sulfide (CAS No. 7783-06-4) In Support of Summary Information on the Integrated Risk Information System (IRIS). June 2003. Accessed at: http://www.epa.gov/ncea/iris/toxreviews/0061tr.pdf

US EPA (United States Environmental Protection Agency). 2003c. User's Manual. Swimmer Exposure Assessment Model (SWIMODEL) Version 3.0. U.S. Environmental Protection Agency. Office of Pesticide Programs. Antimicrobials Division. November 2003.

US EPA (United States Environmental Protection Agency). 2005. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. March 2005. EPA/630/P-03/001F.

US EPA (United States Environmental Protection Agency). 2008. Integrated Science Assessment for Oxides of Nitrogen - Health Criteria. National Center for Environmental Assessment. EPA/600/R-08/071.

US EPA (United States Environmental Protection Agency). 2010a. 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 Rule.

US EPA (United States Environmental Protection Agency). 2010b. Provisional Peer Review Toxicity Values for 1,2,3-trimethylbenzene (CASRN 526-73-8). Accessed at: http://hhpprtv.ornl.gov/issue_papers/Trimethylbenzene123.pdf

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

US EPA (United States Environmental Protection Agency). 2012. Benchmark Dose Technical Guidance. Risk Assessment Forum. EPA/100/R-12/001

US EPA (United States Environmental Protection Agency). 2013. EPA Integrated Science Assessment for Ozone and Related Photochemical Oxidants

US EPA OSW (United States Environmental Protection Agency Office of Solid Waste). 2005. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Final. US Environmental Protection Agency Region 6. Multimedia Planning and Permitting Division. Center for Combustion Science and Engineering, Office of Solid Waste.

Vagaggini, B., Paggiaro, P.L., Giannini, D., Di Franco, A., Cianchetti, S., Carnevali, S., Taccola, M., Bacci, E., Bancalari, L., Dente, F.L. and Giuntini, C. 1996. Effect of short-term NO_2 exposure on induced sputum in normal, asthmatic and COPD subjects. Eur Respir J 9: 1852-1857.

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.

WHO (World Health Organization). 1979. Environmental Health Criteria 8 Sulfur oxides and suspended particulate matter. International Programme on Chemical Safety. ISBN 92 4 154068 0.

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

WHO (World Health Organization). 2005. WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide. Global Update 2005. Summary of Risk Assessment. WHO/SDE/PHE/OEH/06.02.

WHO (World Health Organization). 2010. WHO Guidelines for Indoor Air Quality. Selected Pollutants. WHO Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, ISBN 978 9289002134.

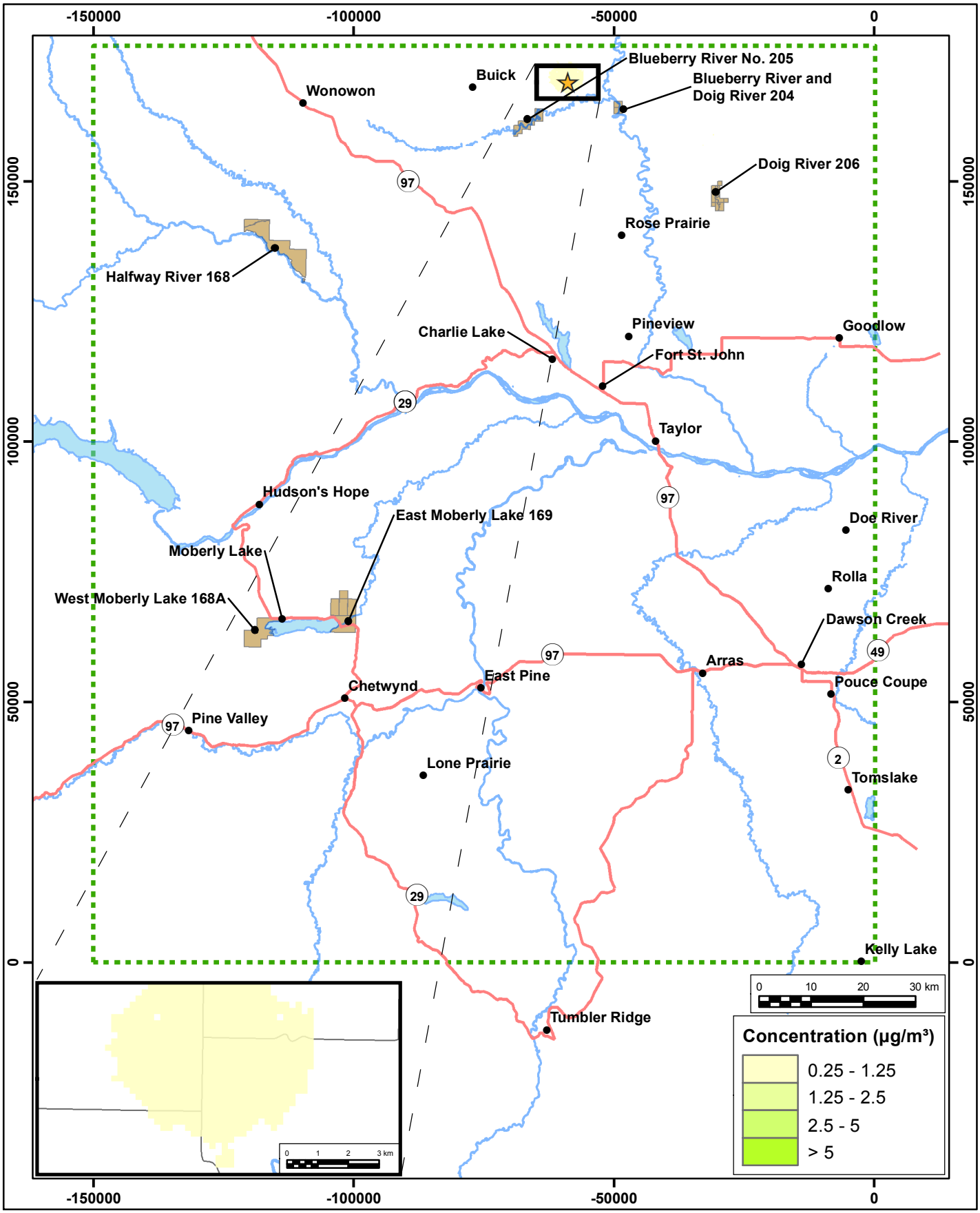
Wilson, R.M. 2005. Guidance on Air Quality Risk Assessment, Version 1. Prepared for Health Canada. HECS-SEP-BC/Yukon-04/05-06. August 8, 2005.

Witter, R., Stinson, K., Sackett, H., Putter, S., Kinney, G., Teitelbaum, D., Newman, L. 2008. Potential Exposure-Related Human Health Effects of Oil and Gas Development: A Literature Review (2003-2008).

Witter, R.Z., McKenzie, L., Stinson, K.E., Scott, K., Newman, L.S. and Adgate, J. 2011. The use of health impact assessment for a community undergoing natural gas development. Am J Public Health. doi:10.2105/AJPH.2012.301017

You, X., Senthilselvan, A., Cherry, N.M., Kim, H-M. and Burstyn, I. 2008. Determinants of airborne concentrations of volatile organic compounds in rural areas of Western Canada. Journal of Exposure Science and Environ

Attachment 1 – Isopleth Figures



Acrolein - 1hr Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

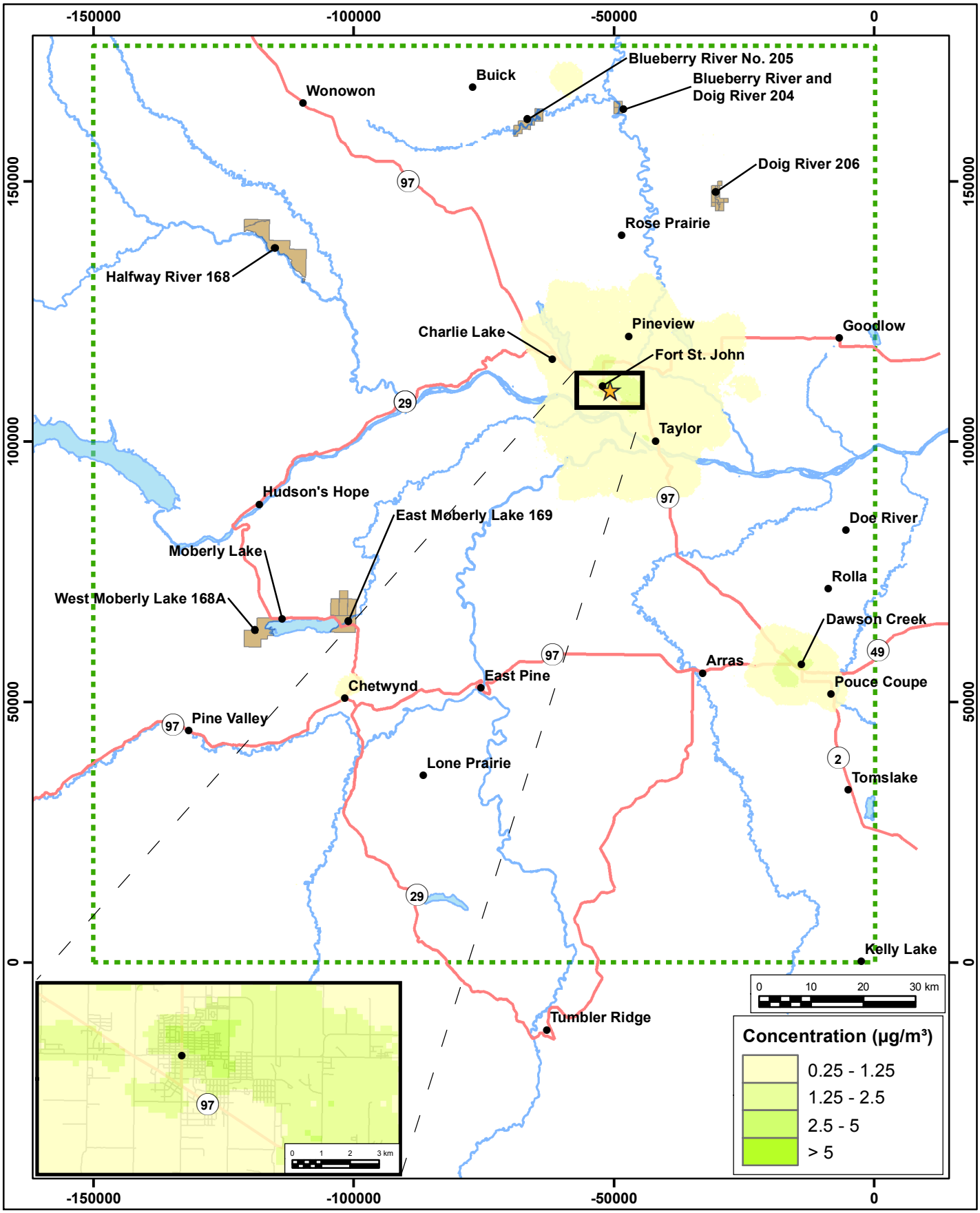
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 1
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Acrolein - 1hr Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

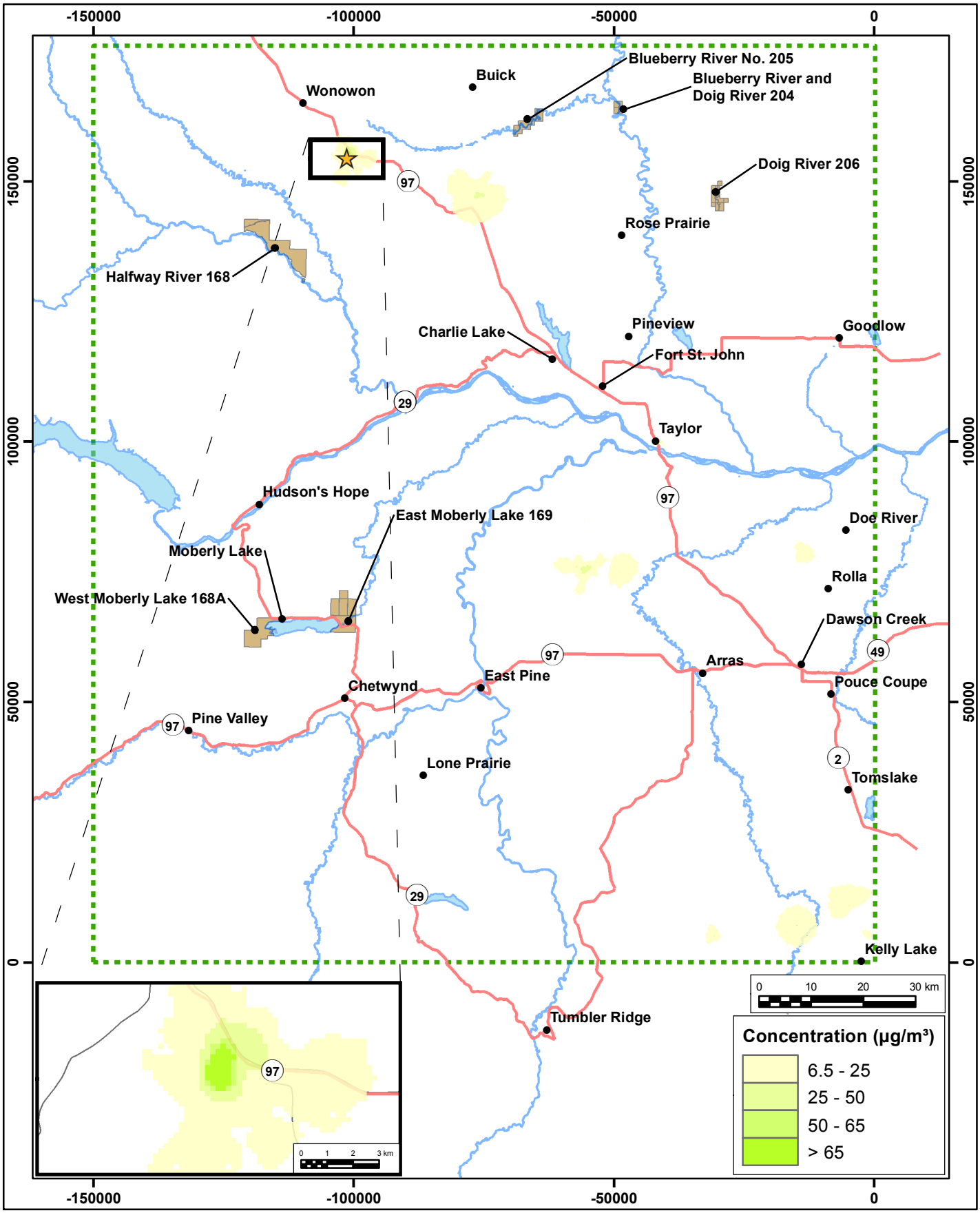
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 2
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - 1hr Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

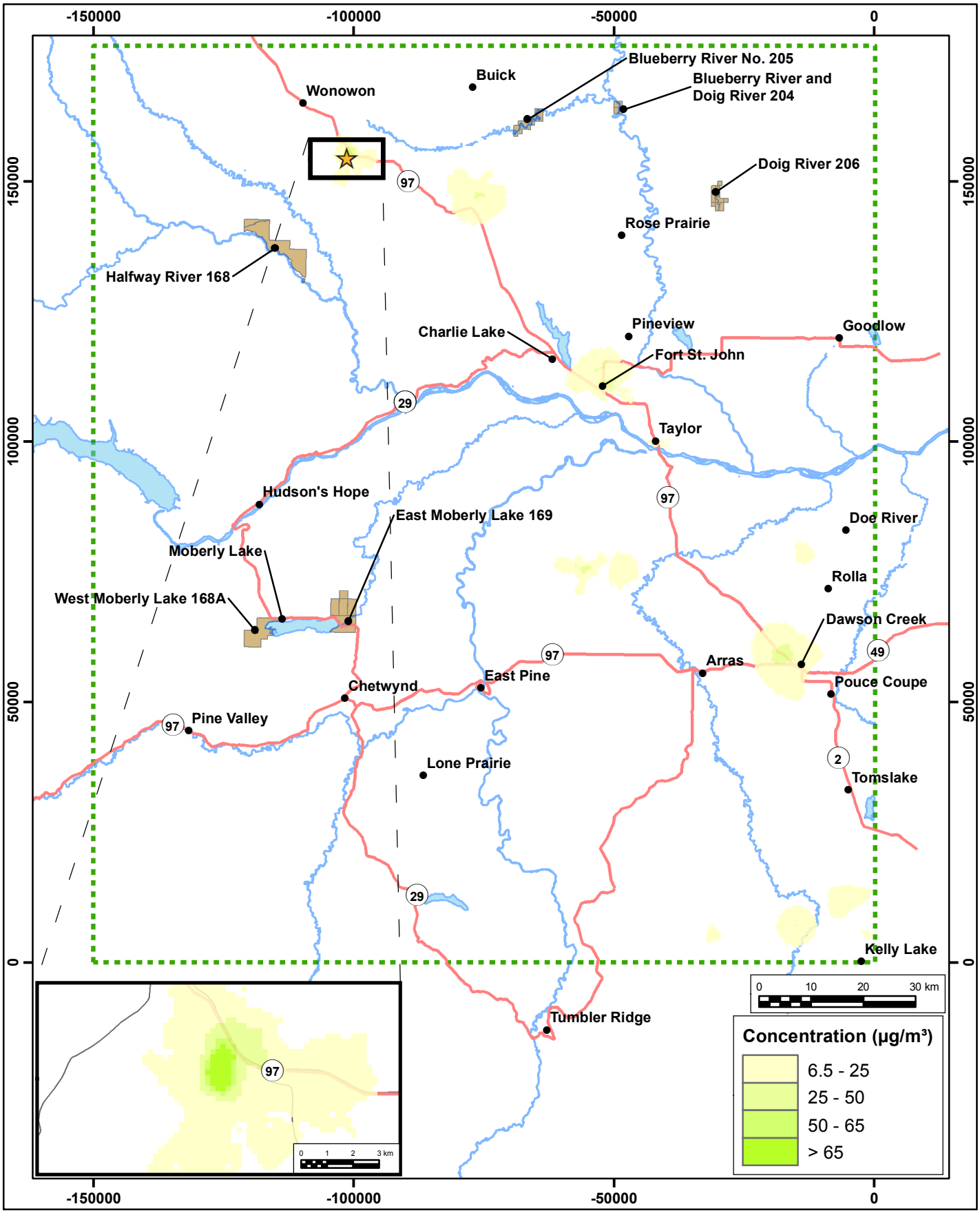
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 3
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - 1hr Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

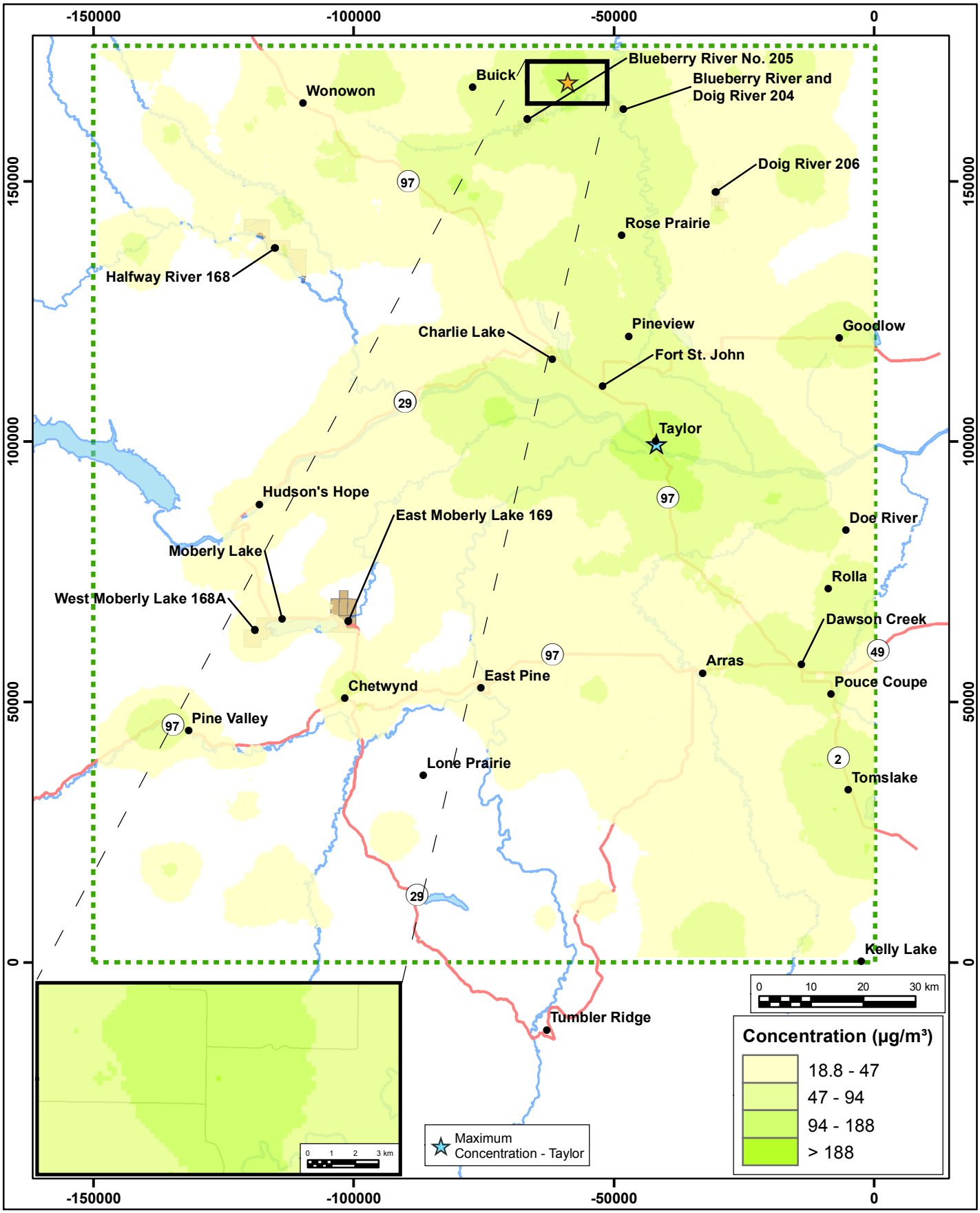
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 4
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



NO_2 - 1hr Averaging Period (98th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

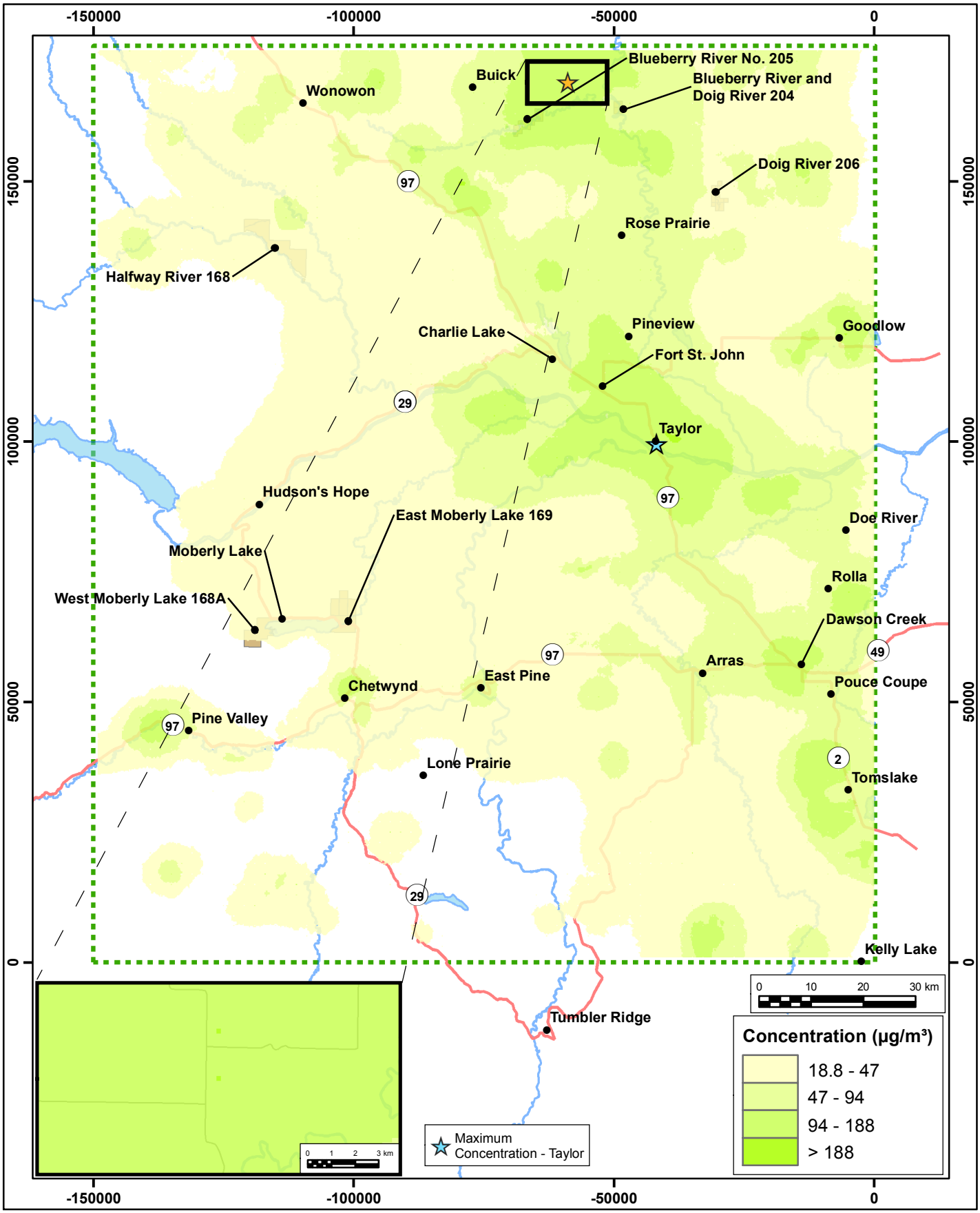
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 5
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



NO_2 - 1hr Averaging Period (98th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

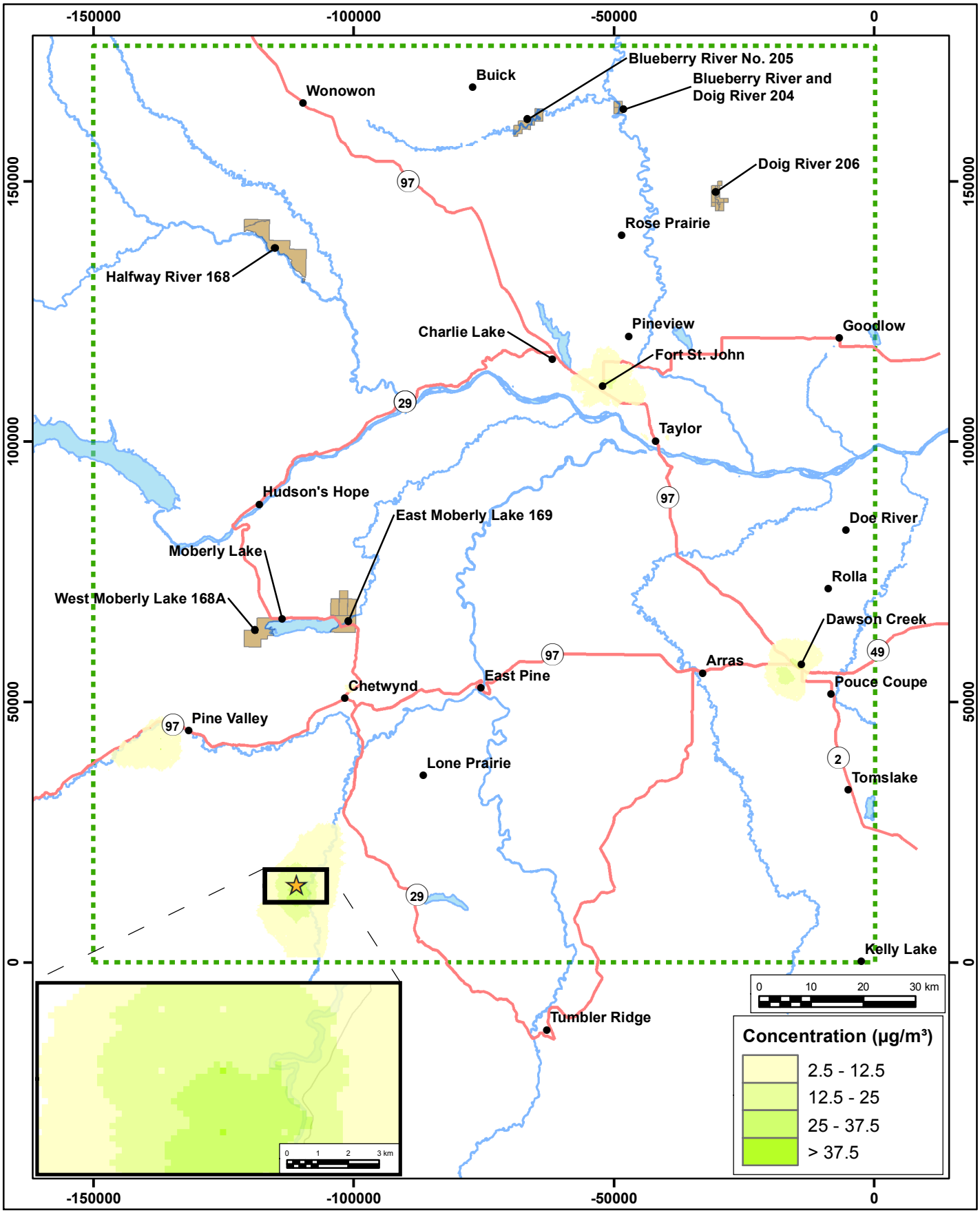
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 6
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - 24hr Averaging Period (98th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

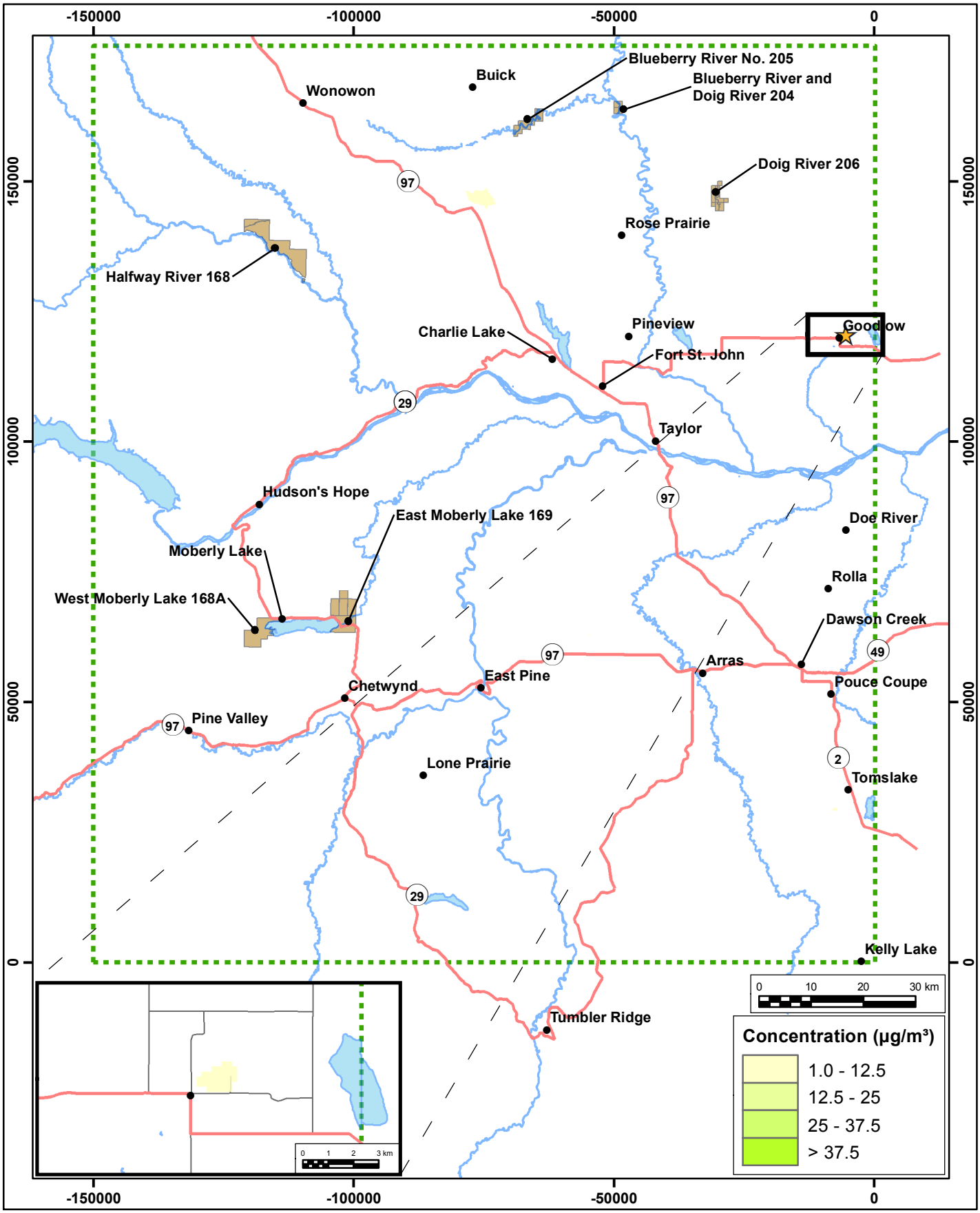
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 7
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - 24hr Averaging Period (98th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

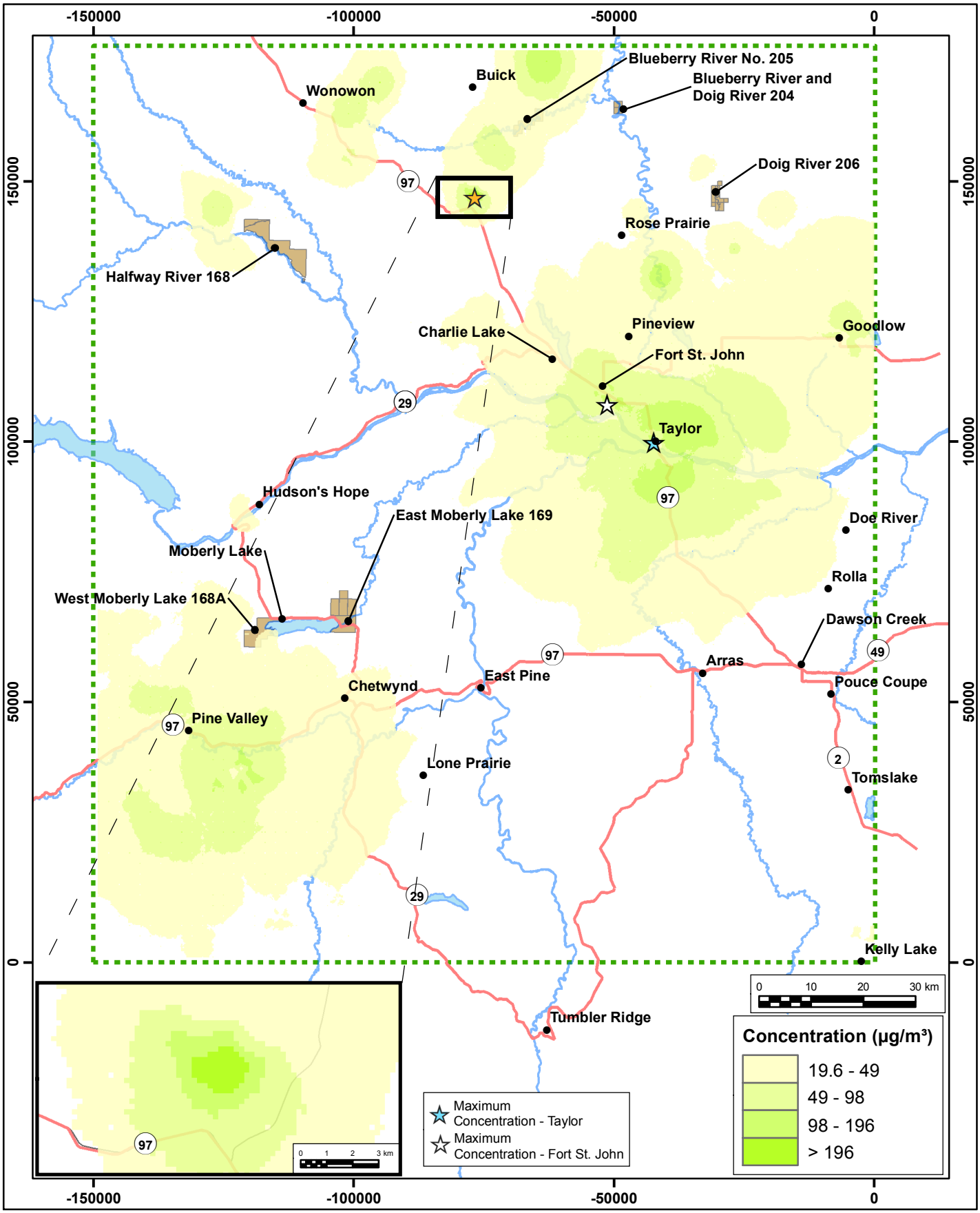
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 8
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



SO_2 - 1hr Averaging Period (99th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

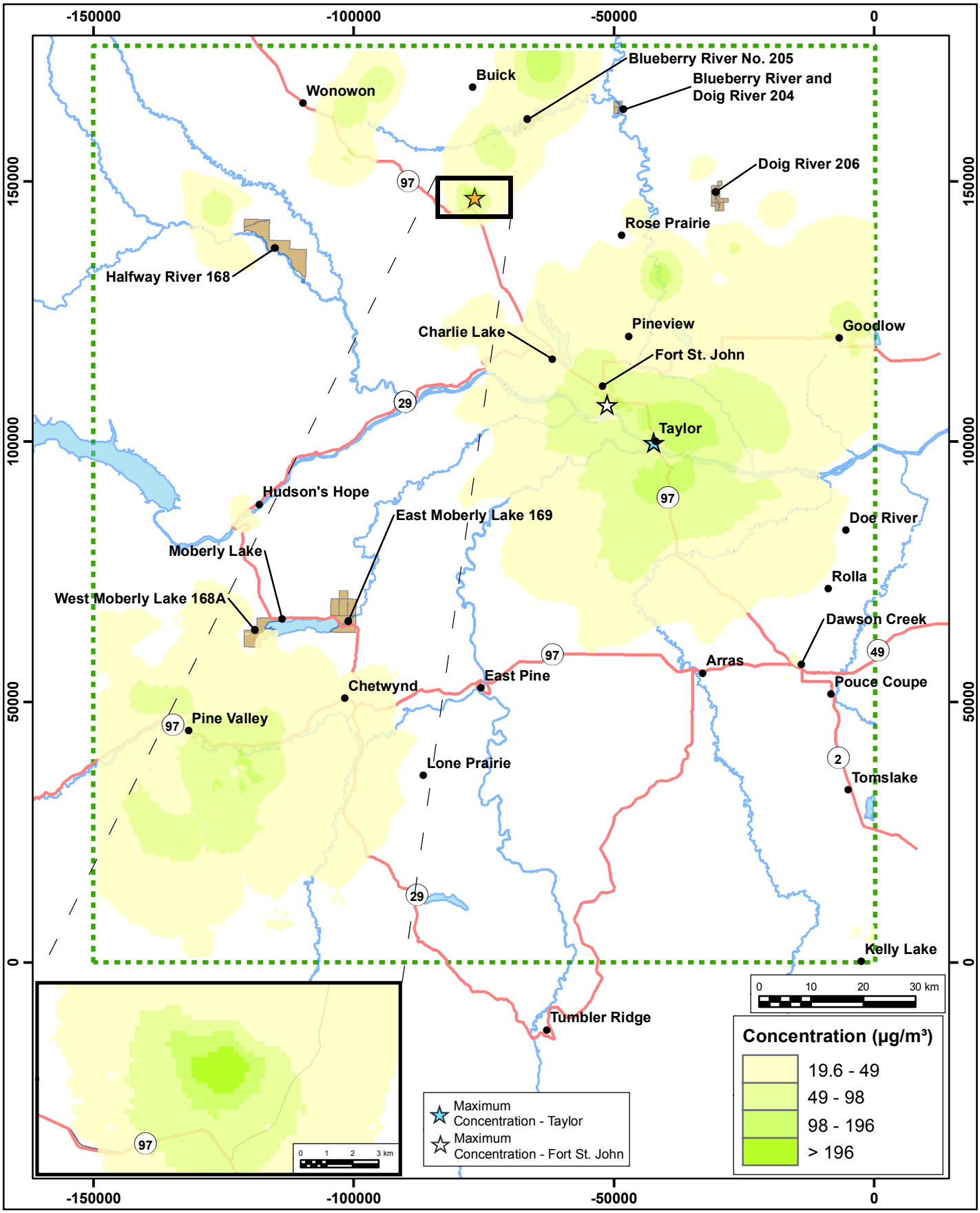
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 9
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



SO_2 - 1hr Averaging Period (99th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

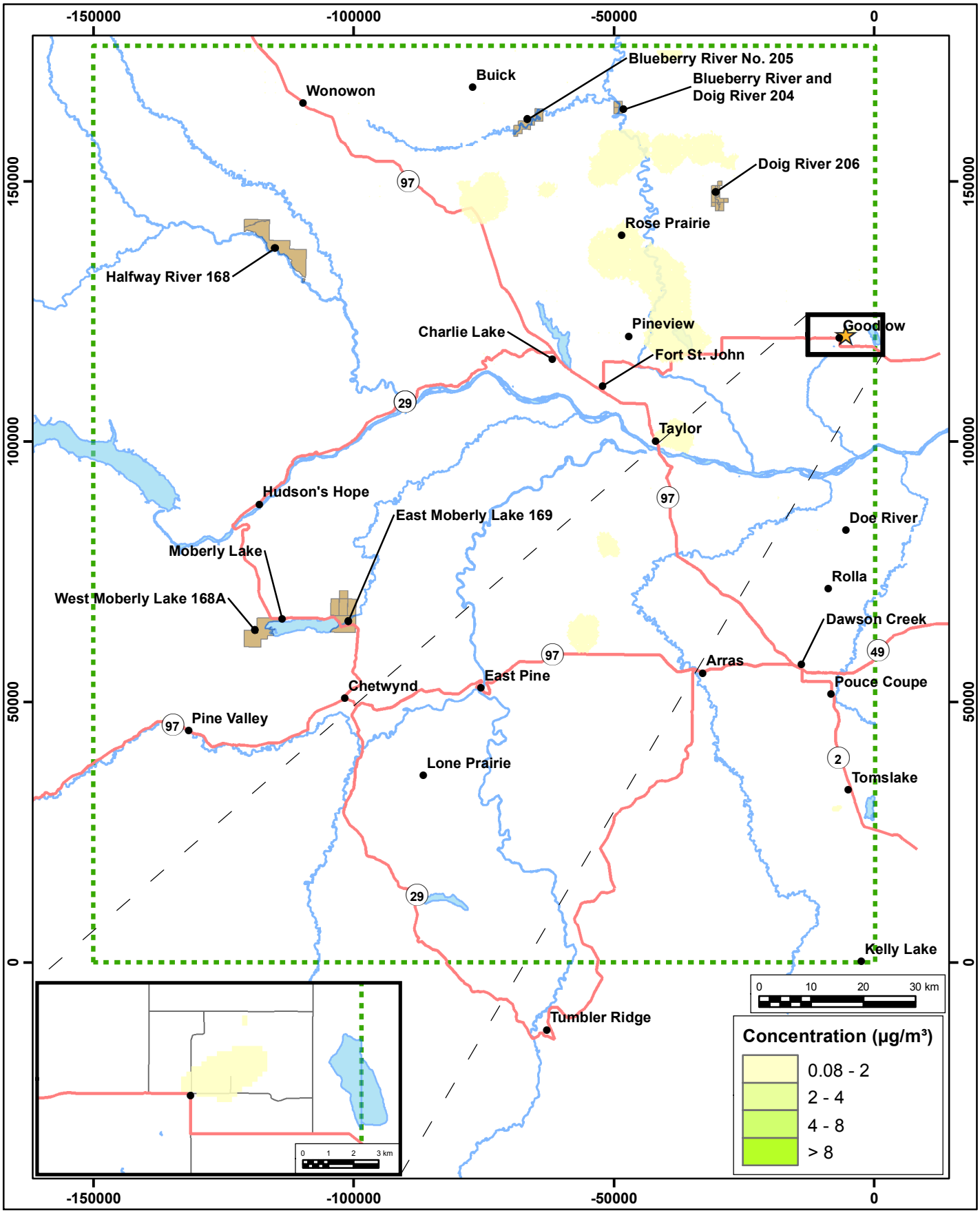
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 10
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - Annual Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

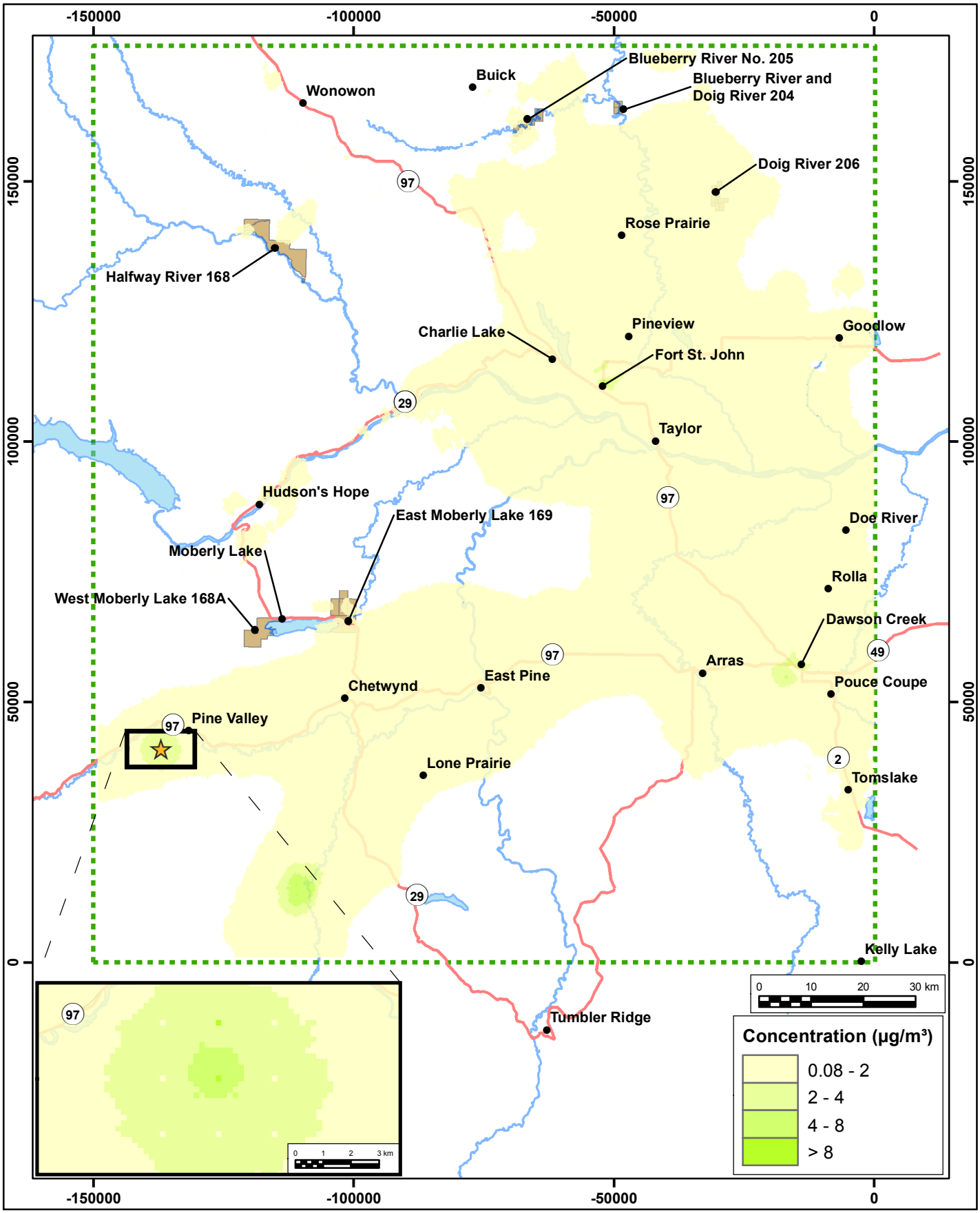
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 11
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - Annual Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

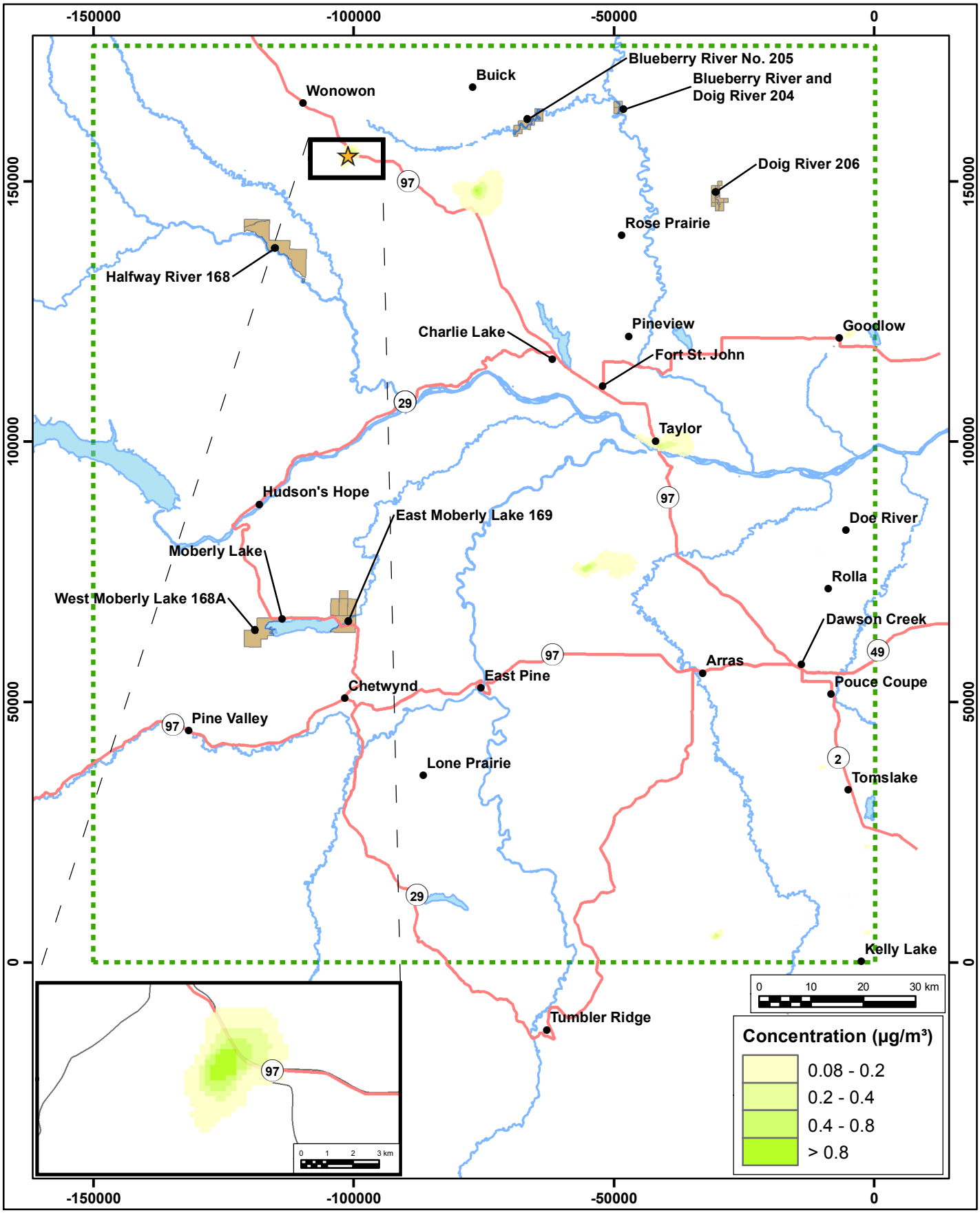
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 12
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - Annual Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Isopleth: 13
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI

Intrinsik Environmental Sciences | 736-8th Avenue SW, Suite 1060 | Calgary, Alberta T2P 1H4 | Tel: (403) 237-0275 | Fax: (403) 237-0291 | intrinsik.com

Appendix A

Tel: 604.730.5688
Fax: 604.730.2915

RWDI AIR Inc.
830 – 999 West Broadway
Vancouver, B.C., Canada
V5Z 1K5
Email: solutions@rwdi.com

Dispersion Modelling in Regards to Oil and Gas Activity in Northeastern British Columbia

Final Report

RWDI # 1202326
August 12, 2014

SUBMITTED BY

David Chadder, Hon. B.Sc., QEP
Senior Project Director/Principal
David.Chadder@rwdi.com

Matthew Sawycky, B.Sc.
Project Manager
Mattew.Sawycky@rwdi.com

Martin Gauthier, M. Sc., ACM
Senior Specialist/Meteorologist
Martin.Gauthier@rwdi.com

Michelle Seguin, Ph.D.
Air Quality Scientist
Michelle.Seguin@rwdi.com

Acknowledgements

The project team would like to acknowledge the additional information and aid provided by multiple people throughout the oil and gas and power generation industries and government agencies. BC Hydro for the use of their Weather Research and Forecasting (WRF) modeling system for the study area, the Canadian Association of Petroleum Producers (CAPP) for organizing and facilitating a meeting with CAPP members who operate in the study area, the cooperation of individual CAPP members for providing the project team with site specific facility site plans and stack parameters, including Canadian Natural Resources Ltd., Encana, ConocoPhillips Canada, Progress Energy Resources Corp., and Shell Canada Limited. Thank you to the individuals from the BC Ministry of the Environment and BC Oil and Gas Commission for assisting in obtaining additional information on stack parameters for other facilities in the area and individuals in Environment Canada for guidance and assistance on their emission calculation tools.

TABLE OF CONTENTS

Tables

Figures

Appendices

Appendix A

Initials and Acronyms

CAC	Criteria Air Contaminants
CAPP	Canadian Association of Petroleum Producers
COPC	Chemicals of Potential Concern
EPA	Environmental Protection Agency
HHRA	Human Health Risk Assessment
GEM-MACH	Global Environmental Multi-scale Modelling Air quality and Chemistry
LCC	Lambert conformal conic
MOE	Ministry of the Environment
MOH	Ministry of Health
MPOI	Maximum Point of Impingement
NE BC	Northeastern British Columbia
NPRI	National Pollutant Release Inventory
NO_X	Oxides of Nitrogen
OGC	Oil & Gas Commission
PAH	Polycyclic Aromatic Hydrocarbon
$PM_{2.5}$	Particulate Matter less than 2.5 micrometers in Diameter
SLRA	Screening Level Risk Assessment
SMOKE	Sparse Matrix Operator Kernel Emissions
SLRA	Screening Level Risk Assessment
SO_2	Sulphur Dioxide
RWDI	RWDI AIR Inc.
VOC	Volatile Organic Compounds
WRF	Weather Research and Forecasting modelling system

1. INTRODUCTION

1.1 Project Overview

The British Columbia Ministry of Health (MOH) commissioned a human health risk assessment focusing on the potential impacts of oil and gas activity on human health in response to concerns raised from residents of northeastern British Columbia (NE BC). The project was segregated into three phases by the MOH including:

- Phase 1: Identification of Health Concerns Relating to Oil and Gas Development in NE BC;
- Phase 2: Human Health Risk Assessment of Oil and Gas Activity in NE BC; and,
- Phase 3: Communication of overall results.

In the Screening Level Risk Assessment (SLRA) component of the Phase 2 HHRA (Intrinsik 2014), Intrinsik Environmental Sciences Inc. (Intrinsik) identified a number of Chemicals of Potential Concern (COPC) for evaluation in the detailed Human Health Risk Assessment (HHRA) with regards to oil and gas activity in NE BC.

As part of the detailed HHRA, RWDI AIR Inc. conducted a comprehensive air quality assessment focusing on emissions from the oil and gas sector in NE BC. The study area was developed as part of the SLRA (Intrinsik 2014), and was based on the area with the highest emission intensities for the criteria air contaminants reported in the National Pollutant Release Inventory (NPRI) and the highest population density in the region.

1.2 Objectives

The two objectives related to the air quality component of the detailed HHRA were to:

1. Develop an emissions inventory for the selected oil and gas sources, as well as other relevant sources in the study area.

2. Complete air quality dispersion modelling, with the aim to providing predicted air concentrations of the selected COPC for the study area. The results of which were used in the detailed HHRA to assess potential health risks that may occur in the region in relation to oil and gas activities.

As part of the first objective an emission inventory was built to account for all air emissions sources within the study area.

The air quality was characterised in a cumulative effects assessment that considered emissions from oil and gas activities and other non-oil and gas emission sources such as residential, commercial, other industries, etc. Two scenarios were considered:

1. The first scenario (Oil and Gas Scenario) focused on all emissions from continuous oil and gas activities (as selected in the SLRA) that were released directly to air within the study area. These

sources included significant emitters such as: sweet and sour gas plants, compressor stations, fugitive emissions from tank storage, and flaring.

2. The second scenario (Cumulative Scenario) included emissions from the first scenario as well as emissions from non-oil and gas sources such as transportation and agriculture, and community activities (e.g. residential wood burning and off road transport). Other industrial sectors, including pulp and paper, forestry and mining, were also represented in this scenario.

Maximum predicted concentrations in the study area along with maximum predicted concentrations in each of the communities in the region were provided to Intrinsik for both scenarios for further analysis on human health effects.

1.3 Chemicals of Potential Concern (COPC)

A 'short-list' of chemicals or contaminants of potential concern (COPC) was identified by Intrinsik in the SLRA (Intrinsik 2014) that are known to be emitted from oil and gas facilities. These chemicals are known to be of particular concern to human health, or are known to be associated with certain health endpoints. This list not only included criteria air contaminants (CACs) such as NO_2 and SO_2 but also a number of volatile organic compounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs).

Table 1: List of COPC modelled, and their Associated Species ID

Species ID	Species Name
so_2	sulphur dioxide
no_2	nitrogen dioxide
pm_25	particulate matter 2.5 microns or less
h2_s	hydrogen sulphide
vo_c	total volatile organic compound
Acet	Acetaldehyde
Acro	Acrolein
Ben	Benzene
bap	Benzo(a)pyrene
b13	Butadiene, 1,3 -
cum	Cumene
cyc	Cyclohexane
eb	Ethylbenzene
form	Formaldehyde
hex	Hexane
naph	Naphthalene
pen	Pentane
tol	Toluene
tri	Trimethylbenzene
xyl	Xylenes
7_12	7,12-dimethylbenz(a)anthracene
dah	dibenz(a,h)anthracene
baa	benzo(a)anthracene
bbf	benzo(b)fluoranthene
bkf	benzo(k)fluoranthene

Species ID	Species Name
chry	chrysene
fluo	fluoranthene
ind	indeno(1,2,3,c,d)pyrene
pheno	phenanthrene

1.4 Communities in the Study Area

The study area for the air quality assessment extends from just north of Tumbler Ridge from the south to approximately 30 km north of Wonowon. The west boundary of the study area starts approximately 40 km west of Hudson Hope and finishes at the Alberta/BC border. The major communities in the study area include Fort St. John, Dawson Creek and Chetwynd, along with smaller communities and First Nation lands as displayed in Figure 1.

2. DEVELOPMENT OF EMISSION INVENTORY

2.1 Source of Emission Data

An emission inventory was developed to account for all sources of interest within the defined study area. Emission inventories typical separate air emissions into three source categories: point, area and mobile. Industrial facilities that operate under air discharge permits are usually expressed as point sources as they typically have a visible stack. Area sources usually represent smaller, more broadly distributed light industrial, commercial, institutional, residential and naturally occurring sources that do not require air discharge permits. In some circumstances industrial emissions such as fugitive or storage emissions can also be represented by area sources. Mobile sources can include on-road vehicles, non-road equipment, railways, aircraft, and marine vessels.

Emissions of COPC were estimated for each of the following categories:

- **Oil and gas facilities reporting to NPRI** – This category represented the major oil and gas facilities in the study area, and included gas plants (both sweet and sour). These facilities had at least one COPC that was reported to NPRI in 2010. Additional information for these facilities was provided by Canadian Associated of Petroleum Producers (CAPP) members, BC Ministry of Environment (MOE) and the BC Oil and Gas Commission (OGC). These sources were represented as point sources and small area sources.
- **Other industrial sources reporting to NPRI** – Mining and forestry facilities report emissions to NPRI for many of the COPC. Emissions were represented as point and/or small area sources. The mining industry contributed to particulate matter less than 2.5 µm in equivalent diameter ($PM_{2.5}$), while the pulp and paper industry contributed to a number of VOCs in the study area.
- **Upstream oil and gas inventory** – Included emissions from flares, diesel engines, propane engines, dehydrators, tanks, loading and fugitive emissions associated with the upstream oil and gas industrial activities. The 2010 Environment Canada small upstream oil and gas facilities inventory (EC, January 21, 2014) was converted to area sources.

- **Mobile OnRoad sources** – Represented sources refer to emissions associated with transportation on roads. Accordingly, emissions from mobile sources were distributed spatially using geospatial information about the locations of roadways. The Global Environmental Multi-scale Modelling Air Quality and Chemistry 2006 Environment Canada (GEM-MACH) Inventory (EC, December 7, 2013) was used to determine emissions which were converted to area sources.
- **Non Road sources** – Represented sources refer to a broad group of sources such as residential heating, industrial activities not reported in NPRI, railway transportation, agricultural activities, etc. Emissions from those sources were distributed spatially using geospatial information (population, dwelling, etc.). The 2006 GEM-MACH Inventory was used to determine emissions which were then converted to area sources.

Additional information regarding how the emission inventories were developed for the Oil and Gas and Cumulative scenarios from each of the above source categories is provided in Sections 2.2 to 2.4.

2.2 NPRI 2010 Inventory

The NPRI is a publicly accessible inventory of annual pollutant releases. It is a key resource for supporting assessment and risk management of chemicals and air quality modelling. The NPRI includes information reported by facilities, and is published by Environment Canada based on the Canadian Environmental Protection Act of 1999 (NPRI, 2014). Each year, approximately 8,000 facilities report to the NPRI with regards to their environmental releases of all types. In the NE BC study area, approximately 200 facilities reported atmospheric releases of COPC to the NPRI. Over 90% of these facilities were identified to be with the oil and gas sector.

It was noted that although Environment Canada implements a number of data quality measures to ensure accuracy of the data sets, a number of locations of facilities did not always match up with locations determined by satellite images or provided by the companies. A list of these facilities is presented in Table 2. Besides the facilities, all other locations provided by NPRI were assumed to be accurate and used for modelling inputs.

The NPRI 2010 reported emissions were selected for use in the emissions inventory over more recently reported NPRI emissions for years 2011 and 2012 for multiple reasons. When the emission inventory was being built in Q4 2013, only preliminary data was available for NPRI reporting year 2012. Since the preliminary 2012 data had not been reviewed and quality controlled by Environment Canada at the time of building the emission inventory, earlier NPRI reporting years were considered to construct the inventory. NPRI reporting year 2010 was selected over year 2011 data for the following reasons:

- Informal guidance from Environment Canada indicated that some quality assurance issues were still being addressed for some of NPRI 2011 records and NPRI year 2010 was recommended, being fully reviewed and confirmed;
- The NPRI 2010 emission inventory would coincide with the available 2010 Upstream Oil and Gas Emission Inventory allowing for direct comparison between the two inventories (this aided in identifying facilities that were considered in both inventories); and,

- Phase 1 of the SLRA considered NPRI 2010 data. To stay consistent with the reported emission intensities and other results in the SLRA, NPRI 2010 was used for the more detailed analysis.

As a check on the variability of the annual reported emissions over the 2010 to 2012 period, total annual oxides of nitrogen (NO_x), sulphur dioxide (SO_2) and total VOC emissions reported to NPRI from all industrial sector point sources in the study area are summarized in Table 2. Relative to the modeled year 2010, the NO_x and SO_2 values for year 2012 are about 5% lower and 7% higher, which are relatively small changes and not expected to significantly influence predicted concentrations overall. The reported total VOC's from point sources were about 37% lower in 2012 relative to year 2010. For natural gas powered reciprocating engines commonly used in the oil and gas sector, Environment Canada moved to a much lower set of emission factors in 2011 and that is expected to help explain the lower reported total VOC emissions. Individual facility emissions are expected to vary as well and not uniformly (i.e., in the same direction).

Table 2: Total point source atmospheric emissions reported to NPRI for years 2010 to 2012 (in tonnes)

NPRI Reporting Year	NO_x Emissions	SO_2 Emissions	Total VOC's
2010	18,464	17,732	5087
2011	15,526	20,417	3116
2012	17,546	18,906	3210

Emissions from NPRI sources were assumed to run continuously throughout the year and throughout the day. Emission rates were determined by taking the value reported to the NPRI for each facility in year 2010 and evenly distributing the emissions over the year. The only exceptions were three facilities (i.e., NPRI #7720, #7959 and #4305) which are located in the same area and operated by the same owner. After a conversation with the operator of those facilities, emissions released from the NPRI 7720 facility was relocated as outlined in Table 3, and, because of an update in the NPRI calculation tool for estimating the release rates of some speciated VOCs between 2010 and 2012, it was found that the NPRI 2012 emission rates were more reliable, and therefore, were used for those three facilities.

Table 3: List of facilities that had modelled locations that differed from NPRI reported locations

NPRI Number	Facility	NPRI Location		Location Modelled		Details
		Latitude	Longitude	Latitude	Longitude	
4305	Spectra Energy Transmission McMahon Gas Plant	56.1508	-120.6679	56.1447	-120.6686	NPRI location was determined to be the location of the AltaGas Ltd - Younger NGL Extraction Plant. For modelling purposes, this location was assumed to be co-located with NPRI 7959 that has a similar name as the facility.
5124	Spectra Energy Highway Gas Plant	56.2278	-120.8190	-	-	NPRI Location is in Fort St. John. Address of the plant does not match with NPRI location given and address is located just north of the modelling domain. As such, this facility was not modelled.

NPRI Number	Facility	NPRI Location		Location Modelled		Details
		Latitude	Longitude	Latitude	Longitude	
7720	Spectra Energy Transmission Station 1 - Taylor	56.1417	-120.6778	56.1447	-120.6686	Based on communications with Spectra Energy, it was determined that atmospheric emissions associated with this facility are located across the highway beside their other facility (NPRI 7959). Emissions are assumed to be co-located with NPRI 7959.
7732	Spectra Energy Transmission CS NO. A1, Gordondale	55.7608	-120.2357	-	-	NPRI Location is Downtown Dawson Creek. Gordondale is located in Alberta. Facility was not modelled
7963	Spectra Energy Transmission BS 9. Nig Creek	56.2478	-120.8470	-	-	NPRI location is in Fort St. John residential area. Nig Creek is north of the modelling domain. Facility was not modelled
7965	Spectra Energy Transmission Booster Station 11 - Rigel	56.2362	-120.8550	-	-	NPRI location was found to be in Fort St. John and did not match with satellite images of the area. Since it was only a very minor source of NO_x it was not modeled
18126	AltaGas Ltd. Younger NGL Extraction Plant	56.1552	-120.3488	56.1508	-120.6679	Facility web page reports location of the plant that differs compared to the NPRI location and agrees with satellite images. Location from AltaGas web page was used as the modelled location.
19386	Conoco Philips Canada Resources Corp. Halfmoon Comp Station C-029-B	55.7667	-120.2333	-	-	NPRI location was in residential area of Dawson Creek and does not match with satellite images of the area. This facility was determined to be located outside the modelling domain and source was not modelled.
19733	Suncor Energy Inc. Suncor Kobes Battery (B-24-A/94-B-9)	56.2478	-120.8470	56.5188	122.0590	NPRI Location was in Downtown Fort St. John. Modelled Location determined from the Registration Report for Oil and Gas Production Facilities and Equipment of the facility.

The NPRI 2010 emissions were summarized in Table A1 for all sources in the modelling domain. The NPRI 2010 inventory is shown as three groups: the NPRI Upstream Oil and Gas, the NPRI Pulp and Paper/Forestry Industry and the NPRI Coal and Mining. The NPRI 2010 Upstream Oil and Gas Inventory represented most of the SO_2 emissions modelled in the study area and almost half of the NO_X emissions; whereas, the NPRI 2010 Coal and Mining sources represented almost 58% of the total $PM_{2.5}$ emissions in the modelling domain for the Cumulative Scenario.

Reported NPRI emissions were categorized into five different types: stack, storage and handling, fugitives, other, and speciated VOC sources. Oil and gas sources were further divided into two categories: stack (see Section 2.2.1) and area sources; which included all other subcategories reported to NPRI (i.e., storage and handling, fugitives, other, and speciated VOCs). A flow chart of how emission sources were

considered is shown in Figure 2. Similarly NPRI sources that were not oil and gas (i.e., from the pulp and paper, forestry or mining industries) were also split into stack and area sources.

2.2.1 Oil and Gas NPRI: Determining Stack Parameters

Stack parameters for stacks greater than 50 m in height must be reported to the NPRI. Stack parameters include stack height, stack diameter, exit velocity and exit temperature. Where available, reported stack emissions were modelled as point sources using the NPRI reported stack parameters. Nine facilities in the study area reported stack parameters to NPRI and accounted for 85% of the SO_2 and 52% of the total VOC emission sources reported in the study area in 2010. For facilities with multiple stacks greater than 50 m in height, stack emissions were assumed to be proportional to exhaust gas flow rate. Pseudo - parameters including pseudo-diameter and height were calculated and velocity and exit temperatures were set to 20 m/s and 1000ºC, respectively, for point sources identified as flares, following direction from *BC Guideline for Air Quality Dispersion Modelling* (2008).

Facilities with no stack parameters reported by the NPRI were divided into two groups: large emitting facilities and other facilities. The large emitters consisted of 15 facilities that made up approximately 7.8% of the reported 2010 NPRI emissions for SO_2, 2.9% for total VOCs, and 34% for NO_X. Since these emitters made up the majority of the emissions, detailed information from these plants were required. With assistance from CAPP, individual facility operators for these larger gas plants were approached with respect to site specific information. Additionally, BC OGC, BC MOE, and the facility approvals aided in determining the majority of the stack parameters for the large oil and gas emitters. Additional stack parameters were calculated and/or estimated based on facilities and/or equipment of similar size. For the 15 large emitters, 93 individual sources were modelled. These individual sources included: continuous flares, compressors, heaters, and boilers. Total stack emissions for each facility were taken to be equivalent to the NPRI stack emissions reported in 2010. The total emissions for each facility were divided among the facilities' individual sources based on ratios of permitted maximum releases or exhaust gas flow rates, if available, and/or assuming emissions were divided evenly between different sources. If a source contributed less than 1% of the facility's emissions, it was not included as a point source and its emissions were spread throughout the other area sources at the facility. Emergency and backup equipment were assumed to be not running for the majority of the time and, were therefore, not included in the emission inventory. The location of the first compressor at a facility was assumed to be at the reported NPRI latitude and longitude and other sources at the site were determined based on the distance from the first compressor using site maps or Google Earth, if available.

Smaller oil and gas stack sources that reported to NPRI were considered as single point sources. It was noted that most of these facilities were compressor stations scattered throughout the study area. Although many of these facilities have multiple sources of atmospheric emissions, the majority of COPC at a compressor station are usually emitted from the compressor itself. Based on experience and professional judgement, typical stack parameters were assigned to these point sources as follows:

- Stack height - 10 m;
- Stack diameter - 0.5 m;

- Exit velocity - 7.1 m/s; and,
- Exhaust temperature - 733 K.

Note that although generic stack parameters were modelled, both location and emission rates of the COPC were taken directly from the NPRI emission inventory.

2.2.2 Oil and Gas NPRI: Determining Area Parameters

The NPRI divides emissions into stack sources (discussed in Section 2.2.1), storage, fugitive and other. Additionally there is a complementary NPRI emission inventory that reports speciated VOC emissions (NPRI 2014). Storage, fugitive and 'other' were considered to be area sources and were grouped together. All speciated VOC emissions in the study area were reported as 'ground' emissions which were defined as emissions from stacks less than 50 m. Although these emissions could potentially be emitted from lower stacks, it was assumed that the speciated VOC emissions were also an area source and were included in that source group.

Oil and gas area sources were divided into two groups based on the amount of emissions: larger NPRI area sources and smaller NPRI area sources. The combined larger NPRI sources released greater than 85% of the NPRI area source emissions. The perimeter of the area source was based on the foot print of the facility determined by satellite images. Any of the larger area sources that were classified as gas plants were assumed to have a release height of 30 m, to be considerate of the height of buildings and the many minor stacks associated with these plants. Sources not classified as gas plants were assumed to have a release height of 10 m, which would be consistent with a generic height of a storage tank. A sigma-z value, a measurement of the initial vertical dispersion, was calculated based on the height of the area source divided by 2.15 which has been recommended by US EPA (EPA, 1995).

For the smaller NPRI area sources, a generic 100 m x 100 m area source, centered on the location reported to the NPRI was considered. The release height was assumed to be 10 m with sigma-z of 4.65 m as recommended by US EPA (EPA, 1995). Area sources were assumed to be continuous throughout the year and time of the day.

2.2.3 NPRI Facilities: Non-Oil and Gas

Seven pulp and paper and forestry facilities reported stack emissions to the NPRI in the NE BC study area. Stack parameters were determined similarly to the oil and gas facilities. Emission rates were assumed to be consistent throughout the year and time of day. Stack parameters were based on information from BC MOE permits and/or estimated based on facilities and/or equipment of similar size.

Area sources were considered for three pulp and paper facilities and two mines in the area. For mines, emissions from stacks were included in the area sources since stack sources were relatively minor relative to the area source produced by the mine. Area sources for mines were determined based on their foot print from satellite images. In most cases, area sources for mines would be much more localized than the whole footprint of the mine, but without additional information this conservative estimate was assumed. Height of the area source was considered to be 30 m for mines. A release height of 10 m

was considered for pulp and paper sources. A sigma-z value was assumed to be equivalent to the height divided by 2.15 (EPA, 1995).

2.3 2010 Environment Canada Small Upstream Oil and Gas Inventory

The 2010 Environment Canada Small Upstream Oil and Gas Inventory (EC, January 21, 2014) contained total annual emissions of small and temporary upstream oil and gas facilities such as well drilling sites and batteries that may not have needed permits through the BC MOE. The small upstream oil and gas inventory provided annual emissions of NO_X, SO_2, $PM_{2.5}$ and total VOCs for the following sub-categories: diesel combustion, flare, dehydrator, leak, load, tank, and vent. Hydrogen sulphide (H_2S) emissions from flares were conservatively estimated to be 2% of the released SO_2. Latitude and longitude coordinates for each point source were provided. In total, 6,034 point sources from 3,817 oil and gas facilities were found to be located in the study area. It was determined that the number of point sources was too high to be directly inputted into the dispersion model. Instead, the emissions were modelled as area sources with a release height ranging from 5 to 10 m and an initial sigma-z of 2 m. The SMOKE Modelling System was used to aggregate the point emissions into area sources. The SMOKE tool is described below in Section 2.4.1. A summary of emissions from the 2010 Environment Canada Small Upstream Oil and Gas Inventory are shown in Table A-1. This inventory represents approximately 43% of the total VOCs in the modelling domain.

2.4 2006 GEM-MACH Environment Canada mobile, non-road, agricultural and other Inventory

The 2006 GEM-MACH mobile, non-road, agricultural and other inventory (EC, December 7, 2013) contained area and mobile total annual emissions of NO_X, SO_2, $PM_{2.5}$ and total VOCs. This inventory was also provided with geospatial information to be able to spatially allocate the emissions into area sources in the modelling domain. The SMOKE modelling system was used to prepare the emissions for input to the dispersion model. The SMOKE tool is described below. Emissions from the 2006 GEM-MACH Inventory are shown in Table A1 as two groups: EC mobile On Road and EC Non Road. The EC mobile On Road represents approximately 13% of the total NO_X emissions in the model domain; whereas, the EC Non Road represents 38% of the total VOCs in the modelling domain.

2.4.1 Emission processing with SMOKE

Emissions from anthropogenic activities are typically compiled as spatially, temporally, and chemically lumped emissions in spreadsheets, databases, or other model-specific file formats.

To prepare the emissions data into a suitable format for the dispersion model, it was first necessary to:

- Spatially allocate emissions from their native formats into individual model grid cells using GIS-based activity data;
- Temporally allocate annual emissions into an hour by hour format using look-up tables that describe typical activity profiles for different emission sources (e.g., industrial facilities, on-road traffic, etc.); and,

- Chemically speciate lumped or aggregated pollutant groups (e.g., total volatile organics or VOCs) into individual chemical species and species groups based on their reactivity rates and other photochemical properties, the specifics of which are dependent upon what options with the chemistry-transport model were selected.

These pre-processing steps were performed using Version 3.1 of the SMOKE processing system. SMOKE uses source classification codes (SCC) tagged to each entry of the emission inventory to do the spatial and temporal allocation as well as chemical speciation. The final outputs of the SMOKE model are temporally averaged hourly emissions per grid cells (4 km by 4 km) for each of compounds in the inventory. Those steps are discussed below.

2.4.2 Spatial allocation of emissions in SMOKE

Spatial surrogate files were used by the SMOKE model to spatially allocate aggregated emissions data to model grid cells during the emission inventory process. Emissions are aggregated at a provincial or sub-provincial level, depending on the emission type and source. The spatial surrogate files are composed of values that represent the percentage of those total aggregated emissions that are expected to be emitted within each modelled grid cell.

In order to compute the values in the spatial surrogate files, GIS software and GIS-format datasets that have geometry and attributes appropriate to represent the activity associated with particular emission sources were utilized. For example, for emissions that are associated with population, census datasets with population counts were used to calculate the expected population in each model cell by comparing the census areas and population counts to the model cells that intersect/overlap with it. Further, emissions associated with on-road sources would leverage road network GIS-format datasets to allocate those emissions accordingly. For this project, spatial surrogate files for population, dwellings, roads, mining, railways, livestock and fertilizer were generated and applied in SMOKE to emissions that are associated with these activities. See Figure 3 for the spatial surrogate file for the allocation of population as an example.

2.4.3 Temporal allocation of emissions in SMOKE

As mentioned before, each entry in the 2006 GEM-MACH Inventory and the 2010 small Upstream Oil and Gas Inventory is tagged with a source classification code (SCC) that allows SMOKE to apply a monthly, weekly and daily profile to annual emissions. However, running SMOKE for an entire year of simulation was time and computational heavy and was found to be unnecessary. Instead, the model was run for a typical weekday (June 21, 2013) and the emissions were temporally averaged to calculate hourly averaged emissions. This method assumes no variation in emissions over the course of the year, the week and the day, which is generally true for the upstream Oil and Gas emissions but not necessarily for some of the sources present in the 2006 GEM-MACH Environment Canada Inventory. For example, the OnRoad emissions have an important daily profile; whereas, the fuel wood combustion emissions vary over the course of the year. Therefore, for these two source groups, specific temporal profiles were used in the dispersion model.

2.4.4 Speciation of the VOC emissions in SMOKE

Each entry in the 2006 GEM-MACH Inventory and the 2010 Small Upstream Oil and Gas Inventory has a SCC that allows SMOKE to speciate VOCs. However, SMOKE carries out the speciation of VOCs based on grouped species and their reactivity rates and other photochemical properties which makes it difficult to use directly in the selected dispersion model. Therefore, SMOKE was not used to do the speciation of VOCs but the SMOKE look-up tables linking SCC to a speciation profile were utilized instead to speciate the total VOC concentrations modelled by the dispersion model. This was made possible by grouping the VOC emissions into categories sharing the same speciation profiles (scaling factors to be applied to the VOC concentrations). Table 4 shows the VOC lumped groups as well as their associated speciation profiles. For example, it can be seen that the benzene represents 3.36% of the On Road VOC exhaust emissions.

The fractions or profiles shown in Table 3 were used to build all the VOC species shown in Table A1. These fractions were based on SMOKE default look-up tables linking SCC to a certain speciation profile with the following exception: The Upstream Oil and Gas 'Tank' category was originally linked to the profile 'petroleum storage' profile 2489. This was assigned to a more specific profile number 0296 called 'Fixed roof tank – Crude Oil production'. The benzene fraction of 20.7% in the profile number 2489 was found to be non-representative of the benzene fraction that is present in tank containing crude oil so profile number 0296 was used for speciation with the benzene fraction of approximately 0.1%.

The SMOKE default look-up tables only contained a few of the PAHs that were flagged as COPC. Therefore, wherever possible, emissions for other PAHs were estimated based on other sources of information. The additional speciation of PAHs is discussed in detail in Section 2.6.

Table 4: VOC Speciation Profiles

Emission type :		NonRoad Groups						Mobile OnRoad Groups		Upstream Oil and Gas						
Source Group :		General	Petroleum storage	Solvents and Coating Related Products	Aerosol Coating	Wood combustion	Non-oxygenated Gasoline Headspace Vapor	Evaporative	Exhaust	Dehydrator	Diesel	Flare	Leak	Load	Tank	Vent
Profile Number:		0000	2489	3144	3149	4642	8737	8753	8751	1011	4674	1011	1012	0297	0296	1011
Species ID	Species Name															
Acet	Acetaldehyde	8.40E-03	na	na	na	1.24E-01	na	na	1.52E-02	na	1.59E-01	na	na	na	na	na
Acro	Acrolein	7.70E-03	na	na	na	4.58E-03	na	na	7.67E-04	na	1.30E-02	na	na	na	na	na
Ben	Benzene	2.82E-02	2.07E-01	na	na	2.79E-02	1.08E-02	3.36E-02	5.29E-02	1.00E-03	1.05E-02	1.00E-03	1.00E-03	2.40E-02	1.00E-03	1.00E-03
bap	Benzo(a)pyrene	na	na	na	na	na	na	na	na	na	na	1.03E-05 [4]	na	na	na	na
b13	Butadiene, 1,3 -	6.70E-03	na	na	na	8.51E-03	2.01E-05	2.96E-03	6.24E-03	na	1.18E-03	na	na	na	na	na
cum	Cumene	3.80E-03	9.00E-04	na	1.42E-04	na	4.19E-04	1.36E-05	1.72E-03	na	0.00E+00	na	na	na	na	na
cyc	Cyclohexane	9.00E-03	4.60E-03	4.70E-04	9.94E-04	na	2.06E-03	5.48E-04	2.60E-03	na	8.01E-04	na	na	na	na	na
eb	Ethylbenzene	6.50E-03	1.41E-02	1.41E-03	2.15E-02	1.67E-03	2.47E-03	2.56E-02	2.32E-02	na	1.79E-03	na	na	na	na	na
form	Formaldehyde	1.55E-02	na	na	na	8.47E-02	0.00E+00	0.00E+00	1.63E-02	na	8.51E-02	na	na	na	na	na
hex	Hexane	1.37E-02	6.21E-02	7.98E-03	4.55E-03	na	1.52E-02	2.25E-02	1.91E-02	na	0.00E+00	na	na	4.70E-02	7.90E-02	na
naph	Naphthalene	1.80E-03	na	na	2.84E-04	1.65E-02	2.44E-04	3.05E-04	4.58E-04	na	2.35E-03	1.20E-03 [4]	na	na	na	na
pen	Pentane	7.10E-03	2.54E-02	na	na	3.42E-04	6.48E-02	6.24E-02	9.34E-04	na	7.09E-03	na	na	1.01E-01	1.46E-01	na
tol	Toluene	2.04E-02	4.11E-02	7.87E-02	1.58E-01	1.15E-02	2.63E-02	9.78E-02	1.04E-01	na	1.52E-02	1.94E-04 [4]	na	1.40E-02	na	na
tri	Trimethylbenzene	4.00E-04	1.10E-02	3.13E-04	5.40E-03	na	3.83E-03	1.86E-02	2.75E-02	na	4.35E-03	na	na	na	na	na
xyl	Xylenes	7.00E-03	na	5.95E-02	1.38E-01	na	na	na	na	na	0.00E+00	1.03E-04 [4]	na	na	na	na
7_12	7,12-dimethylbenzene(a)anthracene	na	na	na	na	na	na	na	na	na	0.00E+00	na	na	na	na	na
dah	dibenz(a,h)anthracene	na	na	na	na	na	na	na	0.00E+00 [2]	na	0.00E+00	na	na	na	na	na
baa	benzo(a)anthracene	na	na	na	na	2.95E-05 [3]	na	na	4.45E-05 [2]	na	1.14E-05	na	na	na	na	na
bbf	benzo(b)fluoranthene	na	na	na	na	9.47E-06 [3]	na	na	0.00E+00 [2]	na	2.63E-06 [1]	na	na	na	na	na
bkf	benzo(k)fluoranthene	na	na	na	na	1.06E-05 [3]	na	na	0.00E+00 [2]	na	4.12E-06 [1]	na	na	na	na	na
chry	chrysene	1.00E-04	na	na	na	3.96E-05 [3]	na	na	2.35E-05 [2]	na	9.38E-06 [1]	4.12E-05 [4]	na	na	na	na
fluo	fluoranthene	1.00E-04	na	na	na	2.22E-04	na	na	6.11E-04 [2]	na	2.02E-04	2.16E-04 [4]	na	na	na	na
ind	indeno(1,2,3,c,d)pyrene	na	na	na	na	2.23E-06 [3]	na	na	0.00E+00 [2]	na	9.96E-06 [1]	na	na	na	na	na
pheno	phenanthrene	6.00E-04	na	na	na	1.14E-03	na	na	1.94E-03 [2]	na	3.55E-04	5.26E-04 [4]	na	na	na	na

Notes :

[1] Speciation of PAH was based on Speciate4.2 profile 4674 and USEPA AP42 Table 3.3-2.
[2] Speciation of PAH was based on Speciate 4.2 profile 8751 and MOVES2010b: Additional Toxics Added to MOVES, EPA-420-B-029a, May 2012
[3] Speciation of PAH was based on the study of Hytone et all, 2009
[4] Speciation of some VOC and PAH was based on the study of Strosher. 1996
na Non applicable, assumed to be negligible

2.5 Speciation of the NPRI VOC emissions

Facilities are required to report individual VOC species to the NPRI when greater than 10 tonnes of total VOCs are reported, and the individual VOCs emission is greater than 1 tonne for a given year. For many of the facilities, individual VOC species were not reported to the NPRI and only total VOCs were reported. To determine individual COPC emission rates, a speciation profile provided by Environment Canada (EC, December 2013) was used to determine individual VOC release for each of the individual facilities based on the total VOC emission rate. All but eight of the facilities that reported to the NPRI in 2010 had a speciation file in the Environment Canada speciation profile. The facilities that did not have a speciation profile had low emissions of VOCs, and individual VOCs for these emitters were considered to be negligible. All COPC outlined by Intrinsik were reported to these speciated profiles except ethyl benzene and PAHs. Speciation of PAHs, and when possible ethyl benzene, was carried out as described in Section 2.6.

2.6 Speciation of the PAHs

As discussed in Section 2.4.4, speciation of individual PAHs were not considered since many of the SCC for the SMOKE model do not include PAHs in their profile. Additionally, PAHs are not reportable to NPRI unless total emissions are greater than 50 kg for the facility (NPRI 2014). Since many of the PAHs are of interested to human health, and were identified as COPC in the SLRA (Intrinsik 2014) estimates of PAH emissions were determined based on a literature review of possible PAH emissions released during operating conditions of different types of Oil and Gas facilities and background sources. PAH emissions from diesel combustion were based on US EPA AP-42 Table 3.3-2 (EPA 1996). Ratios between PAHs to an already speciated VOC were assumed to be similar to what was reported by the US EPA. This PAH profile was also used for NPRI sources that were considered to be boilers, heaters or compressors, since many of these types of sources would be run on a diesel engine. PAH emissions from mobile OnRoad groups exhaust were extracted from EPA-42-B-029A (EPA, 2012).

PAH speciation for flares, both from the small Oil and Gas inventory (Section 2.3) and those identified by the NPRI inventory (Section 2.2) was determined from measurements conducted by Strosher (1996) looking at sour gas flares and speciated VOCs and PAHs in Alberta. Flares in the study area were assumed to have the same benzene to PAH ratio as the study conducted in Alberta (Strosher 1996). Sources from the small oil and gas inventory identified as tanks or fugitive emissions, along with NPRI area sources were assumed to not come from combustion sources. Since the majority of PAH emissions are associated with incomplete combustion (EPA, 1996) atmospheric PAH emissions were assumed to be negligible from these sources.

PAH emissions based on NPRI emissions from the mining industry were assumed to be negligible since total VOCs reported to the NPRI from these sources were relatively small compared to the total VOC emissions in the study area. Mobile mining devices are assumed to be accounted for in the Offroad and Rail subcategory of the GEM-MACH tool for mobile, non-road, agricultural and other Inventory and PAH speciation associated with these are described above.

For background wood combustion and also for a bee hive burner that reported to NPRI, PAH was speciated based on work conducted by Hytone et al. (2009). The pulp and paper industry in the study area were all identified as mechanical separation, and release of PAHs from these sources is expected to be minimal. A search through the NPRI data base of all of Canada indicated that no PAH releases were reported for this kind of pulp and paper facility (mechanical separation). A similar Canada wide search for sawmills was conducted and two large sawmills reported PAH releases in 2010. Ratios between total VOCs and the PAHs in question were determined and applied to all sawmill point sources in the study area.

3. CALMET METEOROLOGICAL MODEL

The CALMET/CALPUFF dispersion modelling system was used to estimate ambient concentrations of COPC in the NE BC modelling domain. CALMET is a meteorological model that develops hourly three-dimensional meteorological fields of wind and temperature used to drive pollutant transport within CALPUFF. CALPUFF is a multi-layer, non-steady-state puff dispersion model. It simulates the effects of time- and space-varying meteorological conditions on pollutant transport, transformation and deposition.

3.1 Model Period

The CALMET model period was conducted for one year (8760 hours) as per the *BC Modelling Guideline* (BC MOE 2008). The CALMET model period was between January 16, 2011 and January 15, 2012. This period was selected based on prognostic meteorological data availability.

3.2 Model Domain

The CALMET model domain was set to a 160 km by 192 km. Domain resolution was set at 2000 m. In the vertical direction, 10 layers were modelled, with the top of each layer set as 20, 40, 80, 160, 320, 600, 1000, 1500, 2200 and 3000 m above ground level. A Lambert conformal conic (LCC) map projection was used due to the size of the modelling domain. Map projection and grid control parameters are displayed in Table 5.

Table 5: Map projection and grid control parameters

Parameter	Default	Project	Comment
PMAP	UTM	LCC	Map Projection
FEAST	0.0	0.0	False easting (km) at the projection origin
FNORTH	0.0	0.0	False northing (km) at the projection origin
RLAT0	-	55.25N	Latitude (decimal degrees) of projection origin
RLON0	-	120W	Longitude (decimal degrees) of projection origin
XLAT1	-	50N	Matching parallel(s) of latitude (decimal degrees) for projection
XLAT2	-	60N	Matching parallel(s) of latitude (decimal degrees) for projection
DATUM	WGS-84	WGS-84	Datum-region for output coordinates
NX	-	80	No. X grid cells
NY	-	96	No. Y grid cells
DGRIDKM	-	2.0	Grid spacing (km)

Parameter	Default	Project	Comment
XORIGKM	-	-160.0	Reference coordinate of SW corner of grid cell (1,1) -X coordinate (km)
YORIGKM	-	0.0	Reference coordinate of SW corner of grid cell (1,1) -Y coordinate (km)
NZ	-	10	Vertical grid definition: Number of vertical layers
ZFACE	-	0,20,40,80,160, 300,600,1000, 1500,2200,3000	Vertical grid definition: Cell face heights in arbitrary vertical grid (m)

3.3 Prognostic Meteorology

The CALMET model was initialized using Weather Research and Forecasting (WRF) prognostic model output at 1 km resolution provided by BC Hydro. The WRF model is a meso-scale numerical weather prediction system designed to serve both atmospheric research and operational forecasting needs. It represents the latest numerical weather forecasting model to be adopted by the United States National Weather Service as well as the United States military and private meteorological services.

3.4 Surface Meteorology

The WRF data were supplemented with hourly meteorological data from all known surface stations within the modelling domain. Hourly measurements of wind speed and direction, ambient temperature, pressure and relative humidity from 10 meteorological stations were included as inputs to CALMET.

3.5 Terrain Elevation and Land Use Characterization

The terrain elevation and land use characterization information used as input into the CALMET model were obtained from GeoBase (http://www.geobase.ca). Terrain elevations were obtained from 1:50,000 scale Canadian Digital Elevation Data.

3.6 Model Options and User Characterization

A list of the switch settings for use in the CALMET model is recommended in the *Guidelines for Air Quality Dispersion Modelling in British Columbia (Guidelines)* (BC MOE 2008). In general, model switch settings were chosen in accordance with these *Guidelines* (BC MOE 2008) with some exceptions provided in Table 6.

Table 6: Specific CALMET model options selected for the study

Parameter	Default	Project	Comment
Radius of Influence Parameters:			
LVARY	F	T	Varying radius of influence
RMAX1	-	10.0	Maximum radius of influence over land in the surface layer (km)
Other Wind Field Input Parameters:			
TERRAD	-	10.0	Radius of influence of terrain features (km)
R1	-	2.0	Relative weighting of the first guess field and observations in the surface layer (km)

Parameter	Default	Project	Comment
Diagnostic Module Data Input Options			
ISURFT	-	8	Surface meteorological station to use for the surface temperature

4. CALPUFF DISPERSION MODEL

4.1 CALPUFF Introduction

Dispersion modelling was conducted using the CALPUFF dispersion model (Version 5.8.4) in accordance with the *Guideline for Air Quality Dispersion Modelling in BC* (2008). CALPUFF is a multi-layer, multi-species, non-steady-state puff dispersion model. It simulates the effects of time and space-varying meteorological conditions on pollutant transport, transformation and deposition. For this study, CALPUFF modelling was driven by CALMET meteorology, as discussed in Section 3. The dispersion model CALPUFF was used to predict ground-level concentrations for the 28 COPC throughout the study area. Additional information about the model selection and modelling parameters are included below.

4.2 Source Types and Groups

To understand the contribution of various source groups, and to enable scaling of model results to predict maximum concentrations of all individual COPC, emission sources were grouped into numerous model runs based on the speciation profiles discussed in Section 2.4.4. Results for all model runs for each source group were summed to determine the combined effects of all sources within the modelling domain.

4.3 Receptors

Locations where the concentrations of COPC were calculated by the model are called receptors. The receptor grid is displayed in Figure 4. Since no major NPRI sources or communities were identified on the outskirts of the HHRA study area to the west and the north and in an effort to reduce the model run time, the receptor grid extent was reduced to 150 km wide by 176 km (slightly smaller than the study area originally proposed in the SLRA). Receptors were placed 2 km apart. A finer grid of receptors (250 m spacing) was placed in areas of interest. These areas included:

- large communities including Fort St. John, Dawson Creek, Chetwynd, Hudson Hope, Pouce Coupe and Taylor;
- within the First Nation lands of West Moberly Lake, East Moberly Lake, Halfway River, Doig River and Blueberry River; and,
- within 3 km from the largest 15 oil and gas emitters in the HHRA study area (using NPRI 2010 emission rates) as displayed in Figure 4. Facilities included in these large emitters include gas plants (11), large compressor stations (2) and booster stations (2).

A single receptor in each of the 13 smaller communities that were shown in Figure 1 was also added. A total of 15,676 receptors were used for this study. It is of note that receptors were placed throughout the

modelling domain, and contrary to regulatory practices, some receptors may have been placed within the fenceline of facilities due to lack of clarity on facility property lines.

4.4 Model Options and User Switches

A list of the switch settings used in the CALPUFF model is recommended in the *Guidelines* (BC MOE 2008). In general, model switch settings were chosen in accordance with the *Guidelines* (BC MOE 2008) with some exceptions provided in Table 7.

Table 7: Specific CALPUFF model options selected for the study

Parameter	Default	Project	Comments
MBDW	1	0	Building downwash not simulated
MCHEM	1	0	No chemistry simulated
MWET	1	0	Wet removal not modelled
MDRY	1	0	Dry deposition not modelled
MDISP	3	2	Dispersion coefficients from internally calculated sigma v, sigma w using micrometeorological variables (u*, w*, L, etc.)
MPDF	0	1	PDF used for dispersion under convective conditions

4.5 NO_X Chemistry

Emissions of NO_X are often reported as NO_2 equivalence. The primary emission is in the form of NO. Reactions in the atmosphere result in the conversion of NO to NO_2. The BC ambient air quality objectives are based on NO_2 and not the sum of the two. The most conservative method to estimate NO_2 is to assume 100% conversion of NO_X into NO_2. Since a more accurate estimate was desired, the ozone-limiting method (OLM) was used. NO_2 concentrations were calculated based on the following equation;

$NO_2 = 0.1*NO_X$ + lesser of (O_3 or 0.9 NO_X).

Ozone concentrations were based on measurements from the ambient air monitoring station located at the Taylor Townsite station. As outlined in the *Guidelines (*BC MOE 2008), both maximum one-hour and 24-hour averaged ozone observations were used to determine the NO_X to NO_2 conversion.

5. AIR QUALITY MODELLING RESULTS

5.1 Maximum Modelled Air Quality Results

Dispersion modelling using CALPUFF was conducted to predict concentrations of COPC within the dispersion modelling study area using the methodology described in Sections 3 and 4 and emission inputs as described in Section 2. Maximum air quality modelling results for upstream oil and gas sources in the study area are presented in Table A2. At the request of Intrinsik, for select species and time averaging periods, 99th or 98th percentile was also represented. Maximum predicted concentrations in each of the communities in the region are also presented. Maximum air quality modelling results for upstream oil and gas sources along with the modelled background and other industrial sources in the

study area are presented in Table A3. Similar, to the upstream oil and gas results, maximum predicted ambient air concentrations for each of the communities in the region are also presented.

5.2 Spatial representation of Select COPC

With guidance from Intrinsik, select COPC were represented spatially including;

- 99th percentile for 1-hour SO_2,
- 98th percentile for 1-hour NO_2 and 24 hour $PM_{2.5}$,
- 1-hour maximum concentrations for H_2S, formaldehyde, and acrolein and;
- annual concentrations for $PM_{2.5}$, formaldehyde and benzene.

Predicted concentration contours for both cases, i.e., Oil and Gas Scenario vs. the Cumulative Scenario were provided. The figures representing COPC predicted concentrations are as follows;

- The 99th percentile for 1-hour SO_2 for the Oil and Gas Scenario is plotted in Figure 5 and for the Cumulative Scenario in Figure 6. The location of the maximum point of impact (MPOI) was in an area that has a number of oil and gas sources of SO_2 such as sour gas plants but were not located within a community.

- Maximum 1-hour H_2S concentrations for the Oil and Gas Scenario are represented in Figure 7 and for the Cumulative Scenario in Figure 8. MPOI occurs in a similar area as where the MPOI of SO_2 was located and is in area where multiple oil and gas sources are located.

- The 98th percentile for 1-hour NO_2 for upstream Oil and Gas Scenario is displayed in Figure 9 and for Cumulative Scenario in Figure 10. The MPOI occurs in the north part of the domain which was associated with oil and gas sources mostly related to multiple sources located in the small upstream oil and gas inventory. Relatively high concentrations were also modelled in Taylor which relate to the multiple oil and gas facilities in the area. The influence of mobile sources, to total NO_2 concentrations is observed in Figure 10.

- The 98th percentile for $PM_{2.5}$ for 24-hour averaging period is shown in Figure 11 for the Oil and Gas Scenario and Figure 12 for $PM_{2.5}$ concentrations for annual averaging periods. For the Cumulative Scenario, the 98th percentile for the 24-hour averaging period and maximum concentration for the annual averaging period is represented in Figure 13 and Figure 14 respectively. MPOI is associated with the mining industry, although there were other influences around some of the communities that included the pulp and paper/wood industry and mobile sources. Oil and Gas sources are relatively minor relative to other sources in the region.

- Maximum 1-hour formaldehyde concentrations for the Oil and Gas Scenario is represented in Figure 15 and for Cumulative Scenario in Figure 16. Annual averaged formaldehyde concentrations are presented in Figure 17 for the Oil and Gas Scenario and Table 18 for the Cumulative Scenario. Sources associated with the transport of oil and gas products along with multiple minor sources from the oil and gas industry are located in the area of the MPOI.

Differences between the Oil and Gas Scenario and the Cumulative Scenario is mostly due the contributions from the pulp and paper/wood industry along with residential fuel wood combustion.

- Annual concentrations for benzene for the Oil and Gas Scenario and the Cumulative Scenario are presented in Figure 19 and Figure 20, respectively (note the scale change between the two scenarios). The MPOI is associated with multiple non-oil and gas sources including mobile sources, combustion of fuel wood and the pulp and paper and wood industry

- Maximum 1-hour concentrations of acrolein are presented in Figure 21 and Figure 22 for the Oil and Gas Scenario and the Cumulative Scenario, respectively. Modelled concentrations associated with the MPOI of acrolein was associated with the combustion of fuel wood with minor contribution from the pulp and paper and wood industry.

5.3 Time series of select species

A time series records the modelled concentrations at a selected receptor for every hour of the year that was modelled. Time series can provide additional information when studying the combined effects of multiple COPC. Time series at selected receptors were requested by Intrinsik. These included 1-hour time series of acrolein, formaldehyde, SO_2, NO_2 and 24-hour averaged time series of $PM_{2.5}$ at the MPOI of individual COPC and/or receptors within town limits.

6. UNCERTAINTIES IN THE ANALYSIS

Dispersion models, by definition can only approximate atmospheric processes. Many assumptions and simplifications are required to describe real phenomena in mathematical equations. This is especially true in a large modelling domain size with thousands of individual sources. Many of the assumptions and emissions are based on typical operating conditions for the year selected and would be representative of most facilities.

Several sources of uncertainty affect the accuracy of the analysis. The main ones are summarized here:

1. Emission rates were based on publicly available NPRI data from the 2010 reporting year. Releases of NPRI substances to air are based on actual operations and are reported by the facility operators. The year 2010 was assumed to be a typical year in terms of operations at a given facility, unless directed otherwise. The accuracy of the emissions and locations used in the model is limited by the accuracy of the NPRI data. The emission rates have uncertainty because the methods used to calculate and report the emissions to NPRI could not be validated and will vary to some extent with time.

2. Non point-source emission data provided in the Environment Canada mobile, non-road, agricultural and other inventory were provided in bulk (i.e., total emissions by source category by province or census division) and these emissions needed to be spatially allocated into the modelling domain. The accuracy of the spatial surrogate generation for area and mobile sources are subject to uncertainties caused in part by assumptions that are made about the source of

emissions and their relationship to particular activity, and also by the potential for error in the GIS data geometry and associated attributes which are used to represent that given activity.

3. Generic VOC speciation was conducted based on source type for all background and for sources in the small oil and gas emission inventory. In reality, individual sources could have different speciation profiles than the generic ones assumed.

4. Emission data was assumed to be constant throughout the year and throughout each day for major NPRI sources and in the small oil and gas inventory. For many industrial processes, emissions can be cyclical throughout the day and even the year.

5. Assumptions on stack dimensions such as stack height and diameter, as well as exit velocity and temperature were simplified at many of the facilities. Although this does not affect the amount of release of COPC from the facility, this can influence the predicted maximum concentrations and locations. This is also true for modelled area sources, where, in many cases release height had to be assumed based on the type of facility and area of the source was calculated based on the facilities' foot print calculated by using satellite images.

6. Atmospheric turbulence and dispersal of pollutants have inherent uncertainty. The U.S. EPA indicates that dispersion model accuracies have been reported in the range of plus or minus 10% to plus or minus 40% for predicting the maximum concentration over a year, independent of time and space (U.S. EPA 2003). The accuracy is reduced when predicting at specific locations in space.

7. The influence of structures and buildings on the point sources was not considered due to lack of available data on the building and structure characteristics. This may result in an underestimation of the near-field impacts from short stacks, as these sources are typically influenced by the aerodynamics of adjacent structures and buildings.

8. Meteorological data have some uncertainty. Meteorological conditions vary somewhat around facilities, and for a practical analytical approach, only a few surface observations were used. Also, the relatively coarse resolution (2-km) of the CALMET output is another source of uncertainty. For example, an uncertainty of 5 to 10 degrees in the measured wind direction can result in concentration errors of 20% to 70% for an individual event (US EPA 2003).

7. CONCLUSIONS

RWDI AIR Inc. assessed the ambient air concentrations in northeastern BC in regards to the oil and gas industry. Maximum concentrations for 28 chemicals of potential concern were predicted for the study area, as well as in each of the individual communities in the region. Averaging periods for each of 28 COPC were determined based on the SLRA (Intrinsik 2014), and included 1-hour, 24-hour and annual averaging periods. Two scenarios were considered; the first was based on emissions from only the oil

and gas industry, and the second, represented both oil and gas industry as well as other non-oil and gas sources.

For both scenarios, the maximum predicted concentrations were determined and additional spatial analysis and/or time series were conducted based on guidance from Intrinsik. All results were provided to Intrinsik for further assessment of potential health risks.

8. REFERENCES

BC Hydro, Personal Communication between Al Strang and David Chadder RWDI AIR Inc. February 2014.

British Columbia Ministry of Environment. 2008. Guidelines for Air Quality Dispersion Modelling in British Columbia. Victoria, BC. 152 pp.

British Columbia Ministry of Environment. 2013a. British Columbia Ambient Air Quality Objectives. Victoria, BC. 4 pp.

Environment Canada. 2014. National Pollutant Release Inventory 2010. Website: http://www.ec.gc.ca/inrp-npri/ Accessed: January 2014.

Environment Canada, Air Quality Modelling and Applications Section, Personal Communication between Mourrad Sassi, Environment Canada and Martin Gauthier RWDI AIR Inc. Dorval, QC. January 2014.

Environment Canada, Personal Communication between David Niemi Environment Canada and Martin Gauthier RWDI AIR Inc. January 2013.

Environment Canada, Personal Communication between Mike Morgan Environment Canada and Martin Gauthier RWDI AIR Inc. December 2013.

Intrinsik Environmental Sciences Inc. 2014. Phase 2 – Human Health Risk Assessment of Oil and Gas Activity in Northeastern British Columbia, Victoria BC, 47 pp.

Hytone et al. 2009. Gas-Particle Distribution of PAHs in Wood combustion Emission Determined with Annular Denuders, Filters, and Polyurethane Foam Adsorbent, Aerosol Science and Technology, 43:5, 442-454.

Scire, Strimaitis and Yamartino. 2000. A User's Guide for the CALPUFF Dispersion Model (Version 5). Concord, MA. 521 pp.

Strosher. 1996. Investigations of Flare Gas Emissions in Alberta. Prepared for Environment Canada Conservative and Protection, Alberta Energy and Utilities Board and the Canadian Association of Petroleum Producers. 158 pp. Website: http://www.ags.gov.ab.ca/publications/SPE/PDF/SPE_005.PDF. Accessed: March 2014.

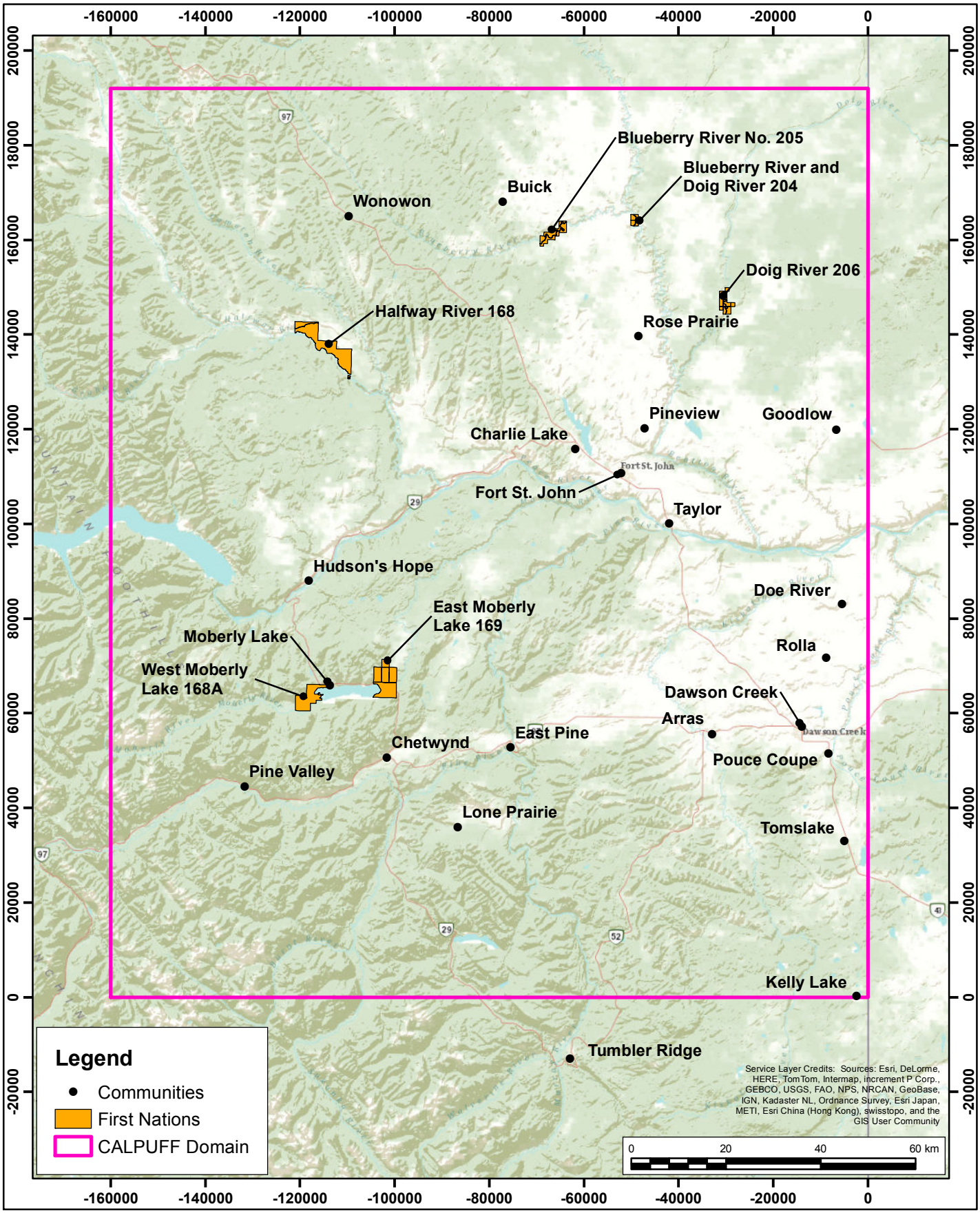
United States Environmental Protection Agency. 1995. User's Guide for the Industrial Source Complex (ISC3) Dispersion Models, Volume I – User Instructions. EPA-454/B-95-003a. 19 pp. Website: http://www.epa.gov/scram001/userg/regmod/isc3v1.pdf. Accessed: January 2014.

United States Environmental Protection Agency. 1996. AP-42, Fifth Edition Compilation of Air Pollutant Emission Factors. Volume I: Stationary Point and Area Sources. Section 3.3: Gasoline and Diesel Industrial Engines. Research Triangle Park, NC. 74 pp.

United States Environmental Protection Agency. 2010. MOVES2010b: Additional Toxics Added to MOVES. EPA-420-B-12-029a. 28 pp. Website: http://www.epa.gov/otaq/models/moves/documents/420b12029a.pdf. Accessed: January 2014.

United States Environmental Protection Agency. 2003, 40 CFR Ch. I (97-1-03 Edition), Part 51, App. W, Section 10.

FIGURES



Communities and First Nations within the HHRA Study Area

First Nations from Geobase Aboriginal lands, 2010.

Projection: Modified Lambert Conic Conformal.

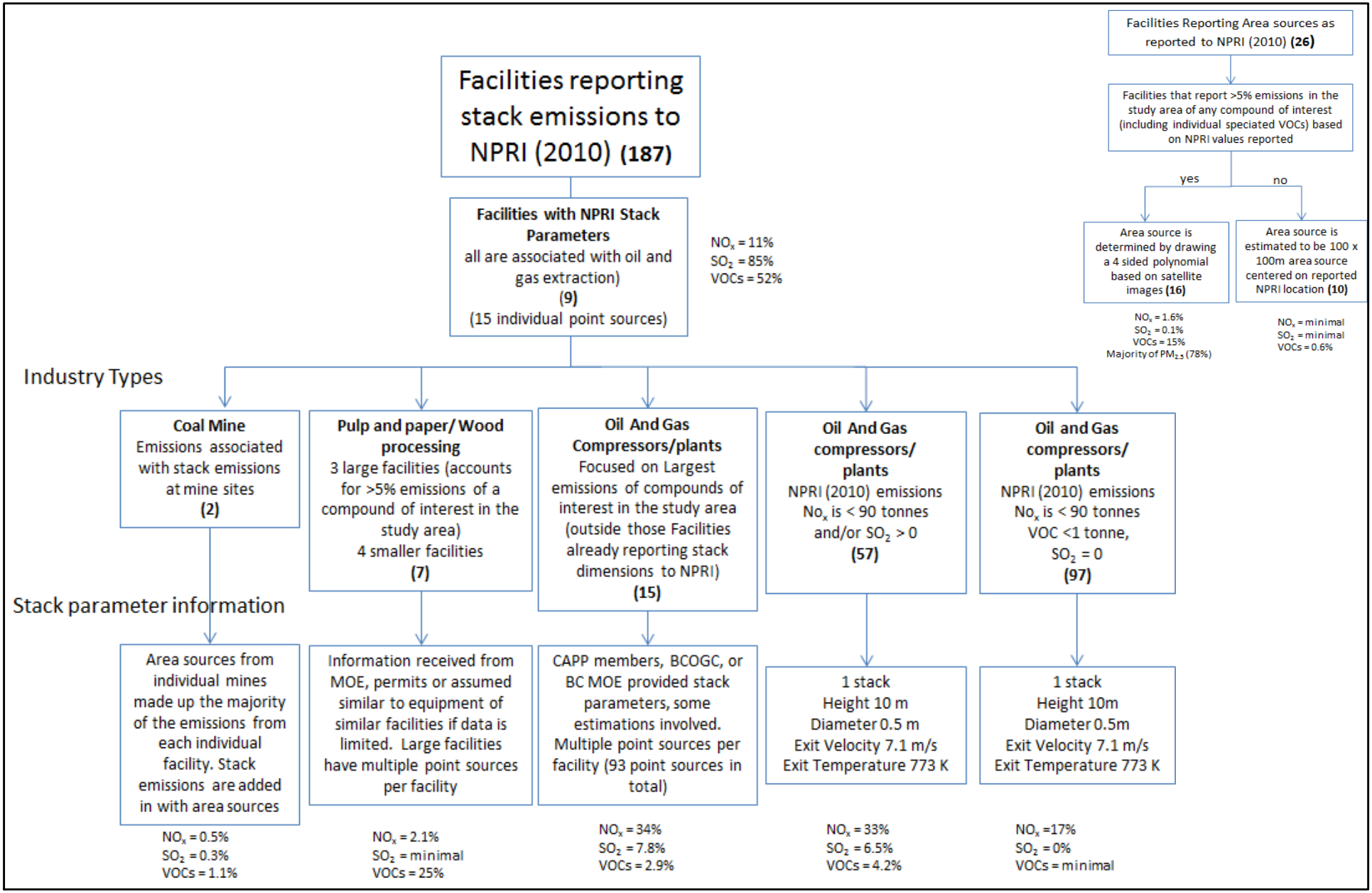
BC Ministry of Health, BC, Canada

True North

Project #1202326

Drawn by: CAM	Figure: 1
Approx. Scale: 1:1,100,000	
Date Revised: March 25, 2014	

RWDI



NPRI Emission Sources

Flow chart of the breakdown of NPRI Source Groups

BC Ministry of Health, BC, Canada

Bolded numbers in brackets represent number of facilities in the study area. The percentage of the total NPRI emissions in the study area for NO_X , SO_2 and total VOCs are reported beside the different subcategories.

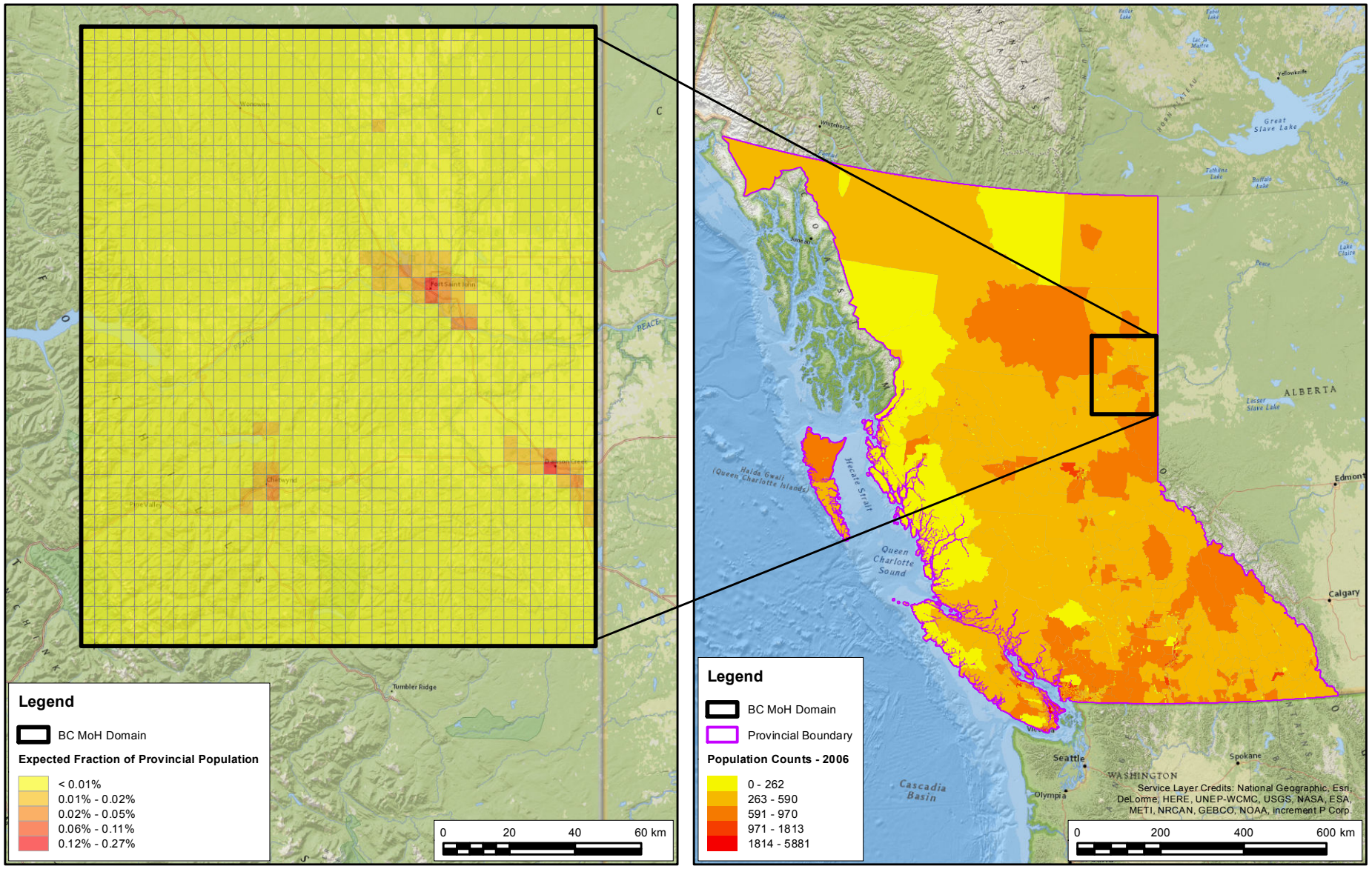
Project #1202326

Drawn by: MSE

Figure: 2

Date Revised: May 30, 2014

RWDI



Spatial Surrogates - Allocation of Population

Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

Provincial Total Population 2006 [1]: 4,113,487.

Total Population of whole Census DA's intersecting with BC MoH Domain [2]: 55,920 (1.4% of [1]).

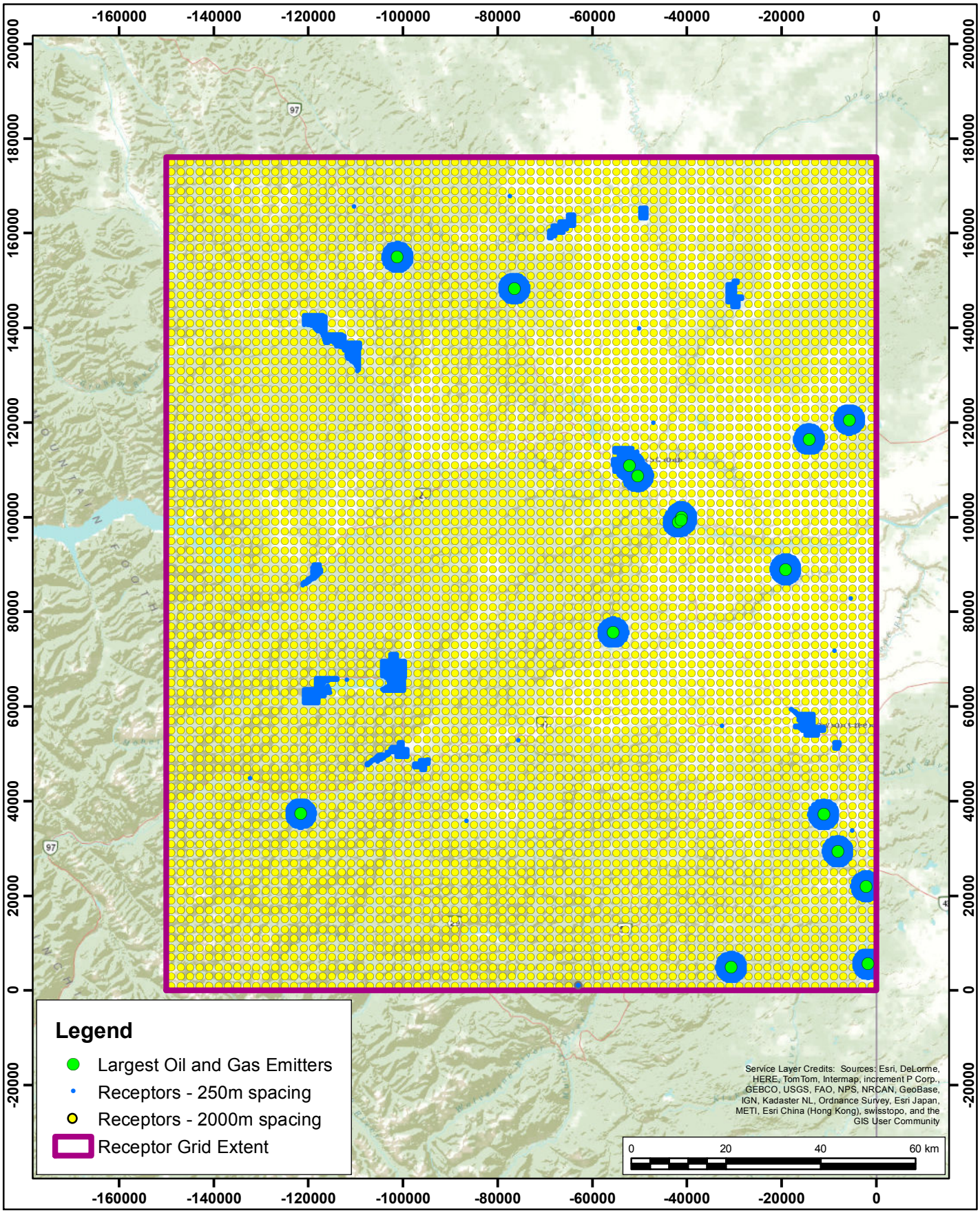
Sum of Surrogate Values found within BC MoH Domain: 0.012689 (1.27% of [1]).

True North

Project #1202326

Drawn by:	CAM	Figure: 3	
Approx. Scale (Left Map):	1:1,700,000		
Approx. Scale (Right Map):	1:13,500,000		
Date Revised:	May 29, 2014		

RWDI



Largest Oil and Gas Emitters and Receptor Grid

BC Ministry of Health, BC, Canada

Receptor Grid dimensions adjusted to 176km x 150km.
250m spacing over all Communities of Interest (Intrinsik), Aboriginal Lands within domain, plus within 3km of the top 17 Emitters.
2000m spacing outside of 250m-spacing areas.
Total Receptors: 15,676.

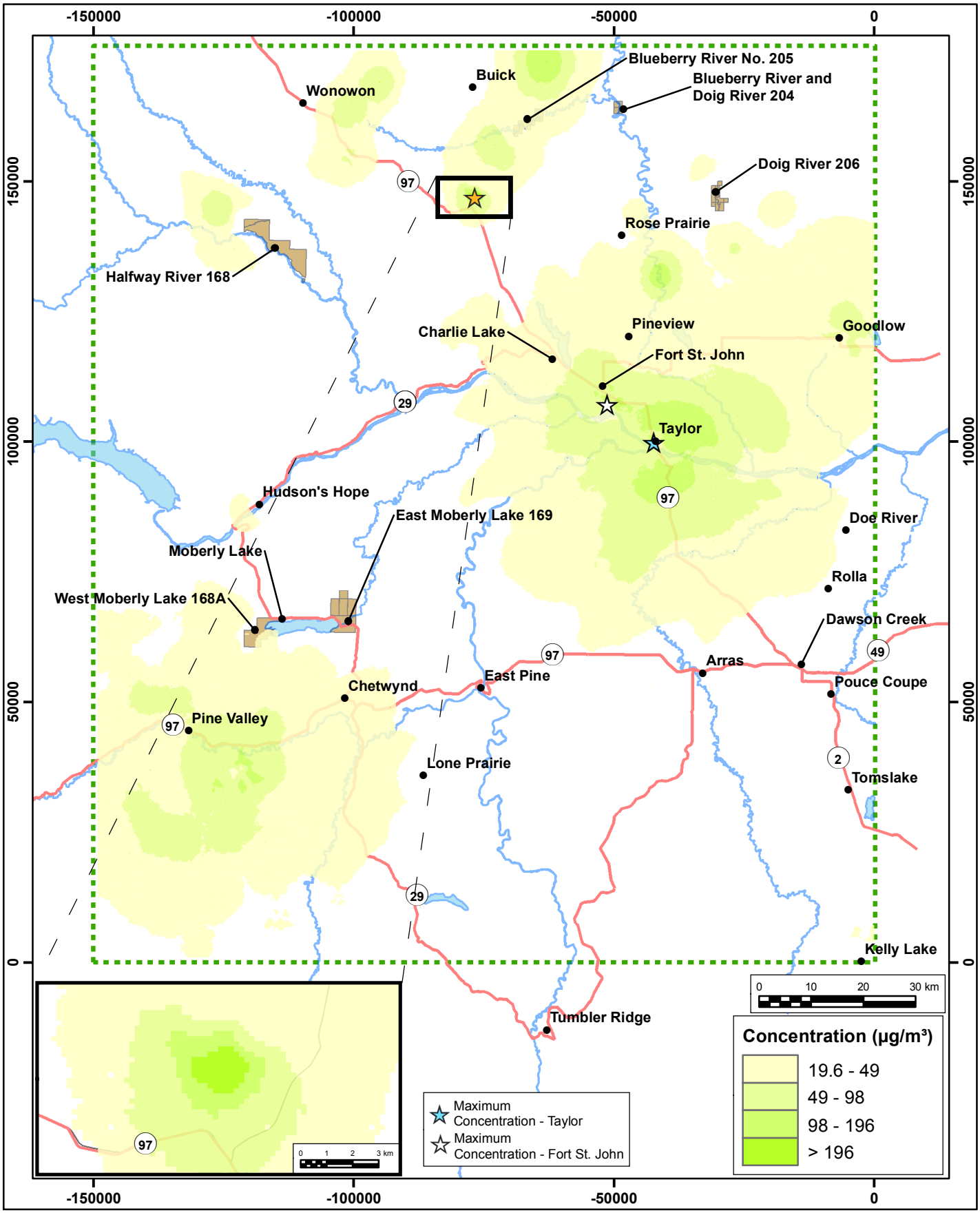
Projection: Modified Lambert Conic Conformal.

True North

Project #1202326

Drawn by: CAM	Figure: 4
Approx. Scale: 1:1,100,000	
Date Revised: March 25, 2014	

RWDI



SO_2 - 1hr Averaging Period (99th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

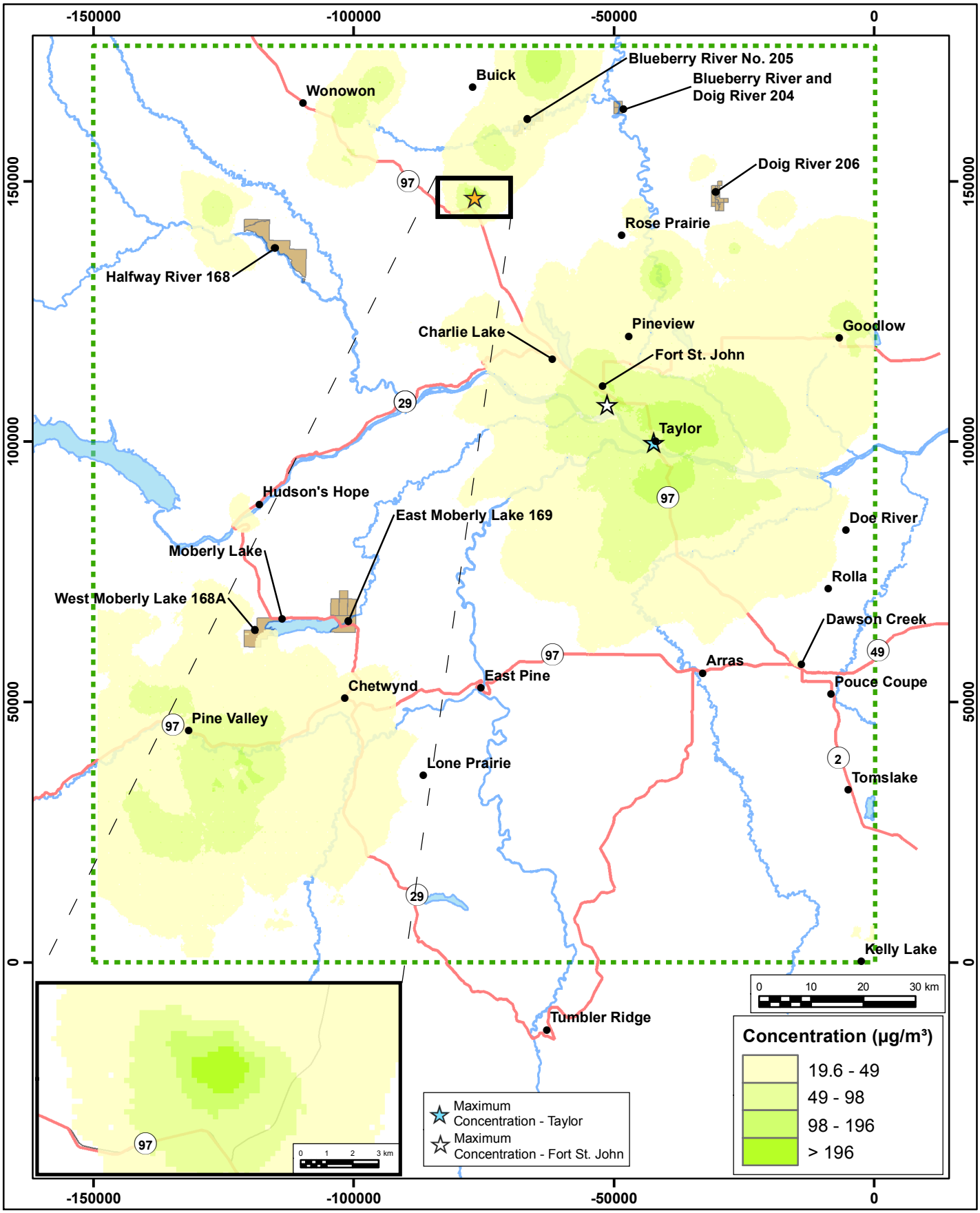
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 5
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



SO_2 - 1hr Averaging Period (99th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

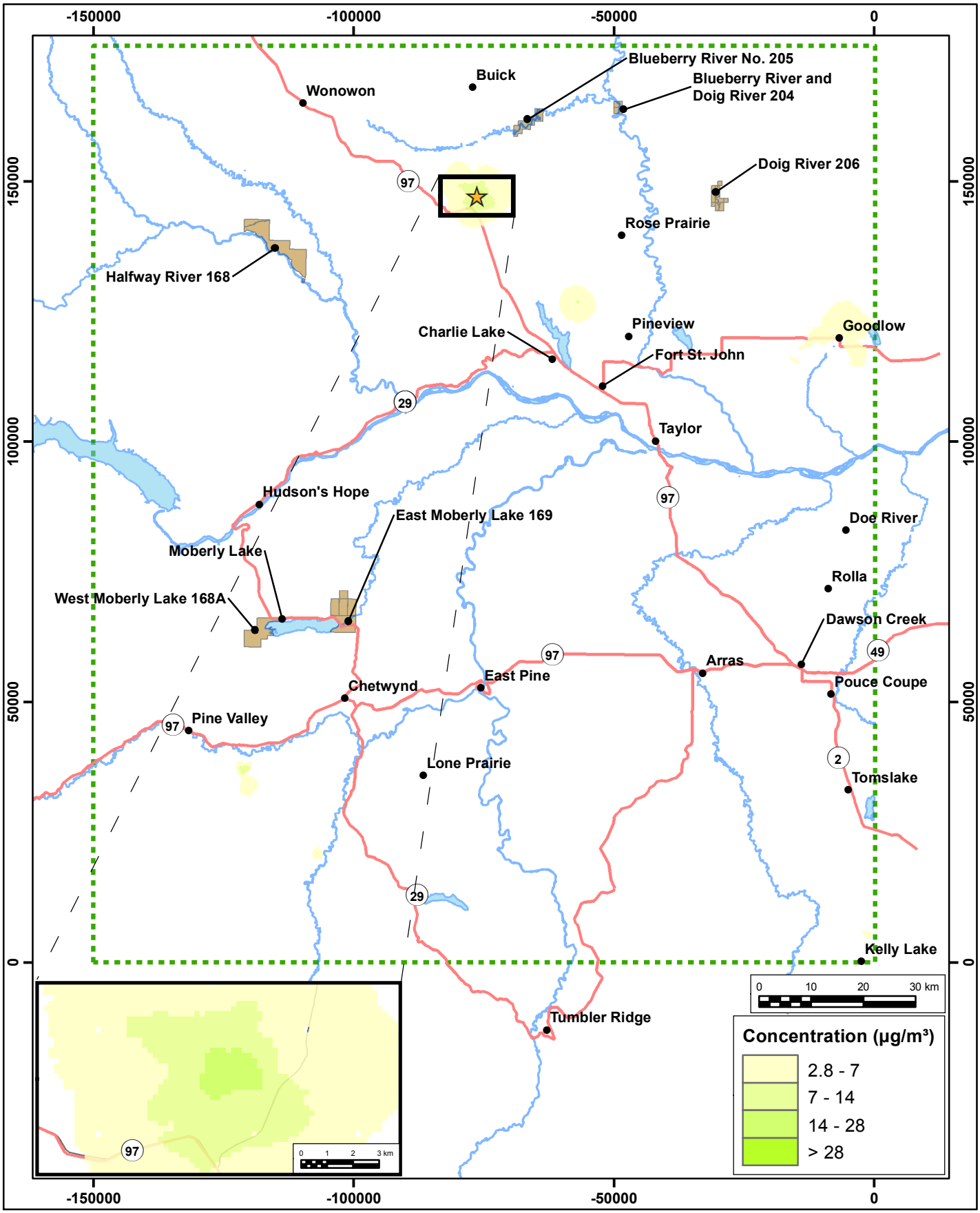
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 6
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Hydrogen Sulfide - 1hr Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

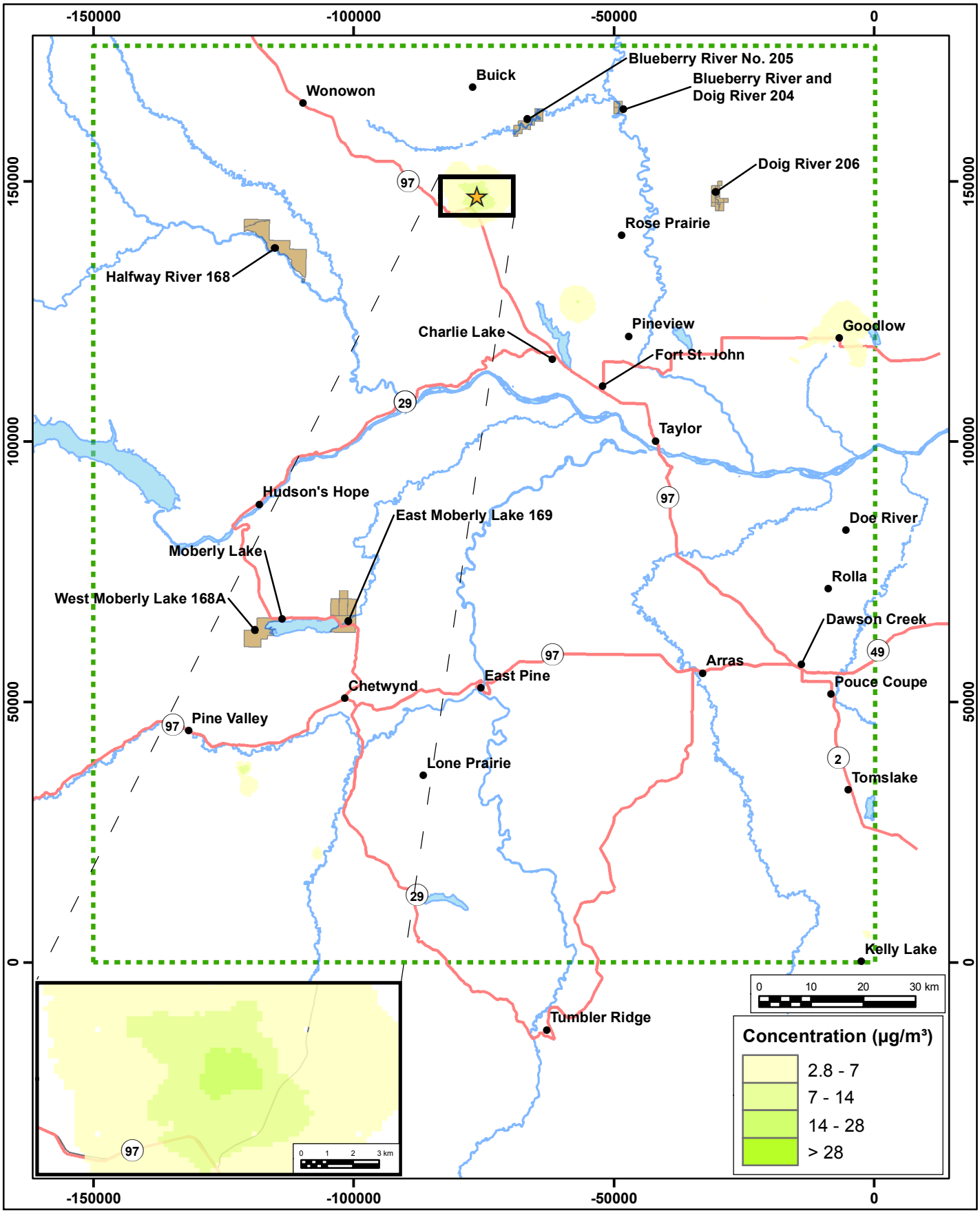
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 7
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Hydrogen Sulfide - 1hr Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

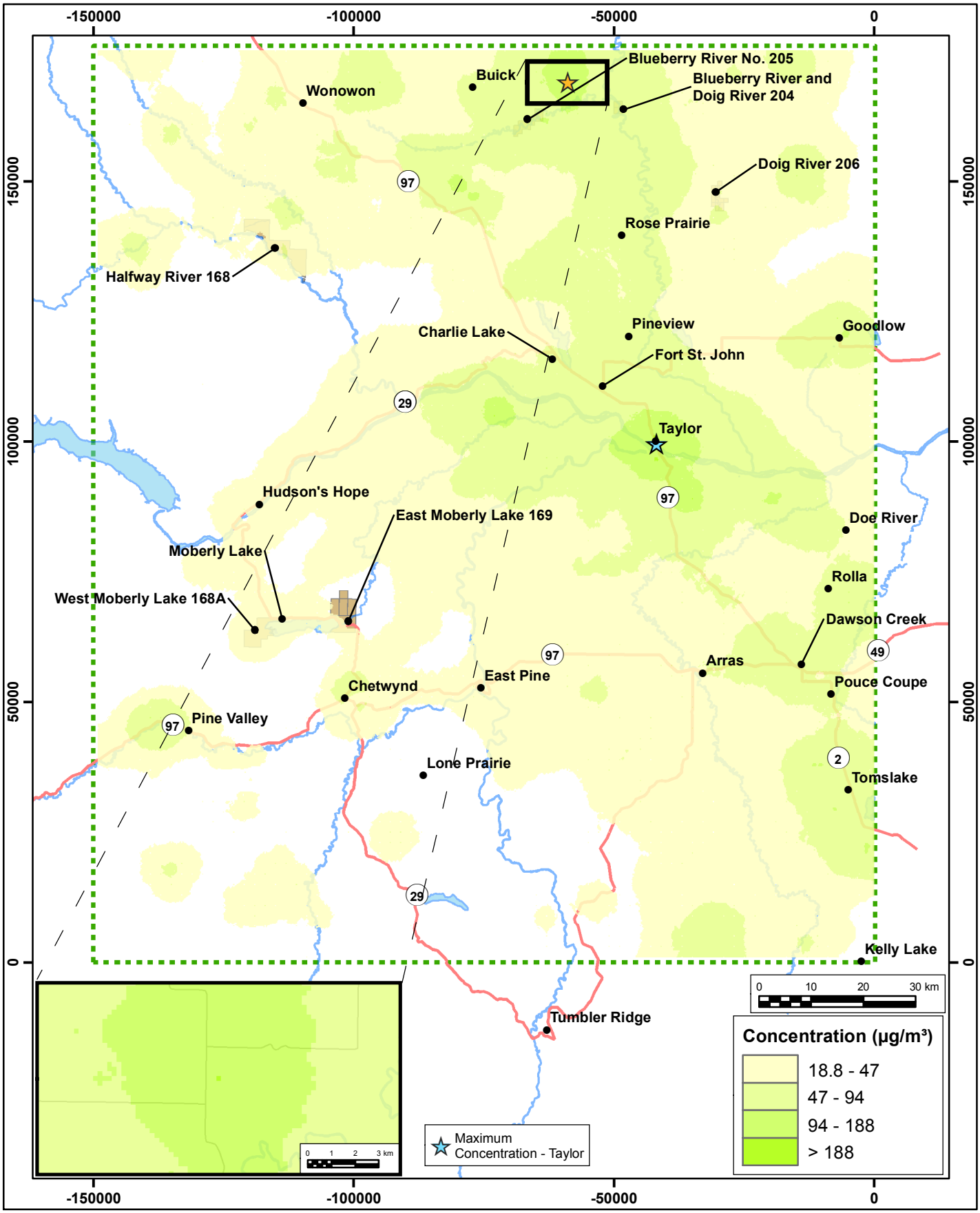
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 8
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



NO_2 - 1hr Averaging Period (98th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

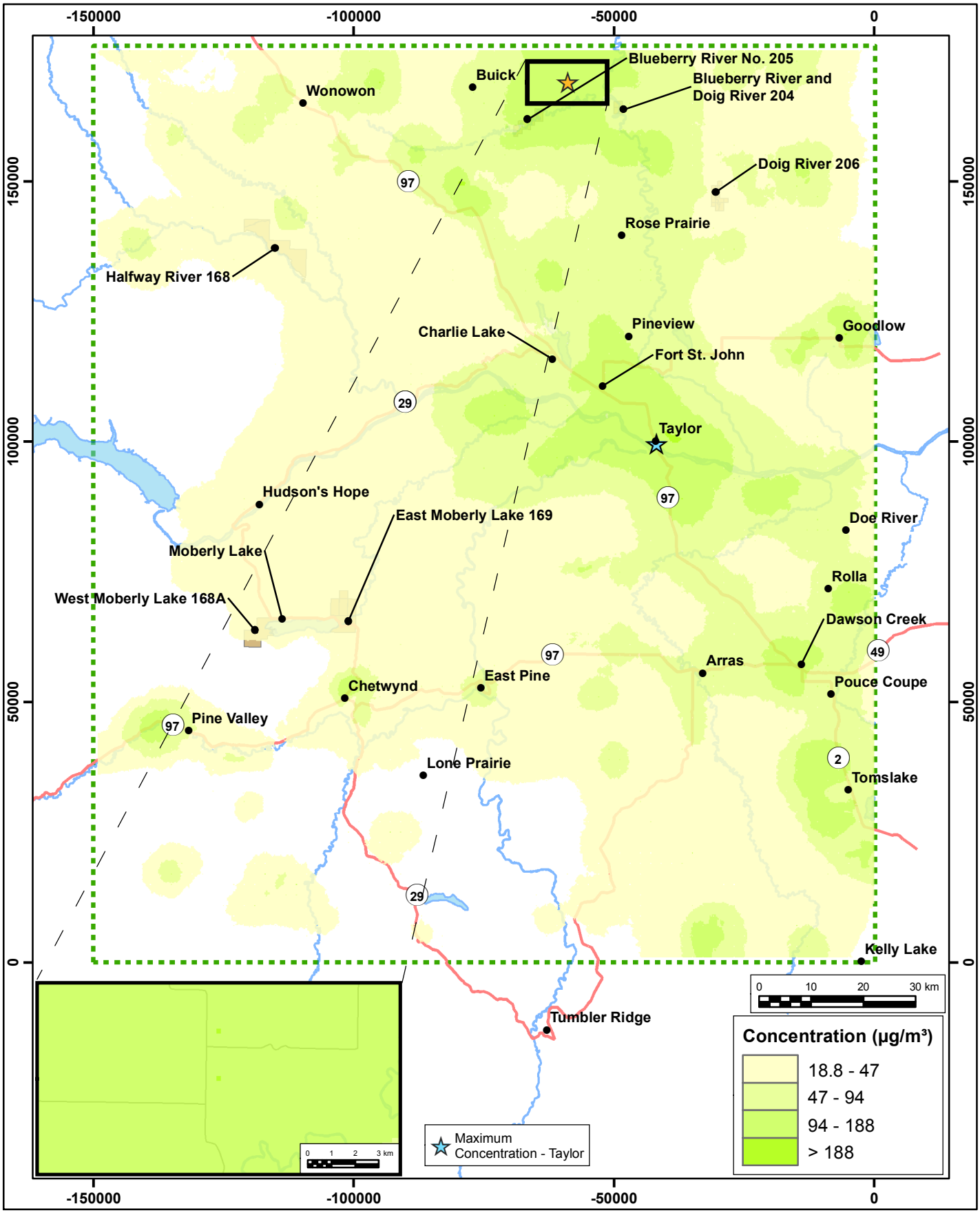
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 9
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



NO_2 - 1hr Averaging Period (98th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

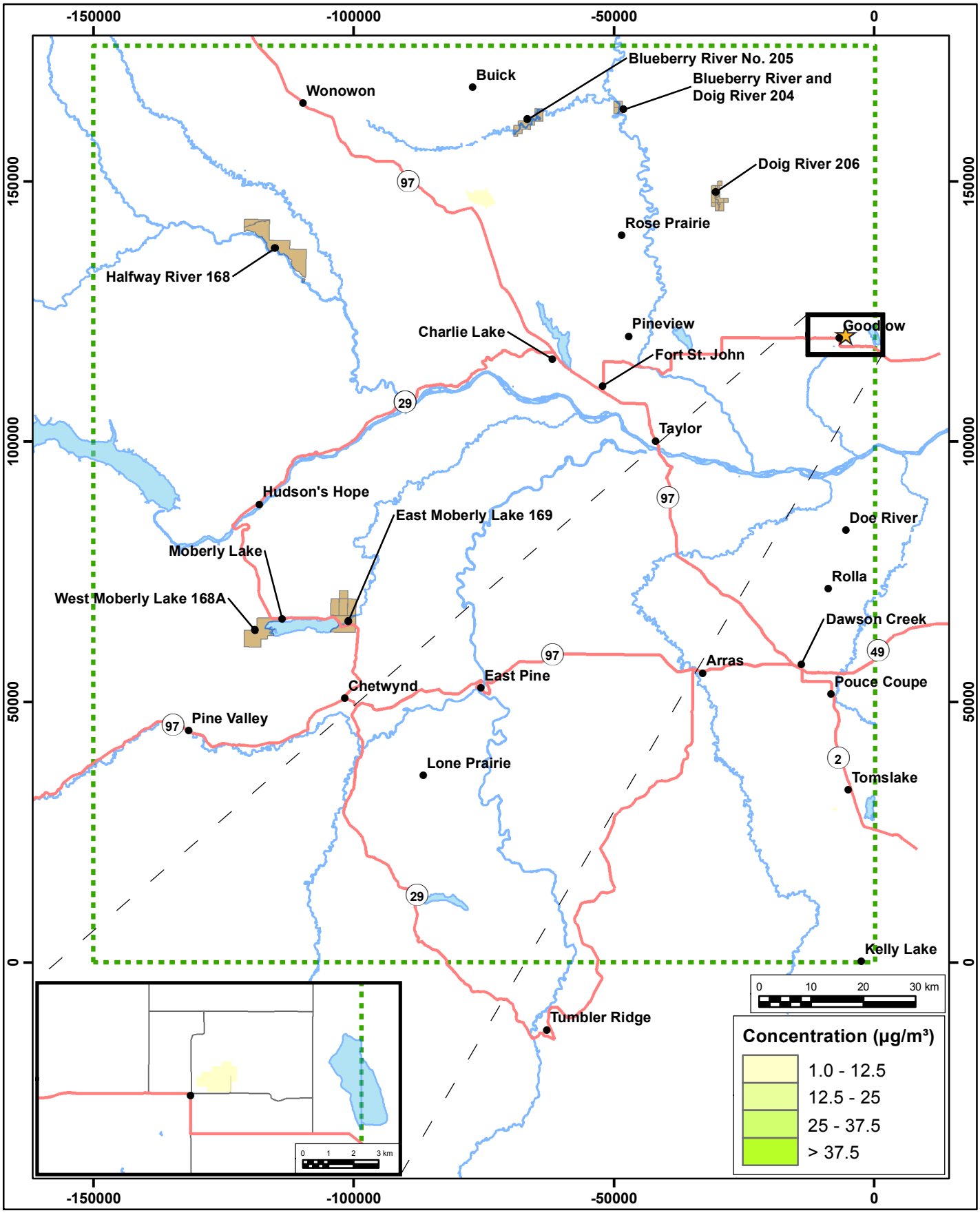
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 10
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - 24hr Averaging Period (98th Perc.)

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

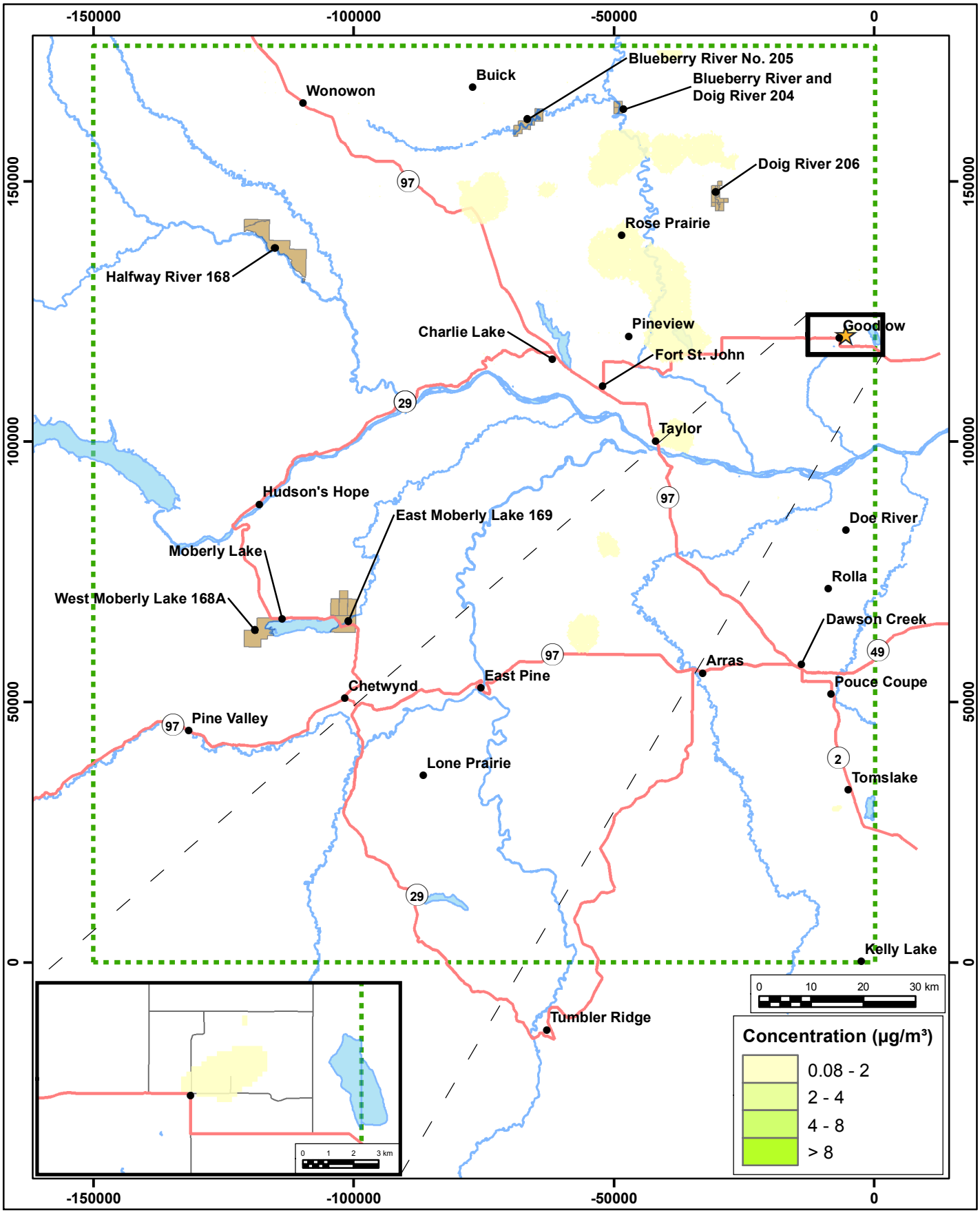
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 11
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - Annual Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

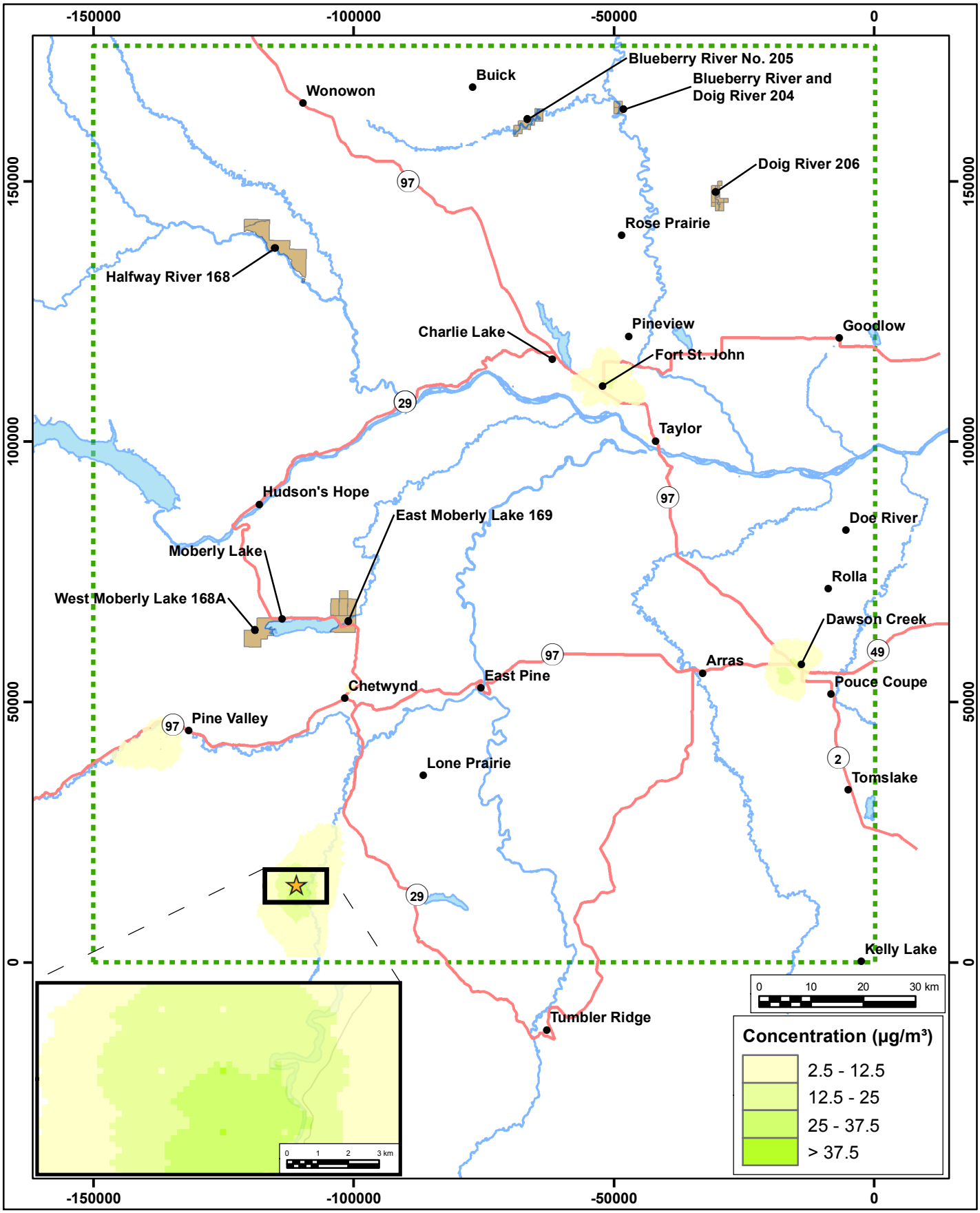
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 12
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - 24hr Averaging Period (98th Perc.)

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

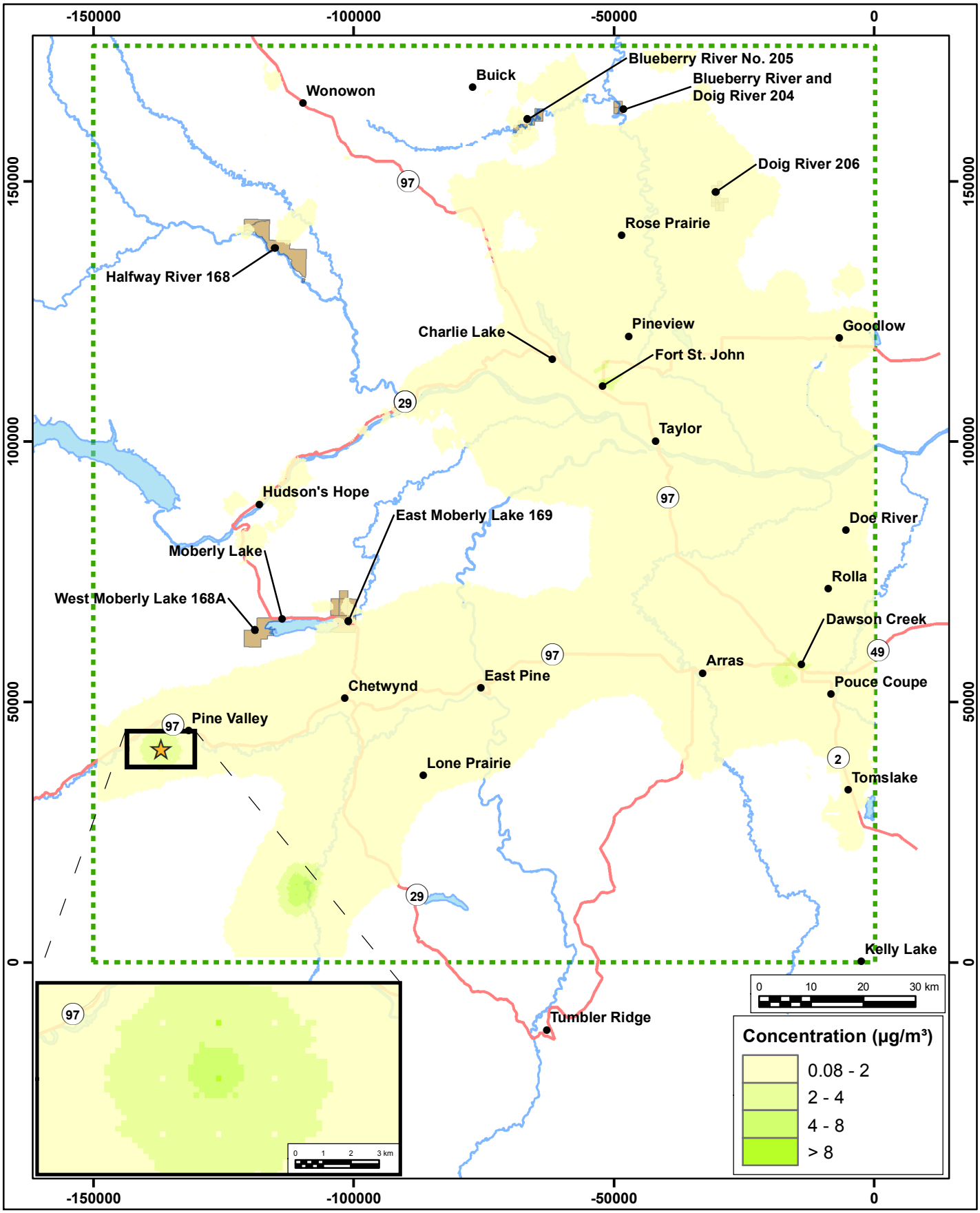
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 13
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



$PM_{2.5}$ - Annual Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

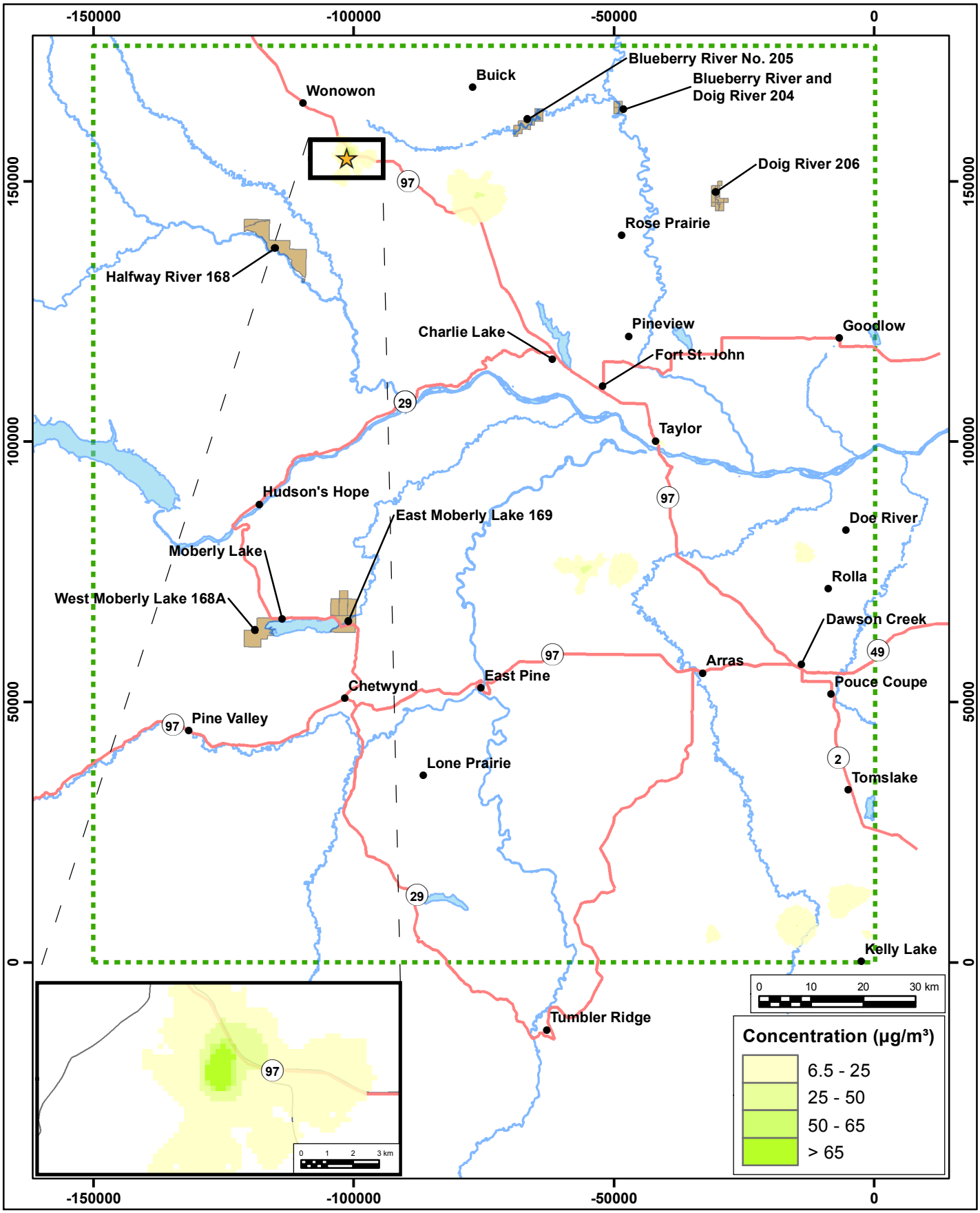
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 14
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - 1hr Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

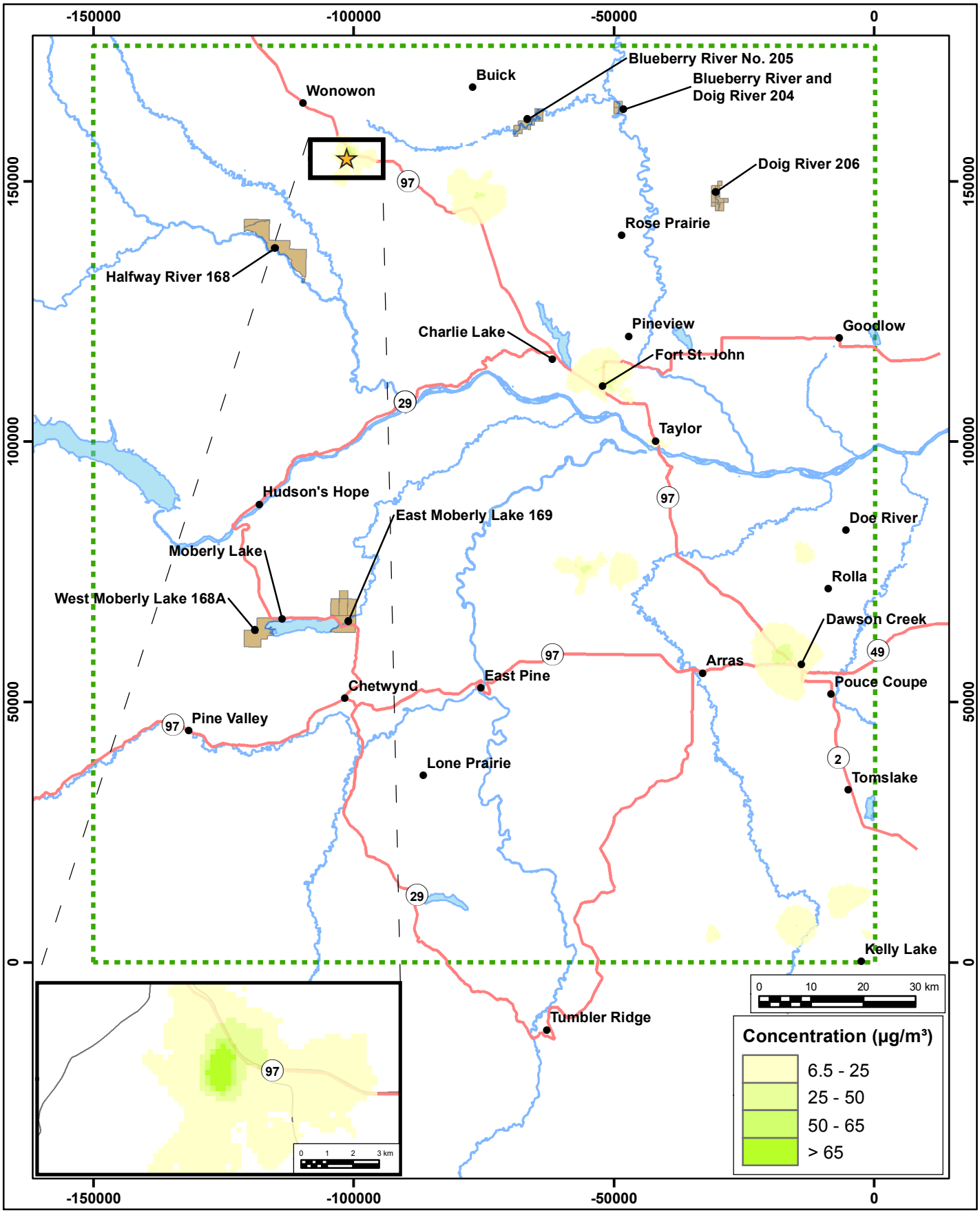
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 15
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - 1hr Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

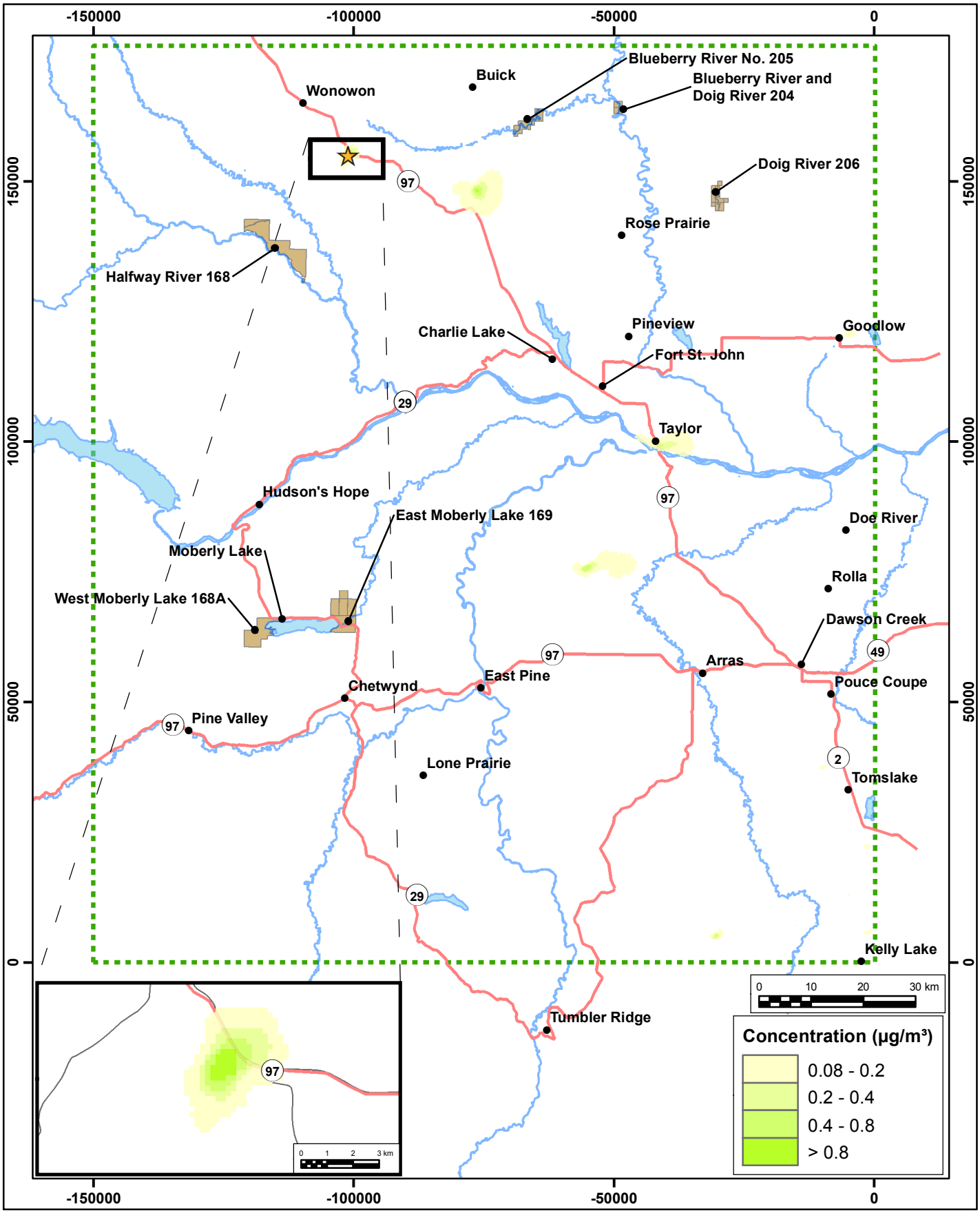
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 16
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - Annual Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

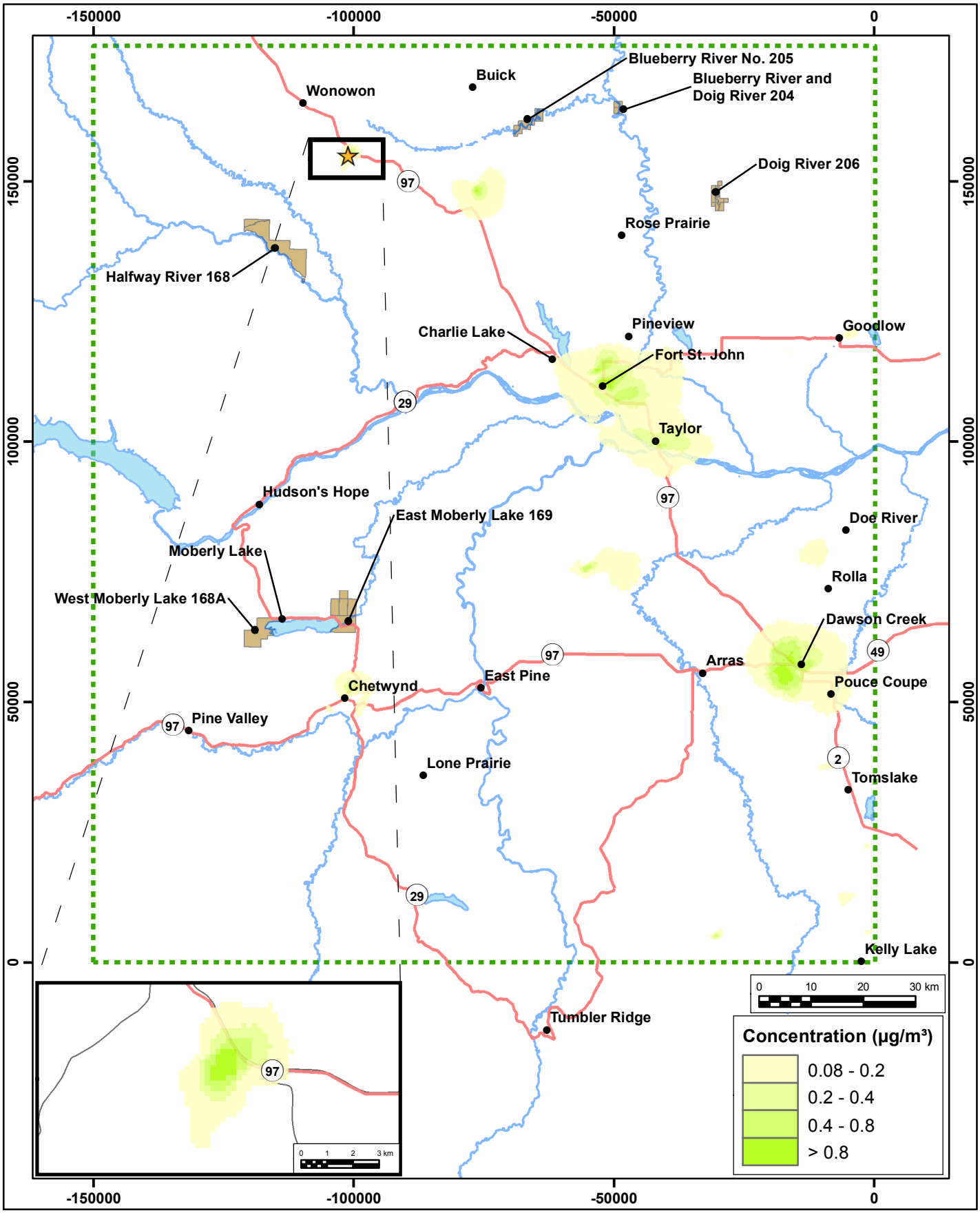
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 17
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Formaldehyde - Annual Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

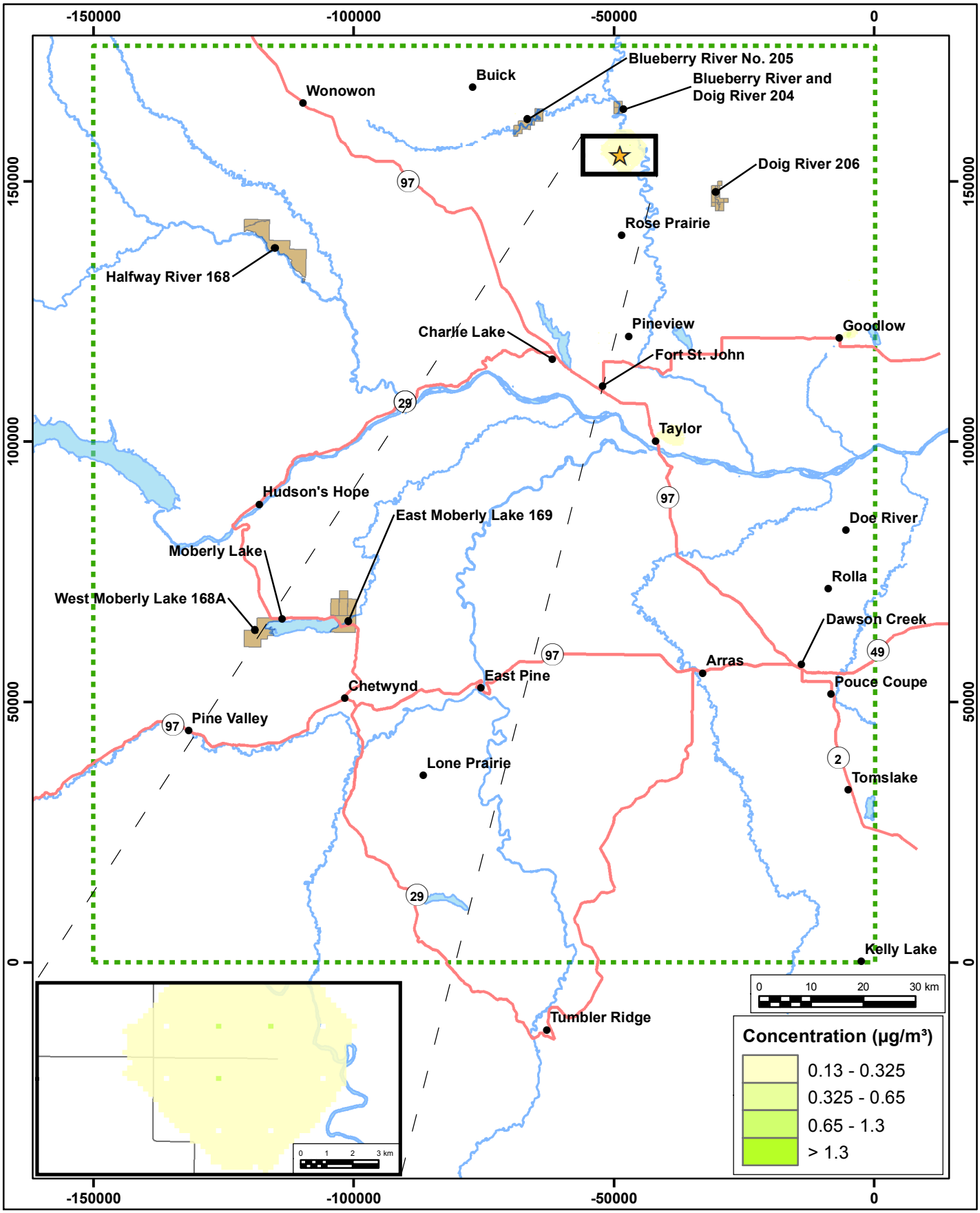
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 18
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Benzene - Annual Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

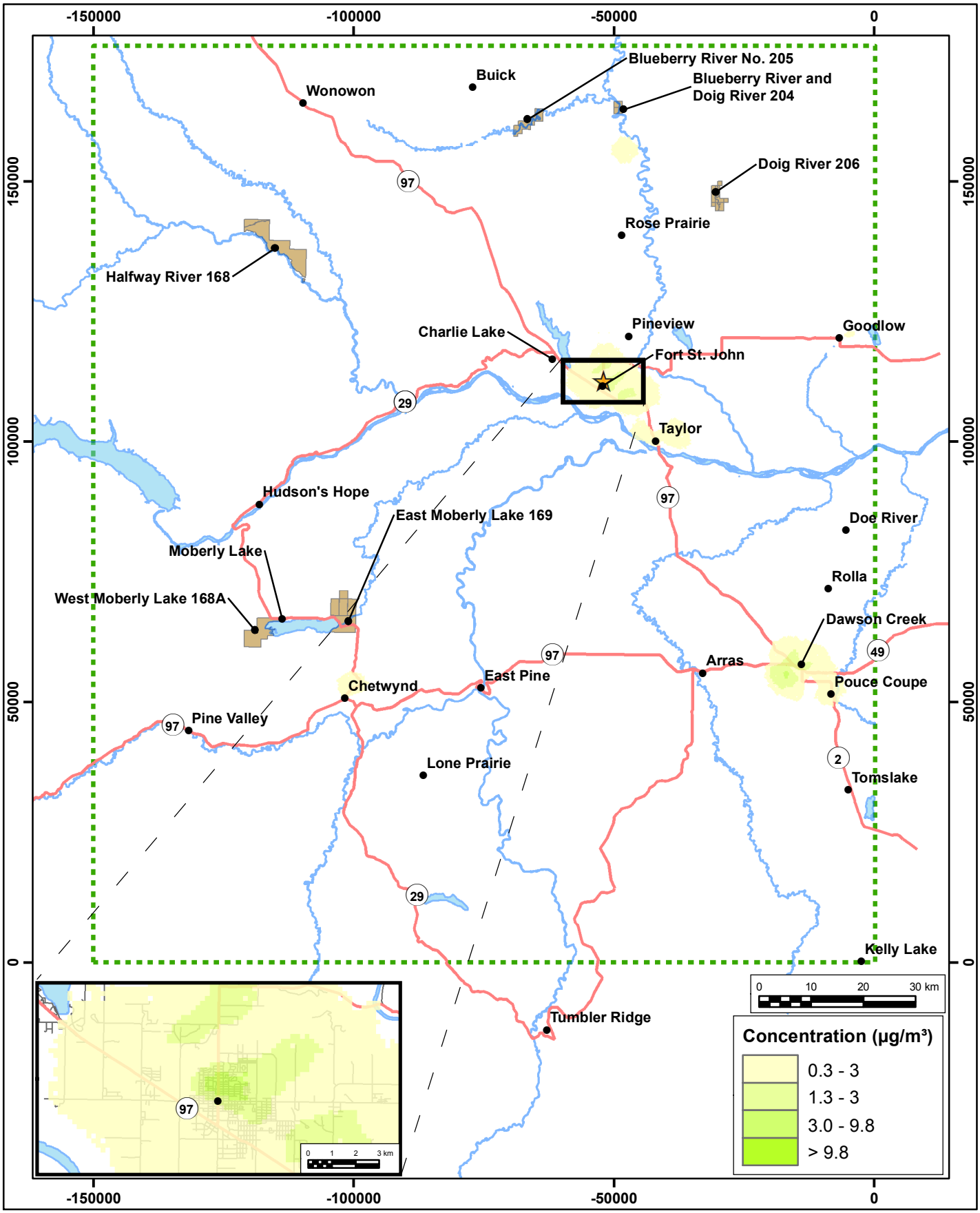
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 19
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Benzene - Annual Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

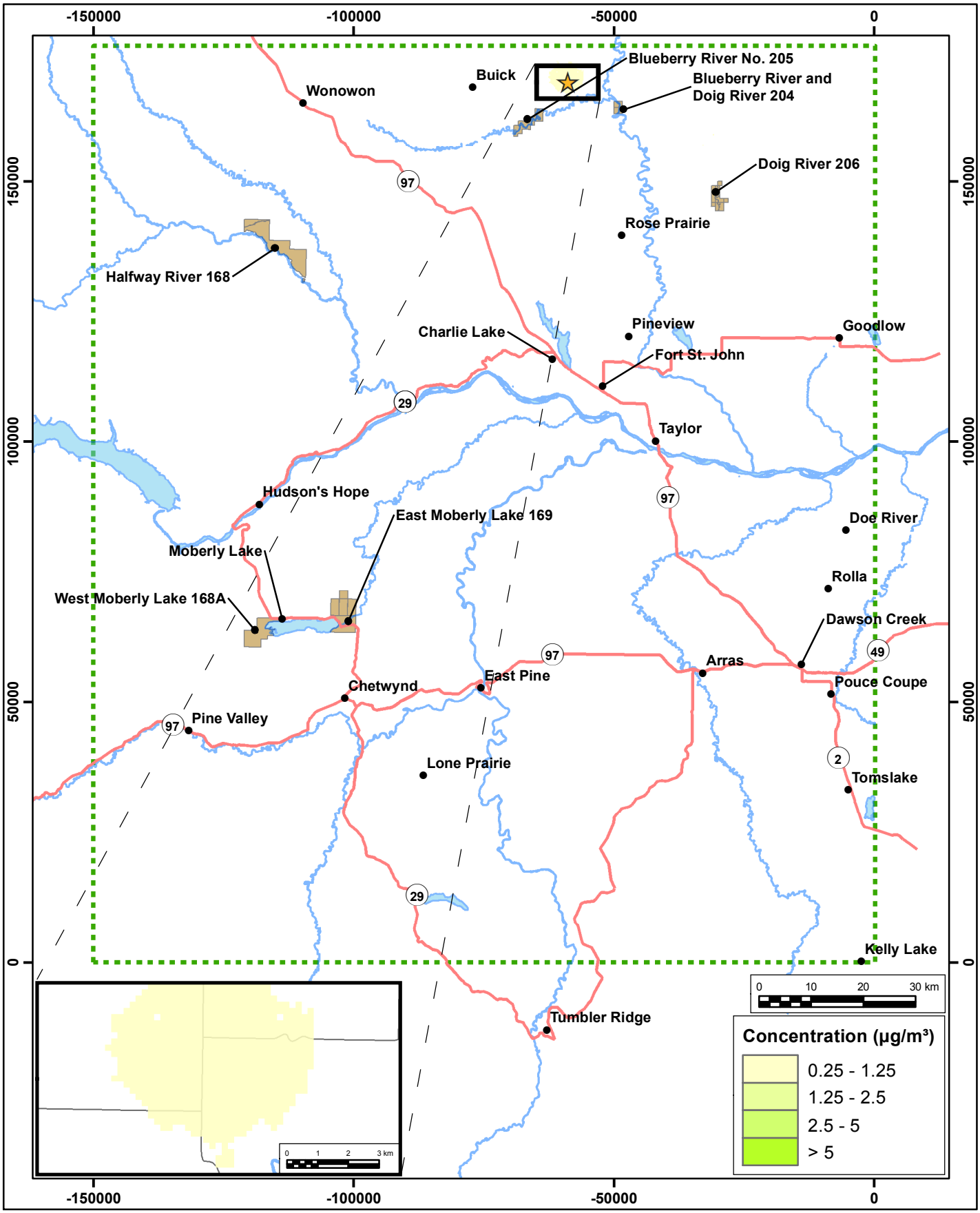
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 20
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI



Acrolein - 1hr Averaging Period

Oil and Gas Activities Only

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

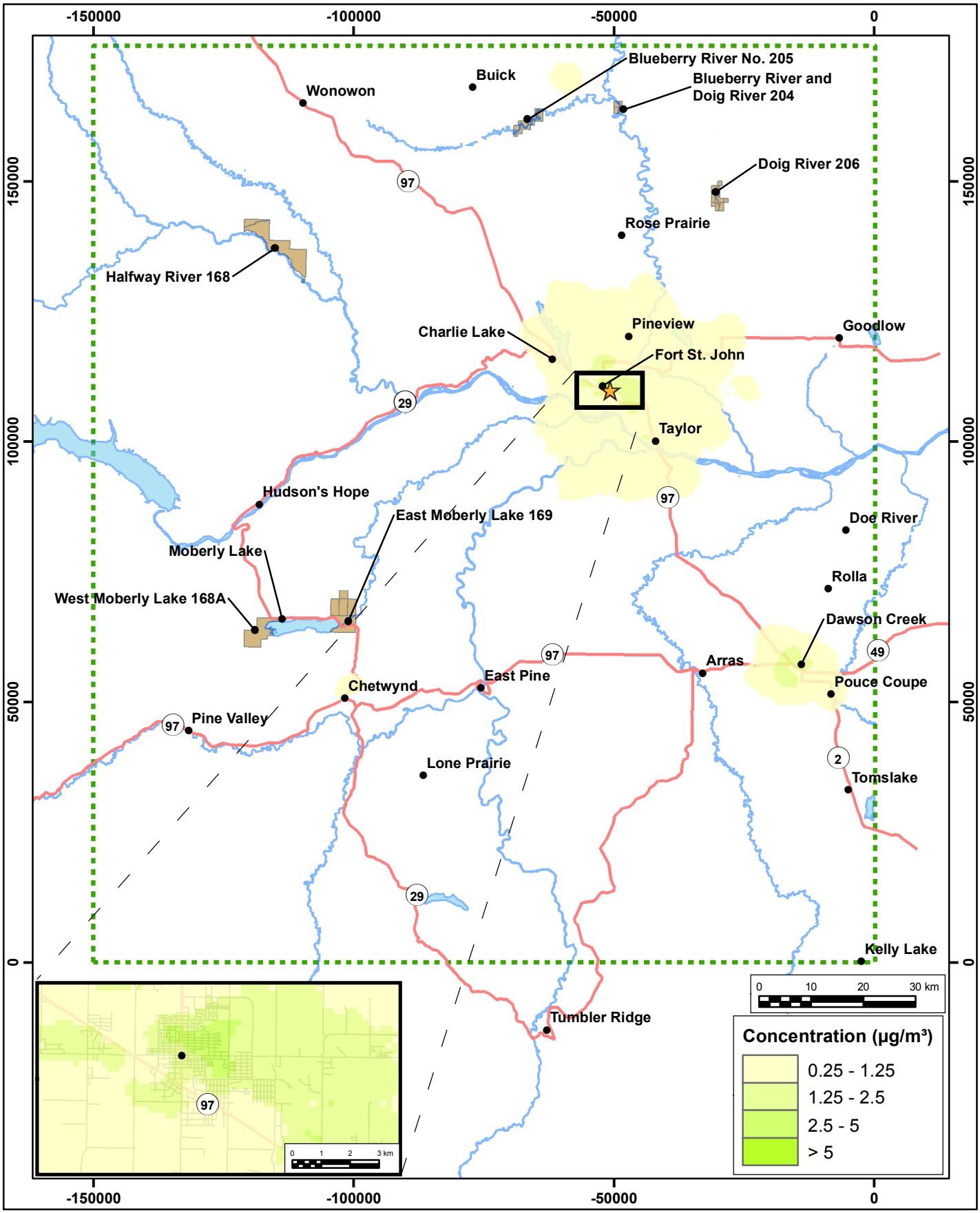
Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 21
Approx. Scale: 1:1,000,000	
Date Revised: Aug. 8, 2014	

RWDI



Acrolein - 1hr Averaging Period

Oil and Gas Activities and Background Sources

First Nations from Geobase Aboriginal lands, 2010.
Waterbodies from GeobaseNational Hydro Network, 2004.
Projection: Modified Lambert Conic Conformal.

BC Ministry of Health, BC, Canada

Maximum Concentration
Communities
Highways
Streets
Study Area Extent
First Nations
Waterbody

True North

Project #1202326

Drawn by: NBN	Figure: 22
Approx. Scale:	1:1,000,000
Date Revised:	Aug. 8, 2014

RWDI

APPENDICES

Table A1: Total Emissions in the modelling domain (tonnes/year)

Source type	Sub-category	SO2	NOx	PM2.5	CO	VOC	H2S	Acetaldehyde	Acrolein	Benzene	Benzo(a)pyrene	Butadiene, 1,3 -
NPRI - Upstream Oil and Gas [1]		17,655	17,828	140		4,494	65	0	0	9	0	0
EC - Small Upstream Oil and Gas												
	Combustion-flare	274	140	69		129	3			0.13	0.00	
	Combustion-diesel [2]	0.13	5,692	3		334		53.25	4.33	3.49		0.39
	Combustion-propane	0.00	0			1						
	Dehydrator					817				0.82		
	Leak					5,854				5.85		
	Load					1,000				24.00		
	Tank					6,493				6.49		
	Vent					1,308				1.31		
	Total	**274**	**5,832**	**72**	**0**	**15,936**	**3**	**53**	**4**	**42**	**0**	**0**
NPRI - Pulp and Paper/wood industry [1]			387	33.2		1,490		15.65	11.92	51.90		0.35
NPRI - Coal and mining		70.3	293	790.8		67		0.26	0.23	1.47		0.59
EC - Mobile On Road												
	Exhaust [3]					1,691		29.33	1.08	55.39		8.81
	Evaporative	8	4,384	11	22,352	1,205		7.83		32.23		3.56
	Total	**8**	**4,384**	**11**	**22,352**	**2,896**	**0**	**37.15**	**1.08**	**87.62**	**0.00**	**12.37**
EC - Non Road												
	Miscellaneous [4]	41	307	173	1,239	6,937		39.89	53.41	76.05		46.48
	Petroleum storage and transportation					151				31.24		
	Industrial utilization of solvent					755						
	aerosol coating					307						
	fuelwood combustion [5]	3	31	101	1,537	4,589		568.83	21.03	127.85		39.06
	Offroad and Rail [2]	87	3,240	21	9,177	1,447				15.69		0.03
	Other Non-Road	87	2,053	16	642	814		6.84	6.27	22.96		5.46
	Total	**218**	**5,630**	**310**	**12,594**	**15,001**	**0**	**615.55**	**80.71**	**273.79**	**0.00**	**91.02**
Total for all categories		**18,225**	**34,354**	**1,356**	**34,946**	**39,883**	**68**	**721.87**	**98.28**	**465.61**	**0.00**	**104.73**

Notes:

[1] VOC from the NPRI Upstream Oil and Gas and the paper industry was speciated further if not included in NPRI data base, based on speciation provided by (EC January 2013)

[2] Speciation of PAH was based on Speciate4.2 profile 4674 and USEPA AP42 Table 3.3-2.

[3] Speciation of PAH was based on Speciate 4.2 profile 8751 and MOVES2010b: Additional Toxics Added to MOVES, EPA-420-B-029a, May 2012

[4] This category includes industrial processes not included in NPRI, residential heating (natural gas and oil), agriculture, etc

[5] Speciation of PAH was based on the study of Hytone et al, 2009

Source type	Sub-category	Cumene	Cyclohexane	Ethylbenzene	Formaldehyde	Hexane	Naphthalene	Pentane	Toluene	Trimethylbenzene	Xylenes
NPRI - Upstream Oil and Gas [1]		0	1	14	132	39	0	0	8	37	5
EC - Small Upstream Oil and Gas											
	Combustion-flare						0.15		0.03		0.01
	Combustion-diesel [2]		0.27	0.60	28.41		0.79	2.37	5.07	1.45	
	Combustion-propane										
	Dehydrator										
	Leak										
	Load					47.00		101.00	14.00		
	Tank					512.95		947.98			
	Vent										
	Total	**0**	**0**	**1**	**28**	**560**	**1**	**1,051**	**19**	**1**	**0**
NPRI - Pulp and Paper/wood industry [1]		0.26	4.56	1.66	50.54	23.22	0.10	25.70	11.82	0.28	0.35
NPRI - Coal and mining		0.12	0.41		3.31	1.51	0.06	1.66	1.79	0.07	0.59
EC - Mobile On Road											
	Exhaust [3]	2.43	3.67	32.67	27.69	26.94	0.65	1.32	146.39	38.73	160.06
	Evaporative		0.66		1.21	27.08		75.16	117.79	22.45	113.95
	Total	**2.43**	**4.33**	**32.67**	**28.90**	**54.02**	**0.65**	**76.48**	**264.18**	**61.19**	**274.01**
EC - Non Road											
	Miscellaneous [4]	26.36	62.43	45.09	35.15	95.03	12.49	49.25	141.51	2.77	107.16
	Petroleum storage and transportation	0.14	0.70	2.13		9.40		3.84	6.22	1.66	
	Industrial utilization of solvent		0.35	1.06		6.03			59.46	0.24	44.92
	aerosol coating	0.04	0.31	6.58		1.40	0.09		48.57	1.66	42.38
	fuelwood combustion [5]			7.64	388.90		75.78	1.57	52.74		
	Offroad and Rail [2]	0.61	2.97	3.57		21.98	0.35	93.81	38.09	5.54	
	Other Non-Road	3.09	7.33	5.29	12.62	11.15	1.47	5.78	16.61	0.33	5.70
	Total	**30.24**	**74.09**	**71.38**	**436.67**	**144.98**	**90.17**	**154.25**	**363.20**	**12.20**	**200.16**
Total for all categories		**33.05**	**84.29**	**119.83**	**680.24**	**823.13**	**91.92**	**1,309.92**	**668.39**	**112.36**	**479.99**

Notes:

[1] VOC from the NPRI Upstream Oil and Gas and the paper industry was speciated further if not included in NPRI data base, based on speciation provided by (EC January 2013)

[2] Speciation of PAH was based on Speciate4.2 profile 4674 and USEPA AP42 Table 3.3-2.

[3] Speciation of PAH was based on Speciate 4.2 profile 8751 and MOVES2010b: Additional Toxics Added to MOVES, EPA-420-B-029a, May 2012

[4] This category includes industrial processes not included in NPRI, residential heating (natural gas and oil), agriculture, etc

[5] Speciation of PAH was based on the study of Hytone et al, 2009

Source type	Sub-category	7,12-dimethylbenzene(a)anthracene	dibenz(a,h)anthracene	benzo(a)anthracene	benzo(b)fluoranthene	benzo(k)fluoranthene	chrysene	fluoranthene	indeno(1,2,3,c,d)pyrene	phenanthrene
NPRI - Upstream Oil and Gas [1]				0.00	0.00	0.00	0.01	0.05	0.00	0.11
EC - Small Upstream Oil and Gas										
	Combustion-flare						0.01	0.03		0.07
	Combustion-diesel [2]			0.00	0.00	0.00	0.00	0.07	0.00	0.12
	Combustion-propane									
	Dehydrator									
	Leak									
	Load									
	Tank									
	Vent									
	Total	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
NPRI - Pulp and Paper/wood industry [1]				0.04	0.01	0.01	0.04	0.25	0.00	1.14
NPRI - Coal and mining										
EC - Mobile On Road										
	Exhaust [3]			0.08			0.04	1.03		3.29
	Evaporative									
	Total	**0.00**	**0.00**	**0.08**	**0.00**	**0.00**	**0.04**	**1.03**	**0.00**	**3.29**
EC - Non Road										
	Miscellaneous [4]						0.69	0.69		
	Petroleum storage and transportation									
	Industrial utilization of solvent									
	aerosol coating									
	fuelwood combustion [5]			0.14	0.04	0.05	0.18	1.02	0.01	5.24
	Offroad and Rail [2]									
	Other Non-Road						0.08	0.08		0.49
	Total	**0.00**	**0.00**	**0.14**	**0.04**	**0.05**	**0.96**	**1.79**	**0.01**	**5.73**
Total for all categories				**0.25**	**0.05**	**0.06**	**1.05**	**3.22**	**0.02**	**10.46**

Notes:

[1] VOC from the NPRI Upstream Oil and Gas and the paper industry was speciated further if not included in NPRI data base, based on speciation provided by (EC January 2013)

[2] Speciation of PAH was based on Speciate4.2 profile 4674 and USEPA AP42 Table 3.3-2.

[3] Speciation of PAH was based on Speciate 4.2 profile 8751 and MOVES2010b: Additional Toxics Added to MOVES, EPA-420-B-029a, May 2012

[4] This category includes industrial processes not included in NPRI, residential heating (natural gas and oil), agriculture, etc

[5] Speciation of PAH was based on the study of Hytone et al, 2009

Table A2 : Maximum[1] Air Quality Modelling Results for the Upstream Oil and Gas Sources (µg/m^3)

Representative_Receptor	1 H SO_2	24 H SO_2	Ann SO_2	1 H $PM_{2.5}$	24 H $PM_{2.5}$	Ann $PM_{2.5}$	1 H H_2S	24 H H_2S	Ann H_2S	1 H VOC	24 H VOC	Ann VOC
MPOI	1.06E+03	1.86E+02	1.54E+01	2.63E+01	1.00E+01	1.91E+00	2.64E+01	5.31E+00	1.03E+00	2.69E+03	6.32E+02	1.30E+02
MPOI Location (Receptor X m)	*-76.875*	*-73*	*-101*	*-5.625*	*-5.625*	*-5.625*	*-76.375*	*-76.375*	*-76.375*	*-49*	*-47*	*-47*
MPOI Location (Receptor Y m)	*146.875*	*157*	*163*	*120.625*	*120.625*	*120.625*	*147.125*	*148.125*	*148.125*	*155*	*157*	*157*
Tumbler Ridge [2]	7.01E+00	1.97E+00	1.17E-01	6.44E-02	2.49E-02	1.58E-03	4.93E-02	1.15E-02	5.84E-04	8.76E+00	2.86E+00	1.21E-01
Kelly Lake [3]	1.10E+01	1.63E+00	1.35E-01	1.38E-01	2.01E-02	2.39E-03	2.78E-01	2.50E-02	1.03E-03	1.41E+01	1.91E+00	1.02E-01
Charlie Lake (Community)	3.65E+01	6.72E+00	7.27E-01	4.53E-01	2.39E-01	2.27E-02	3.50E-01	7.20E-02	6.41E-03	6.83E+01	3.99E+01	3.32E+00
Tomslake	9.78E+00	1.99E+00	2.70E-01	1.07E+00	8.59E-02	8.83E-03	8.17E-02	1.00E-02	1.04E-03	9.37E+00	3.23E+00	2.79E-01
Lone Prairie	1.89E+01	4.44E+00	6.23E-01	1.05E-01	5.09E-02	4.82E-03	6.72E-02	1.67E-02	1.97E-03	1.29E+01	5.10E+00	4.75E-01
Pine Valley	9.03E+01	7.69E+00	3.56E-01	4.51E-01	8.68E-02	5.68E-03	3.34E-01	5.05E-02	2.47E-03	2.67E+01	3.49E+00	3.24E-01
Chetwynd	5.37E+01	8.87E+00	1.25E+00	1.01E-01	5.70E-02	6.27E-03	1.88E-01	3.33E-02	4.02E-03	1.59E+01	6.49E+00	7.18E-01
Pouce Coupe	1.23E+01	2.34E+00	3.88E-01	2.06E-01	5.31E-02	6.21E-03	1.10E-01	1.81E-02	1.62E-03	1.80E+01	5.72E+00	4.12E-01
East Pine	2.15E+01	3.14E+00	7.18E-01	2.27E-01	9.65E-02	8.28E-03	8.82E-02	2.52E-02	2.58E-03	1.70E+01	8.40E+00	6.93E-01
Arras	1.53E+01	4.14E+00	5.69E-01	2.06E-01	8.11E-02	8.06E-03	1.61E-01	2.73E-02	2.12E-03	2.96E+01	1.12E+01	6.43E-01
Dawsons Creek	1.72E+01	3.09E+00	5.08E-01	3.44E-01	6.59E-02	7.84E-03	1.58E-01	2.15E-02	2.07E-03	2.88E+01	9.51E+00	5.70E-01
WEST MOBERLY LAKE 168A	4.27E+01	4.46E+00	4.27E-01	1.70E-01	5.48E-02	5.51E-03	9.77E-02	2.61E-02	2.14E-03	2.28E+01	6.71E+00	5.58E-01
Moberly Lake	1.74E+01	3.48E+00	3.93E-01	1.68E-01	5.07E-02	5.54E-03	7.10E-02	2.41E-02	2.07E-03	2.21E+01	6.79E+00	5.62E-01
EAST MOBERLY LAKE 169	3.63E+01	6.25E+00	6.73E-01	1.59E-01	6.60E-02	6.81E-03	2.33E-01	2.87E-02	2.60E-03	2.31E+01	8.62E+00	7.14E-01
Rolla	3.41E+01	4.26E+00	6.34E-01	4.55E-01	7.23E-02	1.12E-02	3.09E-01	4.52E-02	3.25E-03	2.75E+01	9.73E+00	6.60E-01
Doe River	3.04E+01	4.41E+00	7.64E-01	2.12E-01	7.36E-02	1.41E-02	1.33E-01	3.94E-02	4.30E-03	2.35E+01	1.03E+01	9.49E-01
Hudsons Hope	4.49E+01	4.89E+00	4.35E-01	1.55E-01	6.86E-02	7.38E-03	1.06E-01	2.74E-02	2.44E-03	2.53E+01	1.00E+01	7.61E-01
Taylor	3.27E+02	3.53E+01	9.80E-01	4.71E+00	6.72E-01	1.47E-01	3.95E-01	6.56E-02	5.07E-03	3.73E+02	1.34E+02	2.94E+01
Fort St. John	3.65E+02	2.62E+01	1.31E+00	4.28E+00	8.22E-01	1.85E-01	2.11E+00	2.65E-01	8.53E-03	2.34E+02	9.07E+01	1.28E+01
Pineview	7.79E+01	9.59E+00	8.23E-01	1.44E+00	5.67E-01	7.02E-02	4.65E-01	1.12E-01	7.60E-03	2.86E+02	8.66E+01	1.55E+01
Goodlow	5.62E+01	8.22E+00	1.11E+00	3.62E+00	5.42E-01	5.46E-02	2.60E+00	3.93E-01	2.61E-02	3.67E+02	6.09E+01	1.16E+01
HALFWAY RIVER 168	4.75E+01	5.09E+00	6.80E-01	4.32E-01	1.62E-01	4.19E-02	2.34E-01	4.55E-02	3.06E-03	6.17E+01	2.56E+01	4.67E+00
Rose Prairie	2.12E+01	4.93E+00	6.07E-01	1.17E+00	7.23E-01	1.19E-01	3.87E-01	6.18E-02	6.66E-03	1.62E+02	8.84E+01	1.27E+01
DOIG RIVER 206	1.19E+02	1.37E+01	9.26E-01	2.91E+00	3.06E-01	5.58E-02	3.12E-01	6.28E-02	9.06E-03	9.92E+01	3.15E+01	6.08E+00
BLUEBERRY RIVER NO. 205	7.26E+01	1.46E+01	1.01E+00	2.34E+00	4.31E-01	3.75E-02	1.03E+00	2.01E-01	7.91E-03	2.76E+02	1.03E+02	1.70E+01
BLUEBERRY RIVER AND DOIG RIVER 204	1.29E+01	3.61E+00	5.10E-01	1.18E+00	5.74E-01	3.63E-02	2.22E-01	5.22E-02	4.41E-03	3.17E+02	1.73E+02	9.72E+00
Wonowon	1.80E+01	3.80E+00	3.78E-01	2.71E-01	1.13E-01	1.71E-02	1.36E-01	6.07E-02	2.50E-03	6.09E+01	1.45E+01	2.80E+00
Buick	1.77E+01	3.86E+00	5.75E-01	1.13E+00	1.84E-01	2.32E-02	3.29E-01	6.00E-02	4.31E-03	1.27E+02	3.65E+01	7.06E+00
Charlie Lake (Waterbody)	4.02E+01	5.82E+00	6.67E-01	4.81E-01	2.54E-01	2.55E-02	4.24E-01	8.91E-02	7.54E-03	7.16E+01	3.85E+01	3.64E+00

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H Acet.	24 H Acet.	Ann Acet.	1 H Acro.	24 H Acro.	Ann Acro.	1 H Ben.	24 H Ben.	Ann Ben.
MPOI	9.58E+00	1.70E+00	1.84E-01	7.83E-01	1.39E-01	1.50E-02	2.48E+01	4.68E+00	8.80E-01
MPOI Location (Receptor X m)	*-59*	*-33*	*-33*	*-59*	*-33*	*-33*	*-5.625*	*-5.125*	*-49*
MPOI Location (Receptor Y m)	*169*	*157*	*157*	*169*	*157*	*157*	*122.125*	*120.125*	*155*
Tumbler Ridge [2]	2.08E-02	6.90E-03	3.18E-04	1.70E-03	5.64E-04	2.60E-05	2.53E-02	8.38E-03	3.05E-04
Kelly Lake [3]	1.08E-02	4.03E-03	1.74E-04	8.86E-04	3.29E-04	1.42E-05	1.23E-02	4.88E-03	1.64E-04
Charlie Lake (Community)	1.26E-01	6.40E-02	6.07E-03	1.03E-02	5.23E-03	4.97E-04	1.76E-01	1.04E-01	8.33E-03
Tomslake	1.91E-02	7.09E-03	6.68E-04	1.56E-03	5.80E-04	5.47E-05	2.47E-02	8.52E-03	5.73E-04
Lone Prairie	2.71E-02	1.25E-02	1.26E-03	2.21E-03	1.02E-03	1.03E-04	3.97E-02	1.66E-02	1.01E-03
Pine Valley	2.28E-02	8.69E-03	8.00E-04	1.86E-03	7.11E-04	6.55E-05	3.45E-02	9.24E-03	7.24E-04
Chetwynd	3.42E-02	1.57E-02	1.49E-03	2.80E-03	1.28E-03	1.21E-04	4.31E-02	1.90E-02	1.27E-03
Pouce Coupe	3.64E-02	1.50E-02	1.03E-03	2.98E-03	1.23E-03	8.46E-05	4.84E-02	1.52E-02	1.01E-03
East Pine	4.19E-02	2.05E-02	1.71E-03	3.43E-03	1.67E-03	1.40E-04	5.97E-02	2.75E-02	1.78E-03
Arras	6.56E-02	2.72E-02	1.68E-03	5.36E-03	2.22E-03	1.37E-04	7.68E-02	2.92E-02	1.67E-03
Dawsons Creek	5.58E-02	2.04E-02	1.36E-03	4.57E-03	1.67E-03	1.12E-04	7.73E-02	2.55E-02	1.42E-03
WEST MOBERLY LAKE 168A	4.79E-02	1.48E-02	1.37E-03	3.91E-03	1.21E-03	1.12E-04	6.24E-02	1.94E-02	1.40E-03
Moberly Lake	4.72E-02	1.50E-02	1.39E-03	3.86E-03	1.23E-03	1.14E-04	6.09E-02	1.97E-02	1.42E-03
EAST MOBERLY LAKE 169	5.18E-02	1.94E-02	1.73E-03	4.24E-03	1.59E-03	1.42E-04	6.31E-02	2.50E-02	1.78E-03
Rolla	6.56E-02	2.18E-02	1.57E-03	5.37E-03	1.78E-03	1.28E-04	6.90E-02	2.51E-02	1.69E-03
Doe River	5.35E-02	2.36E-02	2.26E-03	4.37E-03	1.93E-03	1.85E-04	6.10E-02	2.86E-02	2.42E-03
Hudsons Hope	4.98E-02	2.12E-02	2.01E-03	4.07E-03	1.73E-03	1.65E-04	6.65E-02	2.62E-02	1.93E-03
Taylor	9.65E-01	2.07E-01	4.59E-02	7.89E-02	1.69E-02	3.76E-03	2.02E+00	4.54E-01	1.89E-01
Fort St. John	2.52E-01	9.29E-02	1.16E-02	2.07E-02	7.59E-03	9.52E-04	9.23E-01	2.53E-01	2.97E-02
Pineview	2.14E-01	9.46E-02	1.17E-02	1.75E-02	7.74E-03	9.59E-04	1.01E+00	2.03E-01	4.02E-02
Goodlow	2.39E-01	4.60E-02	8.65E-03	1.96E-02	3.76E-03	7.07E-04	3.36E+00	4.86E-01	5.64E-02
HALFWAY RIVER 168	1.42E-01	4.52E-02	6.07E-03	1.16E-02	3.70E-03	4.96E-04	2.06E-01	5.82E-02	9.05E-03
Rose Prairie	2.24E-01	1.06E-01	1.52E-02	1.83E-02	8.64E-03	1.24E-03	5.03E-01	1.93E-01	2.67E-02
DOIG RIVER 206	6.67E-01	1.54E-01	2.28E-02	5.45E-02	1.26E-02	1.87E-03	3.26E-01	7.41E-02	1.47E-02
BLUEBERRY RIVER NO. 205	2.42E+00	3.16E-01	4.44E-02	1.98E-01	2.59E-02	3.63E-03	8.82E-01	3.02E-01	5.00E-02
BLUEBERRY RIVER AND DOIG RIVER 204	1.14E+00	2.29E-01	2.01E-02	9.29E-02	1.87E-02	1.64E-03	1.06E+00	5.39E-01	2.91E-02
Wonowon	2.37E-01	4.82E-02	1.05E-02	1.94E-02	3.94E-03	8.59E-04	1.65E-01	3.72E-02	6.65E-03
Buick	6.37E-01	1.09E-01	1.81E-02	5.21E-02	8.89E-03	1.48E-03	3.04E-01	9.87E-02	1.19E-02
Charlie Lake (Waterbody)	1.16E-01	6.74E-02	6.18E-03	9.48E-03	5.51E-03	5.05E-04	2.07E-01	9.72E-02	9.29E-03

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H bap.	24 H bap.	Ann bap.	1 H b13	24 H b13	Ann b13	1 H cum.	24 H cum.	Ann cum.
MPOI	2.23E-04	6.21E-05	1.71E-05	7.11E-02	1.26E-02	1.36E-03	7.10E-03	1.32E-03	3.39E-04
MPOI Location (Receptor X m)	*-49*	*-49*	*-49*	*-59*	*-33*	*-33*	*-41.125*	*-41.625*	*-40.875*
MPOI Location (Receptor Y m)	*155*	*155*	*155*	*169*	*157*	*157*	*100.125*	*99.375*	*99.375*
Tumbler Ridge [2]	7.06E-07	2.57E-07	1.60E-08	1.55E-04	5.17E-05	2.38E-06	2.24E-06	8.53E-07	2.88E-08
Kelly Lake [3]	7.38E-06	1.03E-06	4.39E-08	8.17E-05	3.03E-05	1.31E-06	3.02E-06	4.84E-07	1.98E-08
Charlie Lake (Community)	5.58E-06	2.97E-06	2.56E-07	9.40E-04	4.79E-04	4.55E-05	5.23E-05	1.15E-05	5.21E-07
Tomslake	1.72E-06	3.11E-07	2.79E-08	1.47E-04	5.32E-05	5.03E-06	7.12E-06	1.90E-06	7.76E-08
Lone Prairie	1.08E-06	5.14E-07	4.42E-08	2.04E-04	9.43E-05	9.47E-06	5.34E-06	2.02E-06	1.06E-07
Pine Valley	3.14E-06	4.77E-07	3.64E-08	1.71E-04	6.56E-05	6.01E-06	5.13E-06	1.28E-06	7.95E-08
Chetwynd	1.16E-06	5.90E-07	6.09E-08	2.57E-04	1.18E-04	1.11E-05	9.94E-06	2.15E-06	1.41E-07
Pouce Coupe	1.32E-06	4.53E-07	3.96E-08	2.83E-04	1.14E-04	7.83E-06	1.46E-05	4.38E-06	1.69E-07
East Pine	2.36E-06	9.27E-07	7.81E-08	3.18E-04	1.53E-04	1.29E-05	1.19E-05	4.45E-06	1.90E-07
Arras	1.91E-06	7.51E-07	6.23E-08	4.99E-04	2.09E-04	1.27E-05	5.22E-05	8.43E-06	2.58E-07
Dawsons Creek	1.98E-06	7.01E-07	5.35E-08	4.39E-04	1.60E-04	1.03E-05	2.97E-05	9.95E-06	2.48E-07
WEST MOBERLY LAKE 168A	1.68E-06	6.06E-07	5.32E-08	3.59E-04	1.12E-04	1.04E-05	1.44E-05	3.16E-06	1.70E-07
Moberly Lake	1.73E-06	5.81E-07	5.36E-08	3.54E-04	1.12E-04	1.05E-05	1.48E-05	3.14E-06	1.74E-07
EAST MOBERLY LAKE 169	1.73E-06	6.26E-07	6.83E-08	3.89E-04	1.46E-04	1.31E-05	1.74E-05	4.26E-06	2.26E-07
Rolla	1.94E-06	6.88E-07	6.04E-08	5.00E-04	1.66E-04	1.19E-05	2.87E-05	5.19E-06	3.15E-07
Doe River	1.78E-06	6.88E-07	7.96E-08	3.97E-04	1.79E-04	1.73E-05	5.22E-05	7.51E-06	5.59E-07
Hudsons Hope	1.79E-06	7.73E-07	6.89E-08	3.69E-04	1.62E-04	1.52E-05	2.08E-05	6.45E-06	2.35E-07
Taylor	8.45E-06	2.69E-06	2.58E-07	7.51E-03	2.30E-03	5.42E-04	5.82E-03	1.32E-03	3.20E-04
Fort St. John	1.75E-05	5.86E-06	5.88E-07	2.15E-03	6.95E-04	8.80E-05	9.65E-04	8.53E-05	2.99E-06
Pineview	1.93E-05	7.30E-06	1.11E-06	1.59E-03	7.03E-04	8.75E-05	7.90E-05	9.27E-06	5.86E-07
Goodlow	3.71E-06	9.92E-07	1.98E-07	1.78E-03	3.42E-04	6.46E-05	2.95E-05	5.80E-06	4.36E-07
HALFWAY RIVER 168	4.81E-06	2.03E-06	5.21E-07	1.05E-03	3.36E-04	4.51E-05	1.33E-05	4.00E-06	1.53E-07
Rose Prairie	1.66E-05	9.34E-06	1.53E-06	1.66E-03	7.86E-04	1.13E-04	2.21E-05	3.09E-06	1.97E-07
DOIG RIVER 206	7.09E-06	2.60E-06	6.48E-07	4.95E-03	1.14E-03	1.70E-04	1.73E-05	2.34E-06	1.63E-07
BLUEBERRY RIVER NO. 205	2.14E-05	7.10E-06	1.27E-06	1.79E-02	2.35E-03	3.30E-04	1.54E-05	3.54E-06	1.01E-07
BLUEBERRY RIVER AND DOIG RIVER 204	2.15E-05	1.15E-05	6.32E-07	8.43E-03	1.70E-03	1.49E-04	1.35E-05	2.79E-06	1.09E-07
Wonowon	3.41E-06	1.10E-06	1.84E-07	1.76E-03	3.57E-04	7.80E-05	7.33E-06	1.56E-06	6.08E-08
Buick	7.29E-06	2.11E-06	2.01E-07	4.73E-03	8.07E-04	1.35E-04	7.32E-06	1.29E-06	7.00E-08
Charlie Lake (Waterbody)	6.40E-06	3.11E-06	2.96E-07	8.65E-04	5.03E-04	4.61E-05	2.88E-05	6.85E-06	3.19E-07

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H cyc.	24 H cyc.	Ann cyc.	1 H eb.	24 H eb.	Ann eb.	1 H form.	24 H form.	Ann form.
MPOI	1.19E+00	2.08E-01	4.03E-02	1.13E-01	2.31E-02	3.13E-03	1.90E+02	4.43E+01	8.06E+00
MPOI Location (Receptor X m)	*-5.125*	*-5.625*	*-71*	*-54.625*	*-55.875*	*-55.125*	*-101.375*	*-101.375*	*-101.125*
MPOI Location (Receptor Y m)	*117.875*	*118.125*	*153*	*76.125*	*75.625*	*75.875*	*154.375*	*154.875*	*154.875*
Tumbler Ridge [2]	3.21E-04	9.91E-05	5.01E-06	3.50E-04	1.28E-04	5.82E-06	7.62E-02	2.39E-02	1.49E-03
Kelly Lake [3]	2.59E-04	7.23E-05	2.91E-06	2.36E-04	8.17E-05	3.15E-06	6.85E-01	6.94E-02	6.18E-03
Charlie Lake (Community)	4.67E-03	1.13E-03	1.00E-04	1.44E-03	7.72E-04	8.06E-05	2.28E-01	9.03E-02	8.70E-03
Tomslake	6.10E-04	1.99E-04	1.03E-05	4.59E-04	1.72E-04	1.22E-05	1.37E+00	1.99E-01	1.60E-02
Lone Prairie	4.73E-04	2.27E-04	1.76E-05	6.53E-04	3.03E-04	2.31E-05	1.04E-01	5.59E-02	4.13E-03
Pine Valley	5.25E-04	1.54E-04	1.29E-05	3.90E-04	1.63E-04	1.36E-05	8.15E-02	2.80E-02	2.20E-03
Chetwynd	6.87E-04	2.65E-04	2.18E-05	6.12E-04	2.75E-04	2.72E-05	1.02E-01	5.07E-02	4.96E-03
Pouce Coupe	1.22E-03	3.82E-04	1.90E-05	1.14E-03	2.42E-04	2.40E-05	6.50E-01	1.03E-01	1.07E-02
East Pine	8.30E-04	3.59E-04	2.75E-05	1.26E-03	5.03E-04	3.88E-05	2.95E-01	1.14E-01	7.45E-03
Arras	3.28E-03	6.51E-04	2.94E-05	1.20E-03	4.27E-04	3.71E-05	2.85E-01	7.55E-02	7.91E-03
Dawsons Creek	2.18E-03	7.44E-04	2.64E-05	3.50E-03	4.50E-04	3.40E-05	6.80E-01	1.26E-01	8.99E-03
WEST MOBERLY LAKE 168A	1.06E-03	3.26E-04	2.50E-05	1.13E-03	2.60E-04	2.35E-05	1.90E-01	4.99E-02	3.56E-03
Moberly Lake	1.07E-03	3.26E-04	2.53E-05	1.05E-03	2.66E-04	2.39E-05	1.58E-01	5.14E-02	3.63E-03
EAST MOBERLY LAKE 169	1.23E-03	3.97E-04	3.18E-05	1.01E-03	3.29E-04	3.05E-05	1.81E-01	6.94E-02	4.66E-03
Rolla	1.93E-03	5.82E-04	3.34E-05	9.15E-03	1.77E-03	7.14E-05	2.71E+00	3.56E-01	1.44E-02
Doe River	2.73E-03	5.88E-04	5.37E-05	3.36E-03	6.33E-04	1.04E-04	1.14E+00	1.50E-01	1.99E-02
Hudsons Hope	1.39E-03	5.08E-04	3.58E-05	8.54E-04	3.84E-04	3.06E-05	1.41E-01	7.14E-02	4.34E-03
Taylor	2.36E-01	2.19E-02	4.09E-03	4.90E-02	7.00E-03	1.08E-03	1.05E+01	2.76E+00	6.36E-01
Fort St. John	7.53E-02	4.66E-03	2.54E-04	5.37E-03	1.21E-03	1.57E-04	1.90E+00	2.15E-01	1.67E-02
Pineview	4.82E-03	1.25E-03	1.36E-04	2.47E-03	1.08E-03	1.45E-04	3.41E-01	9.04E-02	1.14E-02
Goodlow	1.39E-01	1.62E-02	1.01E-03	2.73E-03	5.19E-04	1.10E-04	1.42E+00	2.01E-01	2.75E-02
HALFWAY RIVER 168	2.50E-03	6.68E-04	5.73E-05	1.62E-03	5.31E-04	7.19E-05	5.15E-01	9.57E-02	9.01E-03
Rose Prairie	8.05E-03	1.26E-03	1.49E-04	2.52E-03	1.21E-03	1.78E-04	4.63E-01	9.75E-02	1.36E-02
DOIG RIVER 206	3.83E-03	9.42E-04	1.74E-04	7.52E-03	1.73E-03	2.62E-04	3.73E-01	8.81E-02	1.62E-02
BLUEBERRY RIVER NO. 205	7.96E-02	1.12E-02	5.76E-04	2.72E-02	3.56E-03	5.04E-04	1.69E+00	3.94E-01	3.37E-02
BLUEBERRY RIVER AND DOIG RIVER 204	7.07E-03	3.08E-03	2.02E-04	1.28E-02	2.61E-03	2.30E-04	6.50E-01	1.97E-01	1.61E-02
Wonowon	2.28E-03	8.24E-04	7.43E-05	2.67E-03	5.43E-04	1.20E-04	7.88E-01	1.28E-01	9.86E-03
Buick	1.49E-02	2.37E-03	1.60E-04	7.17E-03	1.22E-03	2.07E-04	5.48E-01	1.58E-01	1.60E-02
Charlie Lake (Waterbody)	3.06E-03	1.03E-03	9.12E-05	1.36E-03	7.99E-04	7.85E-05	2.49E-01	8.55E-02	7.97E-03

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H hex.	24 H hex.	Ann hex.	1 H naph.	24 H naph.	Ann naph.	1 H pen.	24 H pen.	Ann pen.
MPOI	1.67E+02	3.87E+01	7.28E+00	1.42E-01	2.72E-02	3.23E-03	3.15E+02	7.24E+01	1.38E+01
MPOI Location (Receptor X m)	*-49*	*-49*	*-47*	*-59*	*-33*	*-33*	*-49*	*-49*	*-47*
MPOI Location (Receptor Y m)	*155*	*157*	*157*	*169*	*157*	*157*	*155*	*157*	*157*
Tumbler Ridge [2]	3.47E-01	1.10E-01	3.60E-03	3.89E-04	1.29E-04	5.71E-06	6.46E-01	2.03E-01	6.47E-03
Kelly Lake [3]	1.38E-01	5.84E-02	1.88E-03	1.99E-04	7.43E-05	3.11E-06	2.41E-01	1.04E-01	3.35E-03
Charlie Lake (Community)	2.88E+00	1.54E+00	1.15E-01	2.42E-03	1.30E-03	1.18E-04	5.26E+00	2.80E+00	2.10E-01
Tomslake	3.31E-01	1.15E-01	6.14E-03	3.69E-04	1.32E-04	1.17E-05	6.09E-01	2.11E-01	1.10E-02
Lone Prairie	4.86E-01	1.72E-01	1.11E-02	5.19E-04	2.37E-04	2.21E-05	8.79E-01	3.16E-01	1.98E-02
Pine Valley	4.39E-01	1.26E-01	8.47E-03	4.38E-04	1.50E-04	1.42E-05	7.63E-01	2.31E-01	1.51E-02
Chetwynd	5.77E-01	2.26E-01	1.42E-02	6.33E-04	2.94E-04	2.62E-05	1.06E+00	4.12E-01	2.53E-02
Pouce Coupe	6.17E-01	1.90E-01	1.11E-02	7.08E-04	2.77E-04	1.87E-05	1.13E+00	3.48E-01	1.99E-02
East Pine	6.83E-01	3.09E-01	1.74E-02	8.47E-04	3.80E-04	3.23E-05	1.25E+00	5.58E-01	3.12E-02
Arras	1.10E+00	3.84E-01	1.80E-02	1.20E-03	4.98E-04	3.07E-05	2.04E+00	7.07E-01	3.23E-02
Dawsons Creek	9.71E-01	3.06E-01	1.50E-02	1.09E-03	3.94E-04	2.51E-05	1.78E+00	5.58E-01	2.68E-02
WEST MOBERLY LAKE 168A	8.25E-01	2.40E-01	1.67E-02	8.96E-04	2.76E-04	2.48E-05	1.44E+00	4.18E-01	3.00E-02
Moberly Lake	7.97E-01	2.43E-01	1.69E-02	8.72E-04	2.78E-04	2.52E-05	1.42E+00	4.22E-01	3.04E-02
EAST MOBERLY LAKE 169	8.67E-01	3.06E-01	2.11E-02	9.61E-04	3.62E-04	3.13E-05	1.53E+00	5.32E-01	3.78E-02
Rolla	8.59E-01	3.49E-01	1.70E-02	1.21E-03	3.96E-04	2.92E-05	1.58E+00	6.46E-01	3.01E-02
Doe River	8.75E-01	3.60E-01	2.25E-02	9.77E-04	4.34E-04	4.20E-05	1.61E+00	6.38E-01	3.95E-02
Hudsons Hope	9.86E-01	3.86E-01	2.38E-02	9.31E-04	3.85E-04	3.59E-05	1.78E+00	6.93E-01	4.30E-02
Taylor	6.49E+00	2.15E+00	4.57E-01	1.50E-02	3.77E-03	8.89E-04	1.12E+01	4.17E+00	9.10E-01
Fort St. John	9.83E+00	3.55E+00	3.70E-01	4.80E-03	1.92E-03	2.16E-04	1.81E+01	6.52E+00	6.86E-01
Pineview	1.58E+01	2.75E+00	5.85E-01	5.41E-03	2.23E-03	3.02E-04	2.96E+01	5.08E+00	1.09E+00
Goodlow	2.08E+01	3.01E+00	3.49E-01	3.65E-03	7.22E-04	1.49E-04	4.18E+00	9.68E-01	1.19E-01
HALFWAY RIVER 168	2.83E+00	8.37E-01	8.99E-02	2.45E-03	8.77E-04	1.40E-04	5.32E+00	1.54E+00	1.67E-01
Rose Prairie	7.61E+00	2.67E+00	3.27E-01	5.25E-03	2.54E-03	3.99E-04	1.41E+01	4.96E+00	6.08E-01
DOIG RIVER 206	6.00E+00	1.08E+00	2.02E-01	1.06E-02	2.57E-03	3.98E-04	1.12E+01	2.01E+00	3.72E-01
BLUEBERRY RIVER NO. 205	1.51E+01	4.95E+00	7.68E-01	3.60E-02	4.96E-03	7.44E-04	2.83E+01	9.23E+00	1.44E+00
BLUEBERRY RIVER AND DOIG RIVER 204	1.94E+01	9.31E+00	4.70E-01	1.72E-02	4.72E-03	3.67E-04	3.63E+01	1.74E+01	8.76E-01
Wonowon	2.39E+00	5.27E-01	8.91E-02	3.88E-03	8.09E-04	1.75E-04	4.49E+00	9.82E-01	1.66E-01
Buick	4.28E+00	1.50E+00	9.50E-02	9.72E-03	1.85E-03	2.90E-04	8.03E+00	2.77E+00	1.76E-01
Charlie Lake (Waterbody)	2.91E+00	1.40E+00	1.30E-01	2.43E-03	1.36E-03	1.24E-04	5.47E+00	2.55E+00	2.40E-01

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H tol.	24 H tol.	Ann tol.	1 H tri.	24 H tri.	Ann tri.	1 H xyl.	24 H xyl.	Ann xyl.
MPOI	2.38E+01	3.02E+00	8.75E-01	2.22E+01	7.08E+00	1.42E+00	1.76E+01	2.23E+00	6.46E-01
MPOI Location (Receptor X m)	*-1.625*	*-1.625*	*-1.625*	*-41.625*	*-41.625*	*-41.125*	*-1.625*	*-1.625*	*-1.625*
MPOI Location (Receptor Y m)	*5.625*	*5.625*	*5.625*	*99.625*	*99.375*	*99.375*	*5.625*	*5.625*	*5.625*
Tumbler Ridge [2]	1.20E-02	3.70E-03	1.81E-04	8.75E-03	3.07E-03	1.80E-04	3.81E-03	7.47E-04	3.98E-05
Kelly Lake [3]	1.05E+00	9.45E-02	2.66E-03	1.09E-02	2.00E-03	1.26E-04	7.73E-01	6.92E-02	1.83E-03
Charlie Lake (Community)	6.98E-02	4.05E-02	3.45E-03	2.43E-01	5.51E-02	2.27E-03	3.00E-02	6.72E-03	2.97E-04
Tomslake	6.84E-02	2.15E-02	5.26E-04	3.27E-02	1.05E-02	4.98E-04	4.96E-02	1.49E-02	2.11E-04
Lone Prairie	1.47E-02	6.25E-03	5.31E-04	1.93E-02	7.69E-03	8.63E-04	3.63E-03	1.47E-03	8.13E-05
Pine Valley	1.23E-02	4.33E-03	3.50E-04	7.35E-02	6.95E-03	5.11E-04	2.47E-03	7.76E-04	4.81E-05
Chetwynd	1.70E-02	7.55E-03	6.60E-04	4.38E-02	9.89E-03	1.42E-03	4.40E-03	1.45E-03	8.92E-05
Pouce Coupe	2.78E-02	1.02E-02	5.87E-04	8.49E-02	2.77E-02	1.11E-03	1.69E-02	5.88E-03	1.57E-04
East Pine	2.36E-02	9.82E-03	7.99E-04	4.44E-02	1.38E-02	1.27E-03	5.57E-03	1.90E-03	1.25E-04
Arras	4.31E-02	1.53E-02	8.33E-04	1.67E-01	3.31E-02	1.51E-03	2.13E-02	3.65E-03	1.78E-04
Dawsons Creek	4.44E-02	1.48E-02	7.29E-04	1.33E-01	4.69E-02	1.68E-03	1.53E-02	5.48E-03	1.71E-04
WEST MOBERLY LAKE 168A	2.68E-02	7.24E-03	6.31E-04	5.43E-02	1.14E-02	8.05E-04	7.05E-03	1.43E-03	8.95E-05
Moberly Lake	2.60E-02	7.29E-03	6.39E-04	5.64E-02	1.15E-02	8.16E-04	6.30E-03	1.45E-03	9.12E-05
EAST MOBERLY LAKE 169	2.48E-02	9.28E-03	7.95E-04	6.04E-02	1.63E-02	1.09E-03	7.18E-03	1.90E-03	1.15E-04
Rolla	3.80E-02	1.24E-02	8.08E-04	1.00E+00	1.36E-01	4.06E-03	1.21E-02	2.72E-03	1.93E-04
Doe River	3.77E-02	1.30E-02	1.10E-03	3.91E-01	5.09E-02	6.43E-03	2.11E-02	2.99E-03	2.84E-04
Hudsons Hope	2.57E-02	1.02E-02	8.78E-04	7.54E-02	2.18E-02	1.01E-03	9.06E-03	2.65E-03	1.18E-04
Taylor	2.52E+00	8.93E-01	1.88E-01	2.22E+01	7.08E+00	1.42E+00	2.82E+00	9.01E-01	1.81E-01
Fort St. John	5.86E-01	1.18E-01	1.11E-02	5.29E+00	3.20E-01	1.08E-02	6.73E-01	4.05E-02	1.34E-03
Pineview	4.46E-01	7.32E-02	1.56E-02	3.40E-01	3.16E-02	2.43E-03	4.31E-02	3.94E-03	3.07E-04
Goodlow	6.30E-02	1.27E-02	2.70E-03	1.76E-01	2.24E-02	2.98E-03	1.74E-01	2.04E-02	1.38E-03
HALFWAY RIVER 168	9.75E-02	2.27E-02	3.06E-03	4.70E-02	1.28E-02	7.16E-04	5.61E-03	1.69E-03	9.48E-05
Rose Prairie	2.56E-01	6.79E-02	9.39E-03	6.24E-02	1.22E-02	1.26E-03	9.82E-03	1.64E-03	1.74E-04
DOIG RIVER 206	1.47E-01	2.95E-02	6.63E-03	4.73E-02	9.02E-03	1.29E-03	5.94E-03	1.37E-03	1.36E-04
BLUEBERRY RIVER NO. 205	4.20E-01	1.36E-01	2.32E-02	1.03E-01	1.99E-02	1.97E-03	9.64E-02	1.27E-02	5.40E-04
BLUEBERRY RIVER AND DOIG RIVER 204	4.64E-01	2.36E-01	1.30E-02	4.72E-02	1.14E-02	1.11E-03	7.42E-03	2.65E-03	1.67E-04
Wonowon	7.40E-02	1.68E-02	3.03E-03	2.48E-02	5.08E-03	5.55E-04	3.10E-03	7.44E-04	5.20E-05
Buick	1.32E-01	4.24E-02	4.04E-03	3.21E-02	6.84E-03	8.72E-04	1.80E-02	2.43E-03	1.11E-04
Charlie Lake (Waterbody)	1.04E-01	3.73E-02	3.81E-03	1.11E-01	3.00E-02	1.50E-03	1.35E-02	3.55E-03	2.09E-04

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H 7_12.	24 H 7_12.	Ann 7_12.	1 H dah.	24 H dah.	Ann dah.	1 H baa.	24 H baa.	Ann baa.	1 H bbf.	24 H bbf.	Ann bbf.
MPOI	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.19E-04	1.47E-04	1.98E-05	1.67E-04	3.40E-05	4.60E-06
MPOI Location (Receptor X m)	*-149*	*-149*	*-149*	*-149*	*-149*	*-149*	*-54.625*	*-55.875*	*-55.125*	*-54.625*	*-55.875*	*-55.125*
MPOI Location (Receptor Y m)	*1*	*1*	*1*	*1*	*1*	*1*	*76.125*	*75.625*	*75.875*	*76.125*	*75.625*	*75.875*
Tumbler Ridge [2]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.22E-06	8.14E-07	3.70E-08	5.14E-07	1.88E-07	8.55E-09
Kelly Lake [3]	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.50E-06	5.19E-07	2.01E-08	3.47E-07	1.20E-07	4.63E-09
Charlie Lake (Community)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.16E-06	4.92E-06	5.13E-07	2.11E-06	1.13E-06	1.18E-07
Tomslake	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.91E-06	1.09E-06	7.73E-08	6.73E-07	2.52E-07	1.78E-08
Lone Prairie	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.15E-06	1.92E-06	1.47E-07	9.59E-07	4.45E-07	3.40E-08
Pine Valley	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.48E-06	1.04E-06	8.68E-08	5.73E-07	2.40E-07	2.01E-08
Chetwynd	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.89E-06	1.75E-06	1.73E-07	8.99E-07	4.04E-07	3.99E-08
Pouce Coupe	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.25E-06	1.54E-06	1.52E-07	1.68E-06	3.55E-07	3.52E-08
East Pine	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.98E-06	3.20E-06	2.47E-07	1.85E-06	7.39E-07	5.70E-08
Arras	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.64E-06	2.71E-06	2.36E-07	1.77E-06	6.27E-07	5.45E-08
Dawsons Creek	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.22E-05	2.86E-06	2.16E-07	5.14E-06	6.60E-07	4.99E-08
WEST MOBERLY LAKE 168A	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.19E-06	1.65E-06	1.49E-07	1.66E-06	3.82E-07	3.45E-08
Moberly Lake	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.65E-06	1.69E-06	1.52E-07	1.54E-06	3.90E-07	3.51E-08
EAST MOBERLY LAKE 169	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.39E-06	2.09E-06	1.94E-07	1.48E-06	4.84E-07	4.48E-08
Rolla	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.80E-05	1.12E-05	4.53E-07	1.34E-05	2.60E-06	1.05E-07
Doe River	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.13E-05	4.02E-06	6.62E-07	4.93E-06	9.30E-07	1.53E-07
Hudsons Hope	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.43E-06	2.44E-06	1.95E-07	1.25E-06	5.64E-07	4.49E-08
Taylor	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.11E-04	4.44E-05	6.85E-06	7.20E-05	1.03E-05	1.59E-06
Fort St. John	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.40E-05	7.69E-06	9.97E-07	7.88E-06	1.78E-06	2.30E-07
Pineview	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.57E-05	6.89E-06	9.24E-07	3.63E-06	1.59E-06	2.13E-07
Goodlow	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.74E-05	3.31E-06	7.03E-07	4.02E-06	7.63E-07	1.62E-07
HALFWAY RIVER 168	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.03E-05	3.38E-06	4.58E-07	2.38E-06	7.81E-07	1.06E-07
Rose Prairie	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.61E-05	7.73E-06	1.13E-06	3.71E-06	1.78E-06	2.61E-07
DOIG RIVER 206	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.79E-05	1.10E-05	1.67E-06	1.11E-05	2.54E-06	3.85E-07
BLUEBERRY RIVER NO. 205	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.73E-04	2.27E-05	3.21E-06	4.00E-05	5.23E-06	7.40E-07
BLUEBERRY RIVER AND DOIG RIVER 204	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.14E-05	1.66E-05	1.47E-06	1.88E-05	3.83E-06	3.38E-07
Wonowon	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.70E-05	3.46E-06	7.67E-07	3.92E-06	7.97E-07	1.77E-07
Buick	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.57E-05	7.80E-06	1.32E-06	1.05E-05	1.80E-06	3.04E-07
Charlie Lake (Waterbody)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.66E-06	5.09E-06	4.99E-07	2.00E-06	1.17E-06	1.15E-07

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H bkf.	24 H bkf.	Ann bkf.	1 H chry.	24 H chry.	Ann chry.	1 H fluo.	24 H fluo.	Ann fluo
MPOI	2.61E-04	5.31E-05	7.19E-06	9.68E-04	2.60E-04	7.18E-05	1.28E-02	2.61E-03	4.28E-04
MPOI Location (Receptor X m)	*-54.625*	*-55.875*	*-55.125*	*-49*	*-49*	*-49*	*-54.625*	*-55.875*	*-49*
MPOI Location (Receptor Y m)	*76.125*	*75.625*	*75.875*	*155*	*155*	*155*	*76.125*	*75.625*	*155*
Tumbler Ridge [2]	8.04E-07	2.95E-07	1.34E-08	4.60E-06	1.70E-06	9.45E-08	5.40E-05	1.98E-05	9.92E-07
Kelly Lake [3]	5.43E-07	1.88E-07	7.25E-09	2.96E-05	4.14E-06	1.92E-07	1.57E-04	2.18E-05	1.28E-06
Charlie Lake (Community)	3.31E-06	1.78E-06	1.85E-07	2.94E-05	1.59E-05	1.45E-06	2.75E-04	1.50E-04	1.45E-05
Tomslake	1.05E-06	3.95E-07	2.79E-08	6.90E-06	1.91E-06	1.75E-07	6.38E-05	2.47E-05	1.96E-06
Lone Prairie	1.50E-06	6.96E-07	5.32E-08	6.84E-06	3.64E-06	2.98E-07	8.75E-05	4.49E-05	3.54E-06
Pine Valley	8.97E-07	3.76E-07	3.14E-08	1.32E-05	2.19E-06	2.17E-07	8.57E-05	2.54E-05	2.30E-06
Chetwynd	1.41E-06	6.33E-07	6.25E-08	7.27E-06	3.79E-06	3.85E-07	8.53E-05	4.34E-05	4.34E-06
Pouce Coupe	2.63E-06	5.56E-07	5.51E-08	9.62E-06	3.08E-06	2.84E-07	1.34E-04	3.67E-05	3.54E-06
East Pine	2.89E-06	1.16E-06	8.92E-08	1.25E-05	6.23E-06	5.16E-07	1.69E-04	7.56E-05	6.02E-06
Arras	2.76E-06	9.82E-07	8.53E-08	1.17E-05	4.96E-06	4.43E-07	1.64E-04	6.25E-05	5.49E-06
Dawsons Creek	8.05E-06	1.03E-06	7.81E-08	1.84E-05	4.73E-06	3.92E-07	3.96E-04	6.32E-05	4.96E-06
WEST MOBERLY LAKE 168A	2.60E-06	5.97E-07	5.40E-08	1.22E-05	3.49E-06	3.36E-07	1.60E-04	3.72E-05	3.76E-06
Moberly Lake	2.41E-06	6.11E-07	5.50E-08	1.21E-05	3.37E-06	3.40E-07	1.53E-04	3.83E-05	3.82E-06
EAST MOBERLY LAKE 169	2.31E-06	7.57E-07	7.01E-08	1.07E-05	4.16E-06	4.29E-07	1.35E-04	4.91E-05	4.80E-06
Rolla	2.10E-05	4.07E-06	1.64E-07	4.82E-05	9.37E-06	6.15E-07	1.03E-03	2.00E-04	9.32E-06
Doe River	7.71E-06	1.45E-06	2.40E-07	1.93E-05	4.23E-06	8.64E-07	3.88E-04	7.19E-05	1.34E-05
Hudsons Hope	1.96E-06	8.82E-07	7.03E-08	1.01E-05	4.48E-06	4.36E-07	1.17E-04	5.24E-05	4.90E-06
Taylor	1.13E-04	1.61E-05	2.48E-06	2.59E-04	3.81E-05	6.57E-06	5.54E-03	7.91E-04	1.27E-04
Fort St. John	1.23E-05	2.78E-06	3.60E-07	7.21E-05	2.86E-05	2.94E-06	6.32E-04	2.35E-04	2.57E-05
Pineview	5.68E-06	2.49E-06	3.34E-07	9.01E-05	3.49E-05	5.22E-06	6.83E-04	2.75E-04	3.98E-05
Goodlow	6.29E-06	1.20E-06	2.54E-07	1.96E-05	5.57E-06	1.37E-06	3.30E-04	7.02E-05	1.66E-05
HALFWAY RIVER 168	3.73E-06	1.22E-06	1.65E-07	2.22E-05	1.09E-05	2.43E-06	2.47E-04	1.03E-04	1.84E-05
Rose Prairie	5.81E-06	2.79E-06	4.10E-07	7.97E-05	4.35E-05	7.06E-06	6.33E-04	3.29E-04	5.22E-05
DOIG RIVER 206	1.73E-05	3.98E-06	6.03E-07	6.59E-05	1.95E-05	3.79E-06	9.88E-04	2.50E-04	4.12E-05
BLUEBERRY RIVER NO. 205	6.26E-05	8.20E-06	1.16E-06	1.52E-04	4.40E-05	7.48E-06	3.12E-03	4.87E-04	7.85E-05
BLUEBERRY RIVER AND DOIG RIVER 204	2.94E-05	6.01E-06	5.30E-07	1.03E-04	5.97E-05	3.74E-06	1.51E-03	5.36E-04	3.92E-05
Wonowon	6.14E-06	1.25E-06	2.77E-07	2.70E-05	6.97E-06	1.37E-06	3.69E-04	7.87E-05	1.74E-05
Buick	1.65E-05	2.82E-06	4.76E-07	4.79E-05	1.48E-05	1.89E-06	8.64E-04	1.82E-04	2.76E-05
Charlie Lake (Waterbody)	3.13E-06	1.84E-06	1.81E-07	3.20E-05	1.66E-05	1.60E-06	2.81E-04	1.55E-04	1.51E-05

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H ind.	24 H ind.	Ann ind.	1 H pheno.	24 H pheno.	Ann pheno.	99p 1h SO_2	98 p 1h NO_x	98 p 1h NO_2	98p 24h $PM_{2.5}$
MPOI	6.30E-04	1.29E-04	1.74E-05	2.25E-02	4.72E-03	9.96E-04	5.04E+02	1.66E+03	2.92E+02	7.36E+00
MPOI Location (Receptor X m)	*-54.625*	*-55.875*	*-55.125*	*-54.625*	*-33*	*-49*	*-76.875*	*-59*	*-59*	*-5.625*
MPOI Location (Receptor Y m)	*76.125*	*75.625*	*75.875*	*76.125*	*157*	*155*	*146.875*	*169*	*169*	*120.625*
Tumbler Ridge [2]	1.94E-06	7.12E-07	3.24E-08	1.05E-04	3.85E-05	1.97E-06	5.44E+00	7.47E+00	7.47E+00	9.40E-03
Kelly Lake [3]	1.31E-06	4.54E-07	1.75E-08	3.80E-04	5.29E-05	2.86E-06	9.37E+00	1.19E+01	1.19E+01	1.26E-02
Charlie Lake (Community)	8.00E-06	4.30E-06	4.48E-07	5.62E-04	3.05E-04	2.91E-05	3.15E+01	3.30E+01	3.30E+01	1.24E-01
Tomslake	2.55E-06	9.55E-07	6.76E-08	1.20E-04	4.69E-05	3.83E-06	7.51E+00	7.63E+01	7.63E+01	4.15E-02
Lone Prairie	3.63E-06	1.68E-06	1.29E-07	1.63E-04	8.63E-05	6.84E-06	1.22E+01	8.72E+00	8.72E+00	2.84E-02
Pine Valley	2.17E-06	9.08E-07	7.59E-08	1.93E-04	4.94E-05	4.56E-06	4.92E+01	4.48E+01	4.48E+01	2.73E-02
Chetwynd	3.40E-06	1.53E-06	1.51E-07	1.61E-04	8.46E-05	8.49E-06	3.25E+01	9.81E+00	9.81E+00	3.92E-02
Pouce Coupe	6.35E-06	1.34E-06	1.33E-07	2.40E-04	7.10E-05	6.78E-06	1.03E+01	2.45E+01	2.45E+01	3.30E-02
East Pine	6.99E-06	2.80E-06	2.16E-07	3.14E-04	1.46E-04	1.17E-05	1.39E+01	1.34E+01	1.34E+01	5.94E-02
Arras	6.69E-06	2.37E-06	2.06E-07	3.07E-04	1.19E-04	1.05E-05	1.33E+01	1.63E+01	1.63E+01	4.19E-02
Dawsons Creek	1.95E-05	2.50E-06	1.89E-07	6.95E-04	1.19E-04	9.47E-06	1.46E+01	2.00E+01	2.00E+01	3.99E-02
WEST MOBERLY LAKE 168A	6.30E-06	1.44E-06	1.31E-07	3.04E-04	7.17E-05	7.37E-06	2.14E+01	1.06E+01	1.06E+01	4.22E-02
Moberly Lake	5.82E-06	1.48E-06	1.33E-07	2.92E-04	7.36E-05	7.48E-06	1.34E+01	1.10E+01	1.10E+01	4.30E-02
EAST MOBERLY LAKE 169	5.59E-06	1.83E-06	1.70E-07	2.51E-04	9.51E-05	9.40E-06	1.98E+01	1.29E+01	1.29E+01	5.31E-02
Rolla	5.08E-05	9.84E-06	3.97E-07	1.82E-03	3.52E-04	1.72E-05	1.61E+01	4.51E+01	4.51E+01	4.94E-02
Doe River	1.86E-05	3.52E-06	5.80E-07	6.87E-04	1.27E-04	2.47E-05	1.47E+01	2.55E+01	2.55E+01	4.89E-02
Hudsons Hope	4.75E-06	2.13E-06	1.70E-07	2.21E-04	9.83E-05	9.58E-06	2.25E+01	1.59E+01	1.59E+01	5.38E-02
Taylor	2.72E-04	3.89E-05	6.00E-06	9.73E-03	1.39E-03	2.26E-04	1.34E+02	1.01E+03	2.27E+02	5.77E-01
Fort St. John	2.98E-05	6.73E-06	8.72E-07	1.17E-03	4.96E-04	5.33E-05	1.81E+02	1.94E+02	1.46E+02	5.94E-01
Pineview	1.37E-05	6.02E-06	8.08E-07	1.47E-03	5.88E-04	8.57E-05	5.09E+01	5.16E+01	5.16E+01	2.61E-01
Goodlow	1.52E-05	2.89E-06	6.14E-07	5.95E-04	1.32E-04	3.20E-05	4.48E+01	1.14E+02	1.14E+02	3.49E-01
HALFWAY RIVER 168	9.01E-06	2.96E-06	4.00E-07	4.77E-04	2.09E-04	3.97E-05	1.86E+01	3.48E+01	3.48E+01	1.12E-01
Rose Prairie	1.40E-05	6.75E-06	9.90E-07	1.35E-03	7.11E-04	1.14E-04	1.83E+01	6.78E+01	6.78E+01	3.39E-01
DOIG RIVER 206	4.19E-05	9.62E-06	1.46E-06	1.83E-03	4.76E-04	8.03E-05	1.66E+01	3.69E+01	3.69E+01	1.77E-01
BLUEBERRY RIVER NO. 205	1.51E-04	1.98E-05	2.80E-06	5.52E-03	9.55E-04	1.56E-04	4.47E+01	1.50E+02	1.41E+02	1.71E-01
BLUEBERRY RIVER AND DOIG RIVER 204	7.12E-05	1.45E-05	1.28E-06	2.70E-03	1.10E-03	7.79E-05	1.11E+01	5.55E+01	5.55E+01	1.62E-01
Wonowon	1.48E-05	3.02E-06	6.70E-07	6.95E-04	1.54E-04	3.33E-05	1.26E+01	2.16E+01	2.16E+01	5.93E-02
Buick	3.99E-05	6.81E-06	1.15E-06	1.55E-03	3.50E-04	5.13E-05	1.52E+01	4.96E+01	4.96E+01	1.21E-01
Charlie Lake (Waterbody)	7.57E-06	4.44E-06	4.36E-07	5.81E-04	3.17E-04	3.07E-05	3.00E+01	3.37E+01	3.37E+01	1.26E-01

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H NO_2	24 H NO_2	Ann NO_2
MPOI	5.35E+02	8.89E+01	4.75E+01
MPOI Location (Receptor X m)	*-73*	*-5.625*	*-5.625*
MPOI Location (Receptor Y m)	*105*	*120.625*	*120.625*
Tumbler Ridge [2]	1.28E+01	4.08E+00	2.68E-01
Kelly Lake [3]	1.94E+01	3.09E+00	5.71E-01
Charlie Lake (Community)	7.14E+01	2.39E+01	2.60E+00
Tomslake	1.44E+02	2.60E+01	2.37E+00
Lone Prairie	1.20E+01	6.42E+00	6.55E-01
Pine Valley	7.76E+01	1.41E+01	8.19E-01
Chetwynd	1.19E+01	7.16E+00	7.79E-01
Pouce Coupe	3.79E+01	1.39E+01	1.22E+00
East Pine	2.25E+01	1.06E+01	9.67E-01
Arras	3.04E+01	1.10E+01	1.17E+00
Dawsons Creek	3.85E+01	1.36E+01	1.19E+00
WEST MOBERLY LAKE 168A	2.46E+01	7.19E+00	6.60E-01
Moberly Lake	2.35E+01	7.31E+00	6.69E-01
EAST MOBERLY LAKE 169	2.15E+01	1.01E+01	8.06E-01
Rolla	1.16E+02	1.51E+01	1.53E+00
Doe River	4.27E+01	1.19E+01	1.99E+00
Hudsons Hope	2.17E+01	1.04E+01	8.84E-01
Taylor	3.63E+02	7.13E+01	4.22E+01
Fort St. John	1.63E+02	4.48E+01	1.66E+01
Pineview	8.23E+01	3.33E+01	4.70E+00
Goodlow	1.45E+02	2.76E+01	4.45E+00
HALFWAY RIVER 168	1.28E+02	1.87E+01	1.79E+00
Rose Prairie	8.08E+01	3.95E+01	5.87E+00
DOIG RIVER 206	1.39E+02	2.48E+01	3.73E+00
BLUEBERRY RIVER NO. 205	1.98E+02	4.64E+01	1.41E+01
BLUEBERRY RIVER AND DOIG RIVER 204	1.60E+02	4.23E+01	4.41E+00
Wonowon	3.48E+01	1.34E+01	1.69E+00
Buick	1.43E+02	2.48E+01	3.44E+00
Charlie Lake (Waterbody)	4.28E+01	2.17E+01	2.56E+00

Notes:

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Table A3 : Maximum[1] Air Quality Modelling Results for the Upstream Oil and Gas Sources and the Background Sources (µg/m^3)

Representative_Receptor	1 H SO_2	24 H SO_2	Ann SO_2	1 H $PM_{2.5}$	24 H $PM_{2.5}$	Ann $PM_{2.5}$	1 H H_2S	24 H H_2S	Ann H_2S	1 H VOC	24 H VOC	Ann VOC
MPOI	1.06E+03	1.86E+02	1.55E+01	1.55E+03	2.39E+02	2.89E+01	2.64E+01	5.31E+00	1.03E+00	3.26E+03	1.07E+03	2.82E+02
MPOI Location (Receptor X m)	*-76.875*	*-73*	*-101*	*-111*	*-111*	*-137*	*-76.375*	*-76.375*	*-76.375*	*-51.375*	*-51.125*	*-52.125*
MPOI Location (Receptor Y m)	*146.875*	*157*	*163*	*15*	*15*	*41*	*147.125*	*148.125*	*148.125*	*111.625*	*111.625*	*111.625*
Tumbler Ridge [2]	7.02E+00	1.98E+00	1.20E-01	6.81E-01	1.56E-01	1.34E-02	4.93E-02	1.15E-02	5.84E-04	1.53E+01	5.50E+00	2.88E-01
Kelly Lake [3]	1.10E+01	1.63E+00	1.38E-01	3.20E-01	9.50E-02	1.06E-02	2.78E-01	2.50E-02	1.03E-03	1.85E+01	3.51E+00	2.52E-01
Charlie Lake (Community)	3.70E+01	6.92E+00	9.37E-01	1.19E+01	5.18E+00	4.86E-01	3.50E-01	7.20E-02	6.41E-03	3.57E+02	1.62E+02	2.29E+01
Tomslake	9.91E+00	2.08E+00	3.82E-01	1.69E+00	7.20E-01	1.07E-01	8.17E-02	1.00E-02	1.04E-03	5.80E+01	2.27E+01	3.41E+00
Lone Prairie	1.90E+01	4.46E+00	6.38E-01	2.15E+00	5.40E-01	1.11E-01	6.72E-02	1.67E-02	1.97E-03	2.25E+01	1.07E+01	1.13E+00
Pine Valley	9.03E+01	7.79E+00	4.40E-01	2.68E+01	3.13E+00	2.99E-01	3.34E-01	5.05E-02	2.47E-03	2.71E+01	7.42E+00	8.75E-01
Chetwynd	5.48E+01	9.81E+00	1.55E+00	1.68E+01	4.19E+00	1.04E+00	1.88E-01	3.33E-02	4.02E-03	4.73E+02	1.37E+02	4.38E+01
Pouce Coupe	1.29E+01	3.00E+00	8.02E-01	1.22E+01	3.75E+00	7.28E-01	1.10E-01	1.81E-02	1.62E-03	3.57E+02	1.17E+02	3.13E+01
East Pine	2.20E+01	3.54E+00	9.11E-01	2.50E+00	1.06E+00	2.13E-01	8.82E-02	2.52E-02	2.58E-03	4.61E+01	2.42E+01	5.50E+00
Arras	1.54E+01	4.25E+00	6.68E-01	4.19E+00	9.63E-01	1.54E-01	1.61E-01	2.73E-02	2.12E-03	1.22E+02	3.29E+01	5.01E+00
Dawsons Creek	2.66E+01	1.04E+01	2.37E+00	8.08E+01	2.70E+01	4.85E+00	1.58E-01	2.15E-02	2.07E-03	3.02E+03	1.06E+03	2.23E+02
WEST MOBERLY LAKE 168A	4.27E+01	4.59E+00	4.51E-01	3.13E+00	7.60E-01	7.56E-02	9.77E-02	2.61E-02	2.14E-03	4.21E+01	1.69E+01	2.07E+00
Moberly Lake	1.74E+01	3.54E+00	4.12E-01	2.68E+00	7.34E-01	5.97E-02	7.10E-02	2.41E-02	2.07E-03	4.50E+01	1.71E+01	1.47E+00
EAST MOBERLY LAKE 169	3.66E+01	6.38E+00	7.33E-01	2.79E+00	9.83E-01	1.78E-01	2.33E-01	2.87E-02	2.60E-03	7.26E+01	2.87E+01	5.85E+00
Rolla	3.42E+01	4.33E+00	6.86E-01	2.44E+00	8.14E-01	1.07E-01	3.09E-01	4.52E-02	3.25E-03	8.92E+01	2.47E+01	3.39E+00
Doe River	3.05E+01	4.45E+00	8.05E-01	1.40E+00	7.11E-01	8.52E-02	1.33E-01	3.94E-02	4.30E-03	4.80E+01	2.15E+01	2.88E+00
Hudsons Hope	4.51E+01	4.94E+00	4.79E-01	2.43E+00	9.28E-01	1.19E-01	1.06E-01	2.74E-02	2.44E-03	5.11E+01	2.61E+01	4.51E+00
Taylor	3.28E+02	3.61E+01	1.50E+00	1.16E+01	3.58E+00	8.59E-01	3.95E-01	6.56E-02	5.07E-03	5.55E+02	1.75E+02	4.81E+01
Fort St. John	3.66E+02	2.66E+01	3.51E+00	1.04E+02	3.46E+01	6.02E+00	2.11E+00	2.65E-01	8.53E-03	3.26E+03	1.07E+03	2.82E+02
Pineview	7.89E+01	1.03E+01	9.11E-01	6.35E+00	2.30E+00	2.33E-01	4.65E-01	1.12E-01	7.60E-03	3.61E+02	1.37E+02	2.15E+01
Goodlow	5.62E+01	8.23E+00	1.14E+00	3.77E+00	6.25E-01	1.37E-01	2.60E+00	3.93E-01	2.61E-02	3.73E+02	6.53E+01	1.49E+01
HALFWAY RIVER 168	4.75E+01	5.14E+00	7.00E-01	1.85E+00	7.72E-01	1.21E-01	2.34E-01	4.55E-02	3.06E-03	7.04E+01	3.87E+01	8.14E+00
Rose Prairie	2.14E+01	5.00E+00	6.53E-01	4.08E+00	2.23E+00	2.21E-01	3.87E-01	6.18E-02	6.66E-03	1.95E+02	1.30E+02	1.64E+01
DOIG RIVER 206	1.19E+02	1.39E+01	9.57E-01	3.37E+00	1.05E+00	1.27E-01	3.12E-01	6.28E-02	9.06E-03	1.07E+02	5.42E+01	9.21E+00
BLUEBERRY RIVER NO. 205	7.27E+01	1.46E+01	1.10E+00	2.65E+00	1.06E+00	1.21E-01	1.03E+00	2.01E-01	7.91E-03	2.91E+02	1.12E+02	2.00E+01
BLUEBERRY RIVER AND DOIG RIVER 204	1.30E+01	3.89E+00	5.25E-01	2.35E+00	1.26E+00	6.60E-02	2.22E-01	5.22E-02	4.41E-03	3.27E+02	1.91E+02	1.06E+01
Wonowon	1.82E+01	3.86E+00	3.88E-01	8.50E-01	3.36E-01	4.17E-02	1.36E-01	6.07E-02	2.50E-03	6.39E+01	2.21E+01	3.56E+00
Buick	1.82E+01	4.14E+00	6.88E-01	1.56E+00	6.45E-01	1.10E-01	3.29E-01	6.00E-02	4.31E-03	1.56E+02	4.87E+01	1.03E+01
Charlie Lake (Waterbody)	4.04E+01	5.87E+00	7.21E-01	3.81E+00	2.03E+00	1.44E-01	4.24E-01	8.91E-02	7.54E-03	1.31E+02	7.05E+01	8.02E+00

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H Acet.	24 H Acet.	Ann Acet.	1 H Acro.	24 H Acro.	Ann Acro.	1 H Ben.	24 H Ben.	Ann Ben.	1 H bap.	24 H bap.	Ann bap.
MPOI	4.59E+01	1.52E+01	1.98E+00	4.26E+00	9.94E-01	2.06E-01	5.20E+01	1.76E+01	4.57E+00	1.23E-03	8.62E-05	1.72E-05
MPOI Location (Receptor X m)	*-51.375*	*-51.125*	*-51.875*	*-50.875*	*-51.125*	*-51.875*	*-52.625*	*-51.125*	*-52.125*	*-102.375*	*-101.375*	*-49*
MPOI Location (Receptor Y m)	*110.625*	*111.875*	*111.625*	*109.875*	*111.875*	*111.625*	*114.125*	*111.625*	*111.625*	*51.375*	*51.375*	*155*
Tumbler Ridge [2]	1.21E-01	4.45E-02	1.84E-03	1.72E-02	6.08E-03	3.17E-04	1.80E-01	7.14E-02	4.60E-03	1.34E-06	4.63E-07	2.67E-08
Kelly Lake [3]	7.60E-02	2.37E-02	1.51E-03	1.16E-02	4.21E-03	3.73E-04	1.52E-01	4.21E-02	4.12E-03	7.38E-06	1.03E-06	4.96E-08
Charlie Lake (Community)	4.92E+00	2.12E+00	1.56E-01	4.81E-01	1.62E-01	1.94E-02	6.59E+00	2.80E+00	4.02E-01	6.56E-05	6.93E-06	5.74E-07
Tomslake	6.75E-01	2.88E-01	2.70E-02	8.09E-02	2.80E-02	5.93E-03	1.26E+00	4.22E-01	8.58E-02	2.00E-06	6.11E-07	5.11E-08
Lone Prairie	1.86E-01	8.53E-02	6.73E-03	2.72E-02	1.35E-02	1.21E-03	3.90E-01	1.74E-01	1.80E-02	5.80E-06	1.21E-06	1.05E-07
Pine Valley	1.40E-01	6.04E-02	5.18E-03	2.09E-02	8.52E-03	8.81E-04	2.64E-01	1.43E-01	1.65E-02	4.03E-06	1.75E-06	1.17E-07
Chetwynd	7.38E+00	1.71E+00	3.17E-01	4.90E-01	1.14E-01	3.33E-02	1.02E+01	2.30E+00	7.53E-01	1.23E-03	8.62E-05	1.64E-05
Pouce Coupe	5.07E+00	1.56E+00	2.30E-01	3.94E-01	1.26E-01	2.82E-02	6.67E+00	2.10E+00	5.50E-01	3.30E-06	1.10E-06	8.22E-08
East Pine	4.37E-01	2.12E-01	4.19E-02	6.11E-02	2.47E-02	5.10E-03	1.53E+00	6.71E-01	1.73E-01	6.36E-06	1.65E-06	2.06E-07
Arras	1.77E+00	3.69E-01	3.83E-02	1.38E-01	4.06E-02	6.68E-03	2.27E+00	6.58E-01	1.27E-01	7.28E-06	1.98E-06	1.41E-07
Dawsons Creek	3.61E+01	1.18E+01	1.65E+00	2.31E+00	8.14E-01	1.55E-01	4.90E+01	1.74E+01	3.63E+00	4.34E-06	1.33E-06	1.13E-07
WEST MOBERLY LAKE 168A	4.80E-01	1.73E-01	1.36E-02	5.94E-02	1.67E-02	1.91E-03	9.40E-01	3.38E-01	3.28E-02	1.32E-05	3.41E-06	1.51E-07
Moberly Lake	5.21E-01	1.79E-01	9.11E-03	5.50E-02	1.66E-02	1.38E-03	7.73E-01	2.75E-01	2.23E-02	1.53E-05	3.19E-06	1.55E-07
EAST MOBERLY LAKE 169	5.54E-01	2.83E-01	4.35E-02	5.54E-02	2.64E-02	4.24E-03	2.95E+00	6.15E-01	1.58E-01	4.85E-05	5.69E-06	2.19E-07
Rolla	8.04E-01	2.37E-01	2.51E-02	8.89E-02	3.04E-02	4.59E-03	1.73E+00	4.98E-01	7.20E-02	5.85E-06	1.64E-06	1.24E-07
Doe River	4.87E-01	2.04E-01	1.92E-02	6.29E-02	2.96E-02	4.03E-03	1.05E+00	4.47E-01	5.23E-02	6.61E-06	1.54E-06	1.66E-07
Hudsons Hope	4.55E-01	2.34E-01	3.40E-02	5.36E-02	2.90E-02	3.58E-03	9.25E-01	5.41E-01	1.10E-01	5.00E-06	2.24E-06	1.51E-07
Taylor	4.36E+00	1.44E+00	2.64E-01	1.05E+00	1.80E-01	3.62E-02	7.57E+00	2.05E+00	7.09E-01	1.07E-04	2.16E-05	7.29E-07
Fort St. John	4.59E+01	1.52E+01	1.98E+00	4.26E+00	9.94E-01	2.06E-01	5.20E+01	1.76E+01	4.57E+00	6.20E-04	5.92E-05	2.80E-06
Pineview	2.06E+00	8.17E-01	6.12E-02	2.72E-01	8.21E-02	9.70E-03	4.91E+00	1.28E+00	1.63E-01	7.15E-05	1.77E-05	1.56E-06
Goodlow	5.75E-01	2.40E-01	3.92E-02	9.22E-02	3.75E-02	5.20E-03	3.59E+00	6.98E-01	1.73E-01	1.22E-05	3.31E-06	4.78E-07
HALFWAY RIVER 168	4.44E-01	2.09E-01	3.63E-02	4.14E-02	1.66E-02	2.88E-03	1.10E+00	5.12E-01	1.32E-01	5.56E-06	2.53E-06	5.64E-07
Rose Prairie	1.39E+00	6.83E-01	4.76E-02	1.46E-01	7.08E-02	4.75E-03	2.15E+00	1.20E+00	1.48E-01	2.68E-05	1.56E-05	1.67E-06
DOIG RIVER 206	7.89E-01	3.96E-01	5.08E-02	8.77E-02	4.31E-02	4.66E-03	1.30E+00	7.87E-01	1.34E-01	1.40E-05	5.06E-06	7.45E-07
BLUEBERRY RIVER NO. 205	2.51E+00	4.40E-01	7.12E-02	2.04E-01	3.79E-02	7.28E-03	1.30E+00	6.85E-01	1.01E-01	2.14E-05	7.75E-06	1.33E-06
BLUEBERRY RIVER AND DOIG RIVER 204	1.21E+00	4.83E-01	2.82E-02	9.94E-02	4.42E-02	2.86E-03	1.51E+00	1.01E+00	5.32E-02	2.20E-05	1.32E-05	7.02E-07
Wonowon	3.23E-01	1.14E-01	1.74E-02	3.50E-02	1.30E-02	1.55E-03	9.87E-01	2.78E-01	3.56E-02	4.27E-06	1.67E-06	2.08E-07
Buick	7.25E-01	2.51E-01	4.72E-02	6.25E-02	2.43E-02	4.29E-03	1.64E+00	5.62E-01	1.48E-01	9.87E-06	2.41E-06	2.43E-07
Charlie Lake (Waterbody)	1.72E+00	7.97E-01	4.25E-02	3.24E-01	6.33E-02	5.89E-03	1.85E+00	1.01E+00	9.32E-02	4.21E-05	6.92E-06	5.31E-07

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H b13	24 H b13	Ann b13	1 H cum.	24 H cum.	Ann cum.	1 H cyc.	24 H cyc.	Ann cyc.
MPOI	4.72E+00	1.59E+00	2.99E-01	2.02E+00	6.21E-01	1.65E-01	7.59E+00	2.31E+00	6.17E-01
MPOI Location (Receptor X m)	*-52.625*	*-51.125*	*-51.875*	*-51.375*	*-51.125*	*-52.125*	*-51.375*	*-51.125*	*-52.125*
MPOI Location (Receptor Y m)	*114.125*	*111.875*	*111.625*	*111.625*	*111.625*	*111.625*	*111.625*	*111.625*	*111.625*
Tumbler Ridge [2]	1.85E-02	7.30E-03	4.43E-04	6.67E-03	2.86E-03	1.66E-04	1.86E-02	8.08E-03	4.73E-04
Kelly Lake [3]	1.80E-02	4.78E-03	4.81E-04	6.31E-03	1.88E-03	2.02E-04	1.89E-02	5.19E-03	5.34E-04
Charlie Lake (Community)	6.55E-01	2.78E-01	3.25E-02	2.16E-01	9.29E-02	1.40E-02	7.17E-01	3.10E-01	4.56E-02
Tomslake	1.32E-01	4.51E-02	1.00E-02	5.27E-02	1.59E-02	3.93E-03	1.54E-01	4.89E-02	1.01E-02
Lone Prairie	3.23E-02	1.74E-02	1.64E-03	1.09E-02	6.38E-03	6.25E-04	3.36E-02	1.77E-02	1.89E-03
Pine Valley	2.48E-02	1.29E-02	1.59E-03	9.51E-03	4.61E-03	5.14E-04	2.98E-02	1.50E-02	1.52E-03
Chetwynd	8.14E-01	1.92E-01	5.41E-02	2.75E-01	8.59E-02	2.72E-02	9.98E-01	3.14E-01	9.74E-02
Pouce Coupe	6.43E-01	1.98E-01	4.22E-02	2.32E-01	8.13E-02	2.14E-02	7.98E-01	2.79E-01	7.40E-02
East Pine	1.68E-01	7.22E-02	1.82E-02	4.44E-02	2.06E-02	4.94E-03	9.24E-02	4.80E-02	1.13E-02
Arras	2.29E-01	6.49E-02	1.35E-02	7.82E-02	2.36E-02	4.74E-03	2.61E-01	7.78E-02	1.22E-02
Dawsons Creek	3.69E+00	1.27E+00	2.40E-01	1.71E+00	6.05E-01	1.29E-01	6.53E+00	2.30E+00	4.90E-01
WEST MOBERLY LAKE 168A	7.25E-02	3.26E-02	2.69E-03	2.20E-02	1.09E-02	1.13E-03	8.63E-02	3.17E-02	3.82E-03
Moberly Lake	7.52E-02	2.70E-02	1.84E-03	2.37E-02	8.62E-03	7.10E-04	7.55E-02	2.71E-02	2.26E-03
EAST MOBERLY LAKE 169	1.30E-01	5.57E-02	1.52E-02	3.62E-02	1.78E-02	4.32E-03	2.51E-01	5.46E-02	1.12E-02
Rolla	1.39E-01	5.37E-02	7.45E-03	6.20E-02	1.89E-02	2.90E-03	2.10E-01	6.03E-02	7.99E-03
Doe River	1.09E-01	5.13E-02	5.52E-03	4.24E-02	1.80E-02	2.23E-03	1.22E-01	4.75E-02	6.13E-03
Hudsons Hope	9.34E-02	5.19E-02	1.07E-02	2.82E-02	1.71E-02	3.17E-03	7.80E-02	4.88E-02	8.28E-03
Taylor	5.55E-01	1.71E-01	4.18E-02	2.52E-01	7.02E-02	2.22E-02	9.01E-01	2.42E-01	7.64E-02
Fort St. John	4.72E+00	1.59E+00	2.99E-01	2.02E+00	6.21E-01	1.65E-01	7.59E+00	2.31E+00	6.17E-01
Pineview	3.23E-01	1.05E-01	1.07E-02	1.79E-01	4.55E-02	4.75E-03	6.52E-01	1.61E-01	1.52E-02
Goodlow	1.22E-01	4.22E-02	1.21E-02	3.23E-02	1.07E-02	3.08E-03	1.78E-01	3.20E-02	7.90E-03
HALFWAY RIVER 168	1.13E-01	5.24E-02	1.28E-02	2.90E-02	1.33E-02	3.12E-03	7.30E-02	3.30E-02	6.91E-03
Rose Prairie	1.94E-01	1.04E-01	1.26E-02	6.24E-02	3.06E-02	3.26E-03	1.99E-01	9.02E-02	7.72E-03
DOIG RIVER 206	1.33E-01	7.54E-02	1.23E-02	3.18E-02	2.02E-02	3.11E-03	9.08E-02	5.34E-02	7.29E-03
BLUEBERRY RIVER NO. 205	9.49E-02	4.70E-02	5.64E-03	3.85E-02	1.45E-02	2.52E-03	1.54E-01	5.38E-02	8.94E-03
BLUEBERRY RIVER AND DOIG RIVER 204	1.04E-01	5.09E-02	2.53E-03	3.17E-02	1.41E-02	7.26E-04	1.04E-01	4.28E-02	2.13E-03
Wonowon	9.93E-02	2.56E-02	3.16E-03	2.33E-02	6.88E-03	7.45E-04	4.97E-02	1.71E-02	1.63E-03
Buick	1.74E-01	5.46E-02	1.52E-02	4.00E-02	1.36E-02	3.54E-03	7.79E-02	3.11E-02	6.96E-03
Charlie Lake (Waterbody)	1.62E-01	9.68E-02	6.81E-03	6.26E-02	3.32E-02	3.09E-03	2.27E-01	1.13E-01	1.05E-02

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H eb.	24 H eb.	Ann eb.	1 H form.	24 H form.	Ann form.	1 H hex.	24 H hex.	Ann hex.
MPOI	1.44E+01	4.68E+00	1.25E+00	1.90E+02	4.43E+01	8.08E+00	1.67E+02	3.91E+01	7.30E+00
MPOI Location (Receptor X m)	*-51.125*	*-51.125*	*-52.125*	*-101.375*	*-101.375*	*-101.125*	*-49*	*-49*	*-47*
MPOI Location (Receptor Y m)	*111.625*	*111.625*	*111.625*	*154.375*	*154.875*	*154.875*	*155*	*157*	*157*
Tumbler Ridge [2]	5.74E-02	2.37E-02	1.55E-03	1.52E-01	5.65E-02	3.28E-03	4.39E-01	1.48E-01	6.20E-03
Kelly Lake [3]	5.74E-02	1.31E-02	1.41E-03	7.44E-01	9.11E-02	7.86E-03	2.09E-01	8.15E-02	4.21E-03
Charlie Lake (Community)	1.98E+00	8.14E-01	1.33E-01	3.79E+00	1.67E+00	1.41E-01	5.63E+00	2.69E+00	4.10E-01
Tomslake	4.27E-01	1.28E-01	3.36E-02	1.53E+00	3.37E-01	4.58E-02	9.62E-01	3.89E-01	5.64E-02
Lone Prairie	1.07E-01	6.03E-02	5.58E-03	2.32E-01	1.30E-01	1.09E-02	6.04E-01	2.54E-01	2.13E-02
Pine Valley	8.89E-02	4.59E-02	5.81E-03	1.88E-01	8.27E-02	8.00E-03	5.49E-01	1.68E-01	1.76E-02
Chetwynd	2.20E+00	6.70E-01	2.20E-01	5.49E+00	1.27E+00	2.68E-01	6.39E+00	2.01E+00	6.41E-01
Pouce Coupe	1.82E+00	6.16E-01	1.60E-01	4.56E+00	1.26E+00	2.10E-01	4.89E+00	1.71E+00	4.61E-01
East Pine	7.74E-01	3.11E-01	8.06E-02	4.97E-01	2.69E-01	5.18E-02	9.58E-01	4.89E-01	1.04E-01
Arras	6.70E-01	2.13E-01	5.14E-02	1.58E+00	4.54E-01	5.27E-02	1.78E+00	6.77E-01	8.96E-02
Dawsons Creek	1.28E+01	4.56E+00	9.87E-01	4.13E+01	8.65E+00	1.32E+00	4.25E+01	1.51E+01	3.21E+00
WEST MOBERLY LAKE 168A	3.31E-01	1.26E-01	1.07E-02	4.65E-01	1.71E-01	1.52E-02	1.01E+00	3.37E-01	3.87E-02
Moberly Lake	2.37E-01	8.58E-02	7.07E-03	5.11E-01	1.77E-01	1.18E-02	1.02E+00	3.31E-01	3.08E-02
EAST MOBERLY LAKE 169	5.75E-01	2.32E-01	7.02E-02	5.12E-01	2.77E-01	4.61E-02	1.31E+00	4.88E-01	1.07E-01
Rolla	4.68E-01	1.68E-01	2.57E-02	2.90E+00	4.42E-01	4.53E-02	1.25E+00	4.56E-01	5.94E-02
Doe River	3.69E-01	1.60E-01	1.78E-02	1.26E+00	2.57E-01	4.07E-02	1.12E+00	4.46E-01	5.24E-02
Hudsons Hope	3.87E-01	2.14E-01	4.77E-02	4.39E-01	2.62E-01	3.53E-02	1.17E+00	5.27E-01	8.71E-02
Taylor	1.75E+00	4.53E-01	1.42E-01	1.11E+01	3.11E+00	7.25E-01	1.01E+01	3.26E+00	8.87E-01
Fort St. John	1.44E+01	4.68E+00	1.25E+00	3.34E+01	1.12E+01	1.58E+00	4.93E+01	1.52E+01	4.20E+00
Pineview	1.29E+00	3.34E-01	3.95E-02	1.74E+00	7.11E-01	5.95E-02	1.62E+01	3.45E+00	6.75E-01
Goodlow	6.02E-01	1.82E-01	5.66E-02	1.50E+00	2.47E-01	6.19E-02	2.09E+01	3.10E+00	4.10E-01
HALFWAY RIVER 168	4.70E-01	2.21E-01	6.10E-02	5.77E-01	2.23E-01	3.96E-02	2.92E+00	9.80E-01	1.54E-01
Rose Prairie	6.26E-01	3.72E-01	5.81E-02	1.15E+00	5.93E-01	4.69E-02	7.79E+00	3.30E+00	3.92E-01
DOIG RIVER 206	6.02E-01	3.05E-01	5.77E-02	5.96E-01	3.66E-01	4.61E-02	6.08E+00	1.37E+00	2.60E-01
BLUEBERRY RIVER NO. 205	3.37E-01	1.69E-01	2.11E-02	1.81E+00	5.97E-01	5.71E-02	1.53E+01	5.07E+00	7.90E-01
BLUEBERRY RIVER AND DOIG RIVER 204	3.51E-01	1.87E-01	1.04E-02	8.54E-01	4.17E-01	2.46E-02	1.96E+01	9.60E+00	4.84E-01
Wonowon	4.81E-01	1.13E-01	1.46E-02	8.19E-01	2.08E-01	1.72E-02	2.41E+00	5.85E-01	1.04E-01
Buick	8.15E-01	2.49E-01	7.07E-02	7.18E-01	3.06E-01	4.78E-02	4.85E+00	1.59E+00	1.59E-01
Charlie Lake (Waterbody)	4.18E-01	2.71E-01	2.60E-02	1.69E+00	6.53E-01	4.12E-02	3.43E+00	1.63E+00	1.95E-01

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H naph.	24 H naph.	Ann naph.	1 H pen.	24 H pen.	Ann pen.	1 H tol.	24 H tol.	Ann tol.	1 H tri.	24 H tri.	Ann tri.
MPOI	6.60E+00	2.18E+00	3.12E-01	3.15E+02	7.33E+01	1.38E+01	1.25E+02	3.94E+01	1.06E+01	2.23E+01	7.28E+00	1.47E+00
MPOI Location (Receptor X m)	*-51.375*	*-51.125*	*-51.875*	*-49*	*-49*	*-47*	*-51.375*	*-51.125*	*-52.125*	*-41.625*	*-41.625*	*-41.125*
MPOI Location (Receptor Y m)	*110.625*	*111.875*	*111.625*	*155*	*157*	*157*	*111.625*	*111.625*	*111.625*	*99.625*	*99.375*	*99.375*
Tumbler Ridge [2]	1.34E-02	5.04E-03	1.96E-04	8.91E-01	3.02E-01	1.25E-02	3.22E-01	1.35E-01	8.44E-03	5.68E-02	2.40E-02	1.57E-03
Kelly Lake [3]	1.06E-02	3.07E-03	1.82E-04	4.22E-01	1.58E-01	8.30E-03	1.24E+00	1.63E-01	9.82E-03	5.24E-02	1.32E-02	1.31E-03
Charlie Lake (Community)	6.82E-01	2.93E-01	2.13E-02	1.56E+01	7.20E+00	1.07E+00	1.36E+01	5.71E+00	8.86E-01	1.84E+00	7.64E-01	1.28E-01
Tomslake	9.18E-02	3.96E-02	3.40E-03	2.40E+00	9.88E-01	1.18E-01	2.55E+00	8.13E-01	1.68E-01	3.87E-01	1.24E-01	3.01E-02
Lone Prairie	2.04E-02	9.26E-03	7.16E-04	1.21E+00	5.08E-01	4.34E-02	5.88E-01	3.30E-01	3.10E-02	1.05E-01	5.97E-02	5.84E-03
Pine Valley	1.60E-02	6.81E-03	5.06E-04	1.02E+00	3.33E-01	3.39E-02	4.94E-01	2.58E-01	2.99E-02	9.47E-02	4.70E-02	5.90E-03
Chetwynd	1.06E+00	2.44E-01	4.86E-02	2.08E+01	6.46E+00	2.04E+00	1.75E+01	5.31E+00	1.70E+00	2.04E+00	6.47E-01	2.11E-01
Pouce Coupe	7.27E-01	2.25E-01	3.56E-02	1.53E+01	5.35E+00	1.44E+00	1.36E+01	4.63E+00	1.22E+00	1.68E+00	5.80E-01	1.49E-01
East Pine	4.18E-02	2.21E-02	3.41E-03	1.91E+00	9.22E-01	1.79E-01	3.38E+00	1.41E+00	3.63E-01	7.46E-01	3.04E-01	7.85E-02
Arras	2.48E-01	4.89E-02	4.31E-03	5.24E+00	1.42E+00	1.83E-01	4.53E+00	1.34E+00	2.55E-01	6.48E-01	2.24E-01	4.88E-02
Dawsons Creek	5.19E+00	1.70E+00	2.60E-01	1.45E+02	5.09E+01	1.08E+01	1.10E+02	3.90E+01	8.31E+00	1.23E+01	4.41E+00	9.62E-01
WEST MOBERLY LAKE 168A	6.32E-02	2.18E-02	1.76E-03	2.02E+00	7.23E-01	9.46E-02	1.45E+00	6.77E-01	6.53E-02	3.21E-01	1.29E-01	1.08E-02
Moberly Lake	6.90E-02	2.26E-02	1.01E-03	2.04E+00	7.49E-01	6.66E-02	1.51E+00	5.29E-01	4.24E-02	2.39E-01	8.87E-02	7.38E-03
EAST MOBERLY LAKE 169	7.13E-02	3.57E-02	4.21E-03	2.21E+00	1.18E+00	2.21E-01	2.88E+00	1.18E+00	3.45E-01	5.68E-01	2.32E-01	6.89E-02
Rolla	1.19E-01	3.18E-02	3.09E-03	3.85E+00	1.02E+00	1.29E-01	3.38E+00	9.62E-01	1.37E-01	1.13E+00	2.10E-01	2.70E-02
Doe River	6.90E-02	2.52E-02	2.26E-03	2.28E+00	8.78E-01	1.07E-01	2.12E+00	8.15E-01	9.47E-02	6.01E-01	1.53E-01	2.19E-02
Hudsons Hope	5.63E-02	2.77E-02	3.43E-03	2.45E+00	1.04E+00	1.84E-01	1.97E+00	1.10E+00	2.38E-01	3.81E-01	2.14E-01	4.71E-02
Taylor	6.23E-01	1.89E-01	3.64E-02	2.42E+01	7.59E+00	2.26E+00	1.50E+01	3.83E+00	1.25E+00	2.23E+01	7.28E+00	1.47E+00
Fort St. John	6.60E+00	2.18E+00	3.12E-01	1.63E+02	4.94E+01	1.36E+01	1.25E+02	3.94E+01	1.06E+01	1.40E+01	4.43E+00	1.20E+00
Pineview	3.11E-01	1.04E-01	7.09E-03	3.07E+01	7.11E+00	1.34E+00	1.08E+01	2.69E+00	2.77E-01	1.29E+00	3.41E-01	3.85E-02
Goodlow	5.94E-02	2.16E-02	2.42E-03	4.22E+00	1.04E+00	2.27E-01	2.79E+00	8.40E-01	2.60E-01	5.88E-01	1.85E-01	5.80E-02
HALFWAY RIVER 168	5.10E-02	2.23E-02	2.77E-03	5.47E+00	1.88E+00	2.83E-01	2.29E+00	1.06E+00	2.79E-01	4.71E-01	2.24E-01	6.03E-02
Rose Prairie	1.78E-01	7.50E-02	3.21E-03	1.44E+01	6.54E+00	7.30E-01	3.99E+00	2.17E+00	2.80E-01	5.93E-01	3.61E-01	5.74E-02
DOIG RIVER 206	7.76E-02	3.84E-02	2.93E-03	1.14E+01	2.64E+00	4.81E-01	2.77E+00	1.56E+00	2.73E-01	5.93E-01	3.00E-01	5.72E-02
BLUEBERRY RIVER NO. 205	9.89E-02	3.95E-02	4.70E-03	2.87E+01	9.54E+00	1.49E+00	2.39E+00	1.01E+00	1.63E-01	3.40E-01	1.72E-01	2.06E-02
BLUEBERRY RIVER AND DOIG RIVER 204	9.10E-02	3.56E-02	1.22E-03	3.67E+01	1.81E+01	9.07E-01	2.28E+00	1.24E+00	6.47E-02	3.42E-01	1.86E-01	1.09E-02
Wonowon	1.97E-02	7.91E-03	6.62E-04	4.51E+00	1.12E+00	1.89E-01	2.08E+00	5.47E-01	6.70E-02	4.73E-01	1.12E-01	1.47E-02
Buick	4.40E-02	1.71E-02	2.03E-03	8.78E+00	2.90E+00	2.65E-01	3.44E+00	1.12E+00	3.04E-01	7.95E-01	2.46E-01	6.93E-02
Charlie Lake (Waterbody)	2.08E-01	1.08E-01	5.26E-03	7.09E+00	3.40E+00	4.33E-01	3.60E+00	2.00E+00	1.88E-01	4.50E-01	2.60E-01	2.55E-02

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H xyl.	24 H xyl.	Ann xyl.	1 H 7_12.	24 H 7_12.	Ann 7_12.	1 H dah.	24 H dah.	Ann dah.	1 H baa.	24 H baa.	Ann baa.
MPOI	3.51E+01	1.05E+01	2.84E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-02	4.56E-03	7.66E-04
MPOI Location (Receptor X m)	*-51.375*	*-51.125*	*-52.125*	*-149*	*-149*	*-149*	*-149*	*-149*	*-149*	*-52.625*	*-51.125*	*-52.125*
MPOI Location (Receptor Y m)	*111.625*	*111.625*	*111.625*	*1*	*1*	*1*	*1*	*1*	*1*	*114.125*	*111.875*	*111.625*
Tumbler Ridge [2]	5.35E-02	2.15E-02	1.14E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.00E-05	2.27E-05	1.61E-06
Kelly Lake [3]	8.07E-01	8.07E-02	2.79E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.68E-05	1.22E-05	1.30E-06
Charlie Lake (Community)	2.94E+00	1.25E+00	1.81E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.09E-03	8.62E-04	1.08E-04
Tomslake	4.69E-01	1.55E-01	1.98E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.75E-04	1.30E-04	3.00E-05
Lone Prairie	1.08E-01	4.56E-02	4.40E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.87E-04	5.86E-05	6.43E-06
Pine Valley	8.56E-02	4.09E-02	3.04E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.13E-04	5.75E-05	7.09E-06
Chetwynd	4.30E+00	1.33E+00	4.12E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.20E-03	6.11E-04	1.79E-04
Pouce Coupe	3.18E+00	1.11E+00	3.03E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.85E-03	5.45E-04	1.08E-04
East Pine	1.79E-01	8.82E-02	1.59E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.87E-04	3.20E-04	8.55E-05
Arras	9.63E-01	2.75E-01	2.54E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.00E-04	2.17E-04	4.99E-05
Dawsons Creek	2.83E+01	9.91E+00	2.10E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.08E-02	3.52E-03	6.35E-04
WEST MOBERLY LAKE 168A	2.40E-01	9.30E-02	1.30E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.14E-04	1.32E-04	1.14E-05
Moberly Lake	2.63E-01	8.81E-02	6.78E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.12E-04	1.03E-04	7.65E-06
EAST MOBERLY LAKE 169	3.32E-01	1.91E-01	3.28E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.06E-03	3.06E-04	7.23E-05
Rolla	7.53E-01	2.00E-01	1.86E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.76E-04	1.71E-04	2.46E-05
Doe River	3.60E-01	1.18E-01	1.35E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.40E-04	1.65E-04	1.75E-05
Hudsons Hope	2.60E-01	1.32E-01	2.00E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.98E-04	2.25E-04	4.80E-05
Taylor	3.84E+00	1.29E+00	3.22E-01	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.38E-03	4.85E-04	9.21E-05
Fort St. John	3.51E+01	1.05E+01	2.84E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.34E-02	4.56E-03	7.66E-04
Pineview	2.88E+00	6.82E-01	5.52E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.68E-04	3.58E-04	3.37E-05
Goodlow	3.70E-01	8.84E-02	1.30E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.08E-04	1.95E-04	6.04E-05
HALFWAY RIVER 168	2.30E-01	9.99E-02	1.75E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.12E-04	2.40E-04	6.42E-05
Rose Prairie	7.36E-01	2.98E-01	1.64E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.24E-04	4.27E-04	6.11E-05
DOIG RIVER 206	3.51E-01	1.44E-01	1.42E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.56E-04	3.40E-04	6.12E-05
BLUEBERRY RIVER NO. 205	5.69E-01	1.76E-01	3.88E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.74E-04	1.94E-04	1.82E-05
BLUEBERRY RIVER AND DOIG RIVER 204	3.59E-01	1.29E-01	5.31E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.10E-04	2.19E-04	1.20E-05
Wonowon	1.10E-01	3.59E-02	1.92E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.20E-04	1.22E-04	1.62E-05
Buick	1.40E-01	5.42E-02	3.92E-03	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.74E-04	2.68E-04	7.60E-05
Charlie Lake (Waterbody)	9.66E-01	4.66E-01	4.26E-02	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-04	2.90E-04	2.15E-05

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H bbf.	24 H bbf.	Ann bbf.	1 H bkf.	24 H bkf.	Ann bkf.	1 H chry.	24 H chry.	Ann chry.
MPOI	3.30E-03	1.09E-03	1.30E-04	3.69E-03	1.22E-03	1.45E-04	3.26E-02	1.08E-02	2.41E-03
MPOI Location (Receptor X m)	*-51.375*	*-51.125*	*-51.875*	*-51.375*	*-51.125*	*-51.875*	*-52.625*	*-51.125*	*-51.875*
MPOI Location (Receptor Y m)	*110.625*	*111.875*	*111.625*	*110.625*	*111.875*	*111.625*	*114.125*	*111.875*	*111.625*
Tumbler Ridge [2]	1.00E-05	2.36E-06	1.19E-07	1.12E-05	2.72E-06	1.37E-07	1.45E-04	5.81E-05	3.46E-06
Kelly Lake [3]	4.39E-06	1.26E-06	8.03E-08	4.95E-06	1.43E-06	9.18E-08	1.33E-04	4.28E-05	4.64E-06
Charlie Lake (Community)	3.30E-04	1.42E-04	8.54E-06	3.69E-04	1.59E-04	9.60E-06	4.29E-03	1.87E-03	2.26E-04
Tomslake	4.26E-05	1.83E-05	1.05E-06	4.78E-05	2.05E-05	1.18E-06	1.04E-03	3.37E-04	8.10E-05
Lone Prairie	8.55E-05	1.16E-05	6.51E-07	9.56E-05	1.30E-05	7.43E-07	3.67E-04	1.26E-04	1.36E-05
Pine Valley	2.96E-05	8.17E-06	5.64E-07	3.31E-05	9.16E-06	6.39E-07	2.04E-04	1.01E-04	1.05E-05
Chetwynd	2.29E-03	1.49E-04	2.09E-05	2.56E-03	1.67E-04	2.34E-05	9.61E-03	1.28E-03	4.16E-04
Pouce Coupe	3.49E-04	1.08E-04	1.45E-05	3.90E-04	1.21E-04	1.62E-05	4.61E-03	1.47E-03	3.52E-04
East Pine	9.35E-05	9.46E-06	1.71E-06	1.04E-04	1.06E-05	1.94E-06	7.14E-04	3.66E-04	8.63E-05
Arras	1.20E-04	2.22E-05	1.77E-06	1.34E-04	2.50E-05	2.00E-06	1.59E-03	4.68E-04	9.19E-05
Dawsons Creek	2.60E-03	8.53E-04	1.11E-04	2.91E-03	9.54E-04	1.24E-04	2.68E-02	9.45E-03	1.86E-03
WEST MOBERLY LAKE 168A	1.96E-04	1.76E-05	1.12E-06	2.19E-04	1.97E-05	1.26E-06	8.81E-04	2.08E-04	2.11E-05
Moberly Lake	9.74E-05	1.20E-05	7.16E-07	1.09E-04	1.36E-05	8.16E-07	4.64E-04	1.81E-04	1.37E-05
EAST MOBERLY LAKE 169	6.50E-04	4.35E-05	2.26E-06	7.27E-04	4.86E-05	2.55E-06	2.76E-03	3.39E-04	6.85E-05
Rolla	5.06E-05	1.31E-05	1.52E-06	5.67E-05	1.47E-05	1.75E-06	1.11E-03	3.97E-04	5.94E-05
Doe River	3.13E-05	1.20E-05	1.26E-06	3.50E-05	1.35E-05	1.47E-06	8.45E-04	3.98E-04	4.82E-05
Hudsons Hope	3.82E-05	1.33E-05	1.36E-06	4.27E-05	1.52E-05	1.54E-06	5.01E-04	3.16E-04	5.06E-05
Taylor	2.84E-04	9.43E-05	1.51E-05	3.18E-04	1.09E-04	1.72E-05	4.50E-03	1.34E-03	3.91E-04
Fort St. John	3.30E-03	1.09E-03	1.30E-04	3.69E-03	1.22E-03	1.45E-04	3.26E-02	1.08E-02	2.41E-03
Pineview	1.42E-04	4.99E-05	2.96E-06	1.59E-04	5.65E-05	3.40E-06	2.77E-03	7.65E-04	8.82E-05
Goodlow	2.86E-05	1.00E-05	8.54E-07	3.26E-05	1.16E-05	1.03E-06	4.54E-04	1.85E-04	4.73E-05
HALFWAY RIVER 168	2.47E-05	1.09E-05	1.28E-06	2.77E-05	1.23E-05	1.47E-06	5.16E-04	2.36E-04	4.78E-05
Rose Prairie	8.49E-05	3.50E-05	1.19E-06	9.57E-05	4.00E-05	1.45E-06	1.27E-03	6.06E-04	5.68E-05
DOIG RIVER 206	3.76E-05	1.72E-05	1.16E-06	4.22E-05	1.98E-05	1.45E-06	5.97E-04	3.58E-04	4.93E-05
BLUEBERRY RIVER NO. 205	5.01E-05	1.96E-05	2.38E-06	6.43E-05	2.30E-05	2.99E-06	6.63E-04	3.02E-04	4.77E-05
BLUEBERRY RIVER AND DOIG RIVER 204	4.52E-05	1.78E-05	7.15E-07	5.26E-05	2.16E-05	9.51E-07	6.71E-04	3.16E-04	1.59E-05
Wonowon	1.08E-05	3.65E-06	3.25E-07	1.21E-05	4.38E-06	4.42E-07	3.59E-04	1.18E-04	1.28E-05
Buick	1.93E-05	7.34E-06	5.41E-07	2.34E-05	8.93E-06	7.41E-07	6.43E-04	2.29E-04	5.70E-05
Charlie Lake (Waterbody)	1.02E-04	5.30E-05	2.34E-06	1.14E-04	5.96E-05	2.67E-06	1.16E-03	6.82E-04	5.31E-05

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	1 H fluo.	24 H fluo.	Ann fluo	1 H ind.	24 H ind.	Ann ind.	1 H pheno.	24 H pheno.	Ann pheno.
MPOI	1.40E-01	4.73E-02	9.69E-03	7.86E-04	2.61E-04	3.12E-05	6.41E-01	2.16E-01	4.13E-02
MPOI Location (Receptor X m)	*-52.625*	*-51.125*	*-52.125*	*-51.375*	*-51.125*	*-51.875*	*-52.625*	*-51.125*	*-52.125*
MPOI Location (Receptor Y m)	*114.125*	*111.875*	*111.625*	*110.625*	*111.875*	*111.625*	*114.125*	*111.875*	*111.625*
Tumbler Ridge [2]	8.32E-04	3.17E-04	2.22E-05	3.15E-06	1.22E-06	5.84E-08	3.03E-03	1.17E-03	8.04E-05
Kelly Lake [3]	8.08E-04	1.85E-04	2.07E-05	1.65E-06	5.99E-07	3.54E-08	2.97E-03	6.85E-04	7.78E-05
Charlie Lake (Community)	2.47E-02	1.02E-02	1.46E-03	8.13E-05	3.65E-05	2.43E-06	1.00E-01	4.17E-02	5.41E-03
Tomslake	5.29E-03	1.82E-03	4.54E-04	1.17E-05	4.81E-06	3.11E-07	2.04E-02	6.72E-03	1.66E-03
Lone Prairie	2.12E-03	8.28E-04	8.38E-05	2.01E-05	2.83E-06	2.74E-07	1.05E-02	2.97E-03	3.11E-04
Pine Valley	1.22E-03	6.50E-04	8.97E-05	7.72E-06	2.36E-06	2.04E-07	4.59E-03	2.44E-03	3.19E-04
Chetwynd	5.45E-02	6.24E-03	2.07E-03	5.38E-04	3.52E-05	5.06E-06	2.79E-01	2.72E-02	8.09E-03
Pouce Coupe	2.14E-02	6.34E-03	1.46E-03	8.27E-05	2.56E-05	3.54E-06	9.15E-02	2.75E-02	6.01E-03
East Pine	1.11E-02	4.46E-03	1.18E-03	2.20E-05	4.11E-06	6.06E-07	3.61E-02	1.47E-02	3.90E-03
Arras	8.80E-03	3.07E-03	7.12E-04	2.92E-05	6.05E-06	6.10E-07	3.47E-02	1.10E-02	2.50E-03
Dawsons Creek	1.11E-01	3.94E-02	7.83E-03	6.15E-04	2.02E-04	2.62E-05	5.05E-01	1.74E-01	3.34E-02
WEST MOBERLY LAKE 168A	4.82E-03	1.76E-03	1.48E-04	4.61E-05	4.19E-06	3.76E-07	2.41E-02	6.20E-03	5.23E-04
Moberly Lake	3.33E-03	1.20E-03	9.71E-05	2.29E-05	4.20E-06	2.93E-07	1.21E-02	4.41E-03	3.53E-04
EAST MOBERLY LAKE 169	1.57E-02	3.61E-03	9.74E-04	1.53E-04	1.04E-05	6.78E-07	7.99E-02	1.33E-02	3.25E-03
Rolla	5.49E-03	2.37E-03	3.53E-04	5.09E-05	9.85E-06	7.31E-07	2.02E-02	8.86E-03	1.29E-03
Doe River	5.10E-03	2.33E-03	2.55E-04	2.33E-05	4.21E-06	8.40E-07	1.83E-02	8.68E-03	9.41E-04
Hudsons Hope	5.31E-03	2.97E-03	6.54E-04	9.04E-06	5.12E-06	4.80E-07	1.80E-02	1.02E-02	2.19E-03
Taylor	1.66E-02	5.80E-03	1.29E-03	2.90E-04	4.69E-05	7.73E-06	7.38E-02	2.39E-02	5.52E-03
Fort St. John	1.40E-01	4.73E-02	9.69E-03	7.86E-04	2.61E-04	3.12E-05	6.41E-01	2.16E-01	4.13E-02
Pineview	1.03E-02	4.15E-03	4.77E-04	3.69E-05	1.70E-05	1.46E-06	4.38E-02	1.60E-02	1.75E-03
Goodlow	8.37E-03	2.67E-03	8.24E-04	1.55E-05	5.08E-06	7.77E-07	2.70E-02	8.82E-03	2.65E-03
HALFWAY RIVER 168	6.84E-03	3.23E-03	8.89E-04	9.34E-06	4.86E-06	5.95E-07	2.31E-02	1.09E-02	2.86E-03
Rose Prairie	8.73E-03	5.43E-03	8.68E-04	2.60E-05	1.44E-05	1.21E-06	3.22E-02	1.89E-02	2.78E-03
DOIG RIVER 206	8.82E-03	4.45E-03	8.49E-04	4.28E-05	1.01E-05	1.63E-06	2.87E-02	1.49E-02	2.72E-03
BLUEBERRY RIVER NO. 205	5.88E-03	2.68E-03	2.73E-04	1.52E-04	2.00E-05	3.19E-06	1.69E-02	8.89E-03	9.35E-04
BLUEBERRY RIVER AND DOIG RIVER 204	5.27E-03	3.08E-03	1.79E-04	7.16E-05	1.78E-05	1.37E-06	1.84E-02	1.01E-02	5.53E-04
Wonowon	7.22E-03	1.67E-03	2.28E-04	1.51E-05	3.22E-06	7.05E-07	2.28E-02	5.43E-03	7.16E-04
Buick	1.22E-02	3.72E-03	1.06E-03	4.00E-05	7.44E-06	1.21E-06	3.90E-02	1.19E-02	3.39E-03
Charlie Lake (Waterbody)	5.13E-03	3.38E-03	2.85E-04	2.68E-05	1.51E-05	9.61E-07	2.13E-02	1.41E-02	1.08E-03

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Representative_Receptor	99p 1h SO_2	98 p 1h NO_x	98 p 1h NO_2	98p 24h $PM_{2.5}$	1 H NO_2	24 H NO_2	Ann NO_2
MPOI	5.04E+02	1.66E+03	2.93E+02	1.13E+02	5.35E+02	8.89E+01	4.78E+01
MPOI Location (Receptor X m)	*-76.875*	*-59*	*-59*	*-111*	*-73*	*-5.625*	*-5.625*
MPOI Location (Receptor Y m)	*146.875*	*169*	*169*	*15*	*105*	*120.625*	*120.625*
Tumbler Ridge [2]	5.45E+00	8.82E+00	8.82E+00	8.87E-02	1.82E+01	6.03E+00	4.22E-01
Kelly Lake [3]	9.41E+00	1.40E+01	1.40E+01	5.43E-02	2.06E+01	4.28E+00	7.43E-01
Charlie Lake (Community)	3.18E+01	7.53E+01	7.53E+01	1.89E+00	1.37E+02	4.44E+01	1.06E+01
Tomslake	7.63E+00	9.30E+01	9.30E+01	3.53E-01	1.46E+02	3.84E+01	7.24E+00
Lone Prairie	1.23E+01	1.49E+01	1.49E+01	3.71E-01	2.04E+01	1.29E+01	1.26E+00
Pine Valley	4.92E+01	4.99E+01	4.99E+01	1.45E+00	7.95E+01	1.62E+01	1.84E+00
Chetwynd	3.28E+01	1.63E+02	1.43E+02	2.94E+00	1.48E+02	4.44E+01	2.18E+01
Pouce Coupe	1.08E+01	9.08E+01	9.08E+01	2.59E+00	1.38E+02	4.27E+01	1.32E+01
East Pine	1.39E+01	8.19E+01	8.19E+01	6.66E-01	1.04E+02	4.23E+01	1.11E+01
Arras	1.35E+01	5.40E+01	5.40E+01	5.73E-01	8.60E+01	3.45E+01	6.47E+00
Dawsons Creek	2.36E+01	3.90E+02	1.65E+02	1.77E+01	1.79E+02	5.53E+01	3.98E+01
WEST MOBERLY LAKE 168A	2.14E+01	2.26E+01	2.26E+01	3.32E-01	3.81E+01	1.79E+01	1.52E+00
Moberly Lake	1.34E+01	2.13E+01	2.13E+01	2.81E-01	3.59E+01	1.56E+01	1.29E+00
EAST MOBERLY LAKE 169	2.00E+01	3.73E+01	3.73E+01	5.54E-01	4.59E+01	2.62E+01	5.71E+00
Rolla	1.62E+01	5.41E+01	5.41E+01	5.06E-01	1.24E+02	2.86E+01	4.06E+00
Doe River	1.49E+01	4.05E+01	4.05E+01	3.74E-01	5.88E+01	2.76E+01	3.89E+00
Hudsons Hope	2.26E+01	3.30E+01	3.30E+01	4.49E-01	4.36E+01	2.70E+01	4.33E+00
Taylor	1.35E+02	1.05E+03	2.31E+02	2.49E+00	3.68E+02	7.25E+01	4.29E+01
Fort St. John	1.82E+02	4.70E+02	1.73E+02	2.04E+01	1.90E+02	5.95E+01	4.39E+01
Pineview	5.10E+01	7.72E+01	7.72E+01	9.79E-01	1.09E+02	4.30E+01	7.85E+00
Goodlow	4.48E+01	1.22E+02	1.22E+02	4.21E-01	1.45E+02	3.55E+01	8.46E+00
HALFWAY RIVER 168	1.87E+01	4.26E+01	4.26E+01	3.38E-01	1.35E+02	2.35E+01	5.98E+00
Rose Prairie	1.85E+01	9.36E+01	9.36E+01	6.92E-01	1.15E+02	4.43E+01	1.01E+01
DOIG RIVER 206	1.66E+01	5.06E+01	5.06E+01	4.04E-01	1.41E+02	3.88E+01	7.43E+00
BLUEBERRY RIVER NO. 205	4.48E+01	1.57E+02	1.42E+02	4.57E-01	2.00E+02	4.68E+01	1.71E+01
BLUEBERRY RIVER AND DOIG RIVER 204	1.12E+01	6.90E+01	6.90E+01	3.44E-01	1.61E+02	4.38E+01	5.27E+00
Wonowon	1.26E+01	3.58E+01	3.58E+01	1.97E-01	6.54E+01	1.81E+01	2.80E+00
Buick	1.59E+01	1.16E+02	1.16E+02	3.53E-01	1.45E+02	4.21E+01	1.10E+01
Charlie Lake (Waterbody)	3.01E+01	4.46E+01	4.46E+01	6.82E-01	5.64E+01	3.56E+01	4.25E+00

Notes

[1] All numbers represent maximum modelled concentration unless column is labelled as 99 p or 98 p (indicating 99th and 98th percentile, respectively).

[2] The community of Tumbler Ridge is outside the modelling domain. The closest receptor to this community was used to assess this location.

[3] The community of Kelly Lake is just on the outskirt of the modelling domain. The closest receptor to this community was used to assess this location.

Appendix B – Background Data

Table of Contents

List of Tables

Glossary

%	percent
95UCLM	95th upper confidence limit on the mean
BC EMS	British Columbia Environmental Monitoring System
BC MOE	British Columbia Ministry of the Environment
BC OGC	British Columbia Oil and Gas Commission
BC	British Columbia
CAC	criteria air contaminants
COPC	chemical(s) of potential concern
H_2S	hydrogen sulphide
HHRA	human health risk assessment
i.e.	Latin for “such as”
LRDW	Land Resource Data Warehouse
MAML	mobile air monitoring laboratory
Max	maximum
MDL	method detection limit
Min	minimum
NAPS	National Air Pollution Surveillance Network administered by Environment Canada
NE BC	northeastern British Columbia
NO_2	nitrogen dioxide
O_3	ozone
PAH	polycyclic aromatic hydrocarbons
$PM_{2.5}$	fine particulate matter
SO_2	sulphur dioxide
Stdev	standard deviation

B1.0 BACKGROUND DATA APPENDIX

In effort to capture potential existing health risks associated with the identified chemicals of potential concern (COPC) in environmental media, a comprehensive search of multiple databases was completed. Searches were performed in relation to measured ambient air, surface water, soil, vegetation, fish and animal tissue concentrations. To help ensure that any information was relevant and representative of conditions in the NE BC region that people may be exposed to, the focus of the search was on information compiled since 2000.

The databases and resources considered in this search were as follows:

- BC Ministry of Health Drinking Water Quality
- BC Environmental Assessment Office, Project Information Centre
- BC Environmental Monitoring System Database[1]
- BC EcoCat Ecological Reports Catalogue
- BC MOE Habitat Wizard
- BC Soils Information Service
- BC Waterbucket
- BC Water Smart
- BC Water Resources Atlas
- BC MOE Water Quality
- BC MOE Terrestrial Ecosystem Information
- BC OGC North East Water Tool
- BC OGC Water Information Portal
- Data BC
- Environment Canada GENIE Database
- Environment Canada OpenData
- FlowWorks
- Geoscience BC
- Northern Health
- Water Environment Hub

In addition to the above list of resources, the websites for various communities within the study area were evaluated for the presence of drinking water monitoring data. In the event that this information was identified, it was determined that data was not available for the COPCs included in this human health risk assessment (HHRA).

Overall, a very limited amount of data was available for the COPCs for environmental media in the region. A brief summary of the database findings is presented in Table 1–1 in association with the above list of resources. The only relevant information identified in relation to the detailed HHRA for the COPCs was a relatively small data set for measured PAH concentrations in soil for the Peace Region, and some ambient air quality data for the criteria air contaminants (CACs) from various locations in NE BC. A summary of the available information for each COPC is provided in Table 1–2.

[1] The BC EMS database includes the Land Resource Data Warehouse (LRDW), GeoBC Data Discovery, and BC Geographic Warehouse databases.

Table 1–1
Summary of Database and Resource Search Findings

Database / Resource	Findings
BC Ministry of Health Drinking Water Quality	Primarily information relating to water quality guidelines and regulation. No measured chemical data available.
BC Environmental Assessment Office, Project Information Centre	Focus of database and site information is on water management. No measured chemical data available.
BC Environmental Monitoring System Database[2]	Queries performed for air, soil, vegetation, water, wildlife tissue, fish tissue. Limited amount of PAH data in soils available for Peace region. Some animal and fish tissue concentration data available, but limited to metals. No relevant information for COPC included in this HHRA.
BC EcoCat Ecological Reports Catalogue	Contains reports from a variety of disciplines, including aquatic species and habitats, terrestrial species and habitats, floodplain mapping, reservoirs, ground water and vegetation. Fish tissue data identified for pre-2000. No other measured chemical data available.
BC MOE Habitat Wizard	Contains information about fish and fish habitat. No measured chemical data available.
BC Soils Information Service	Soil database from 1960s to 1980s. No comment about more recent data on website.
BC Waterbucket	Information relates to water management and sustainability. No measured chemical data available.
BC Water Smart	Provides links to several references. Focus is on water use and management. No measured chemical data available.
BC Water Resources Atlas	Provides information related to the water resources of British Columbia, such as watersheds, water quantity and quality monitoring sites, aquifers, water wells and flood protection works. No measured chemical data available.
BC MOE Water Quality	Contains links to water quality and sediment reports for various communities in NE BC, but majority of links were dead ends. No measured chemical data could be obtained.
BC MOE Terrestrial Ecosystem Information	Contains several data bases, seems to be focused on GIS and ecosystem mapping
BC OGC North East Water Tool	Provides hydrology data. No water quality information.
BC OGC Water Information Portal	Provides links to water quality data from BC EMS and Northern Health. Water quality information limited to metals and aesthetic parameters. No information for COPC.
Data BC	Some water quality information available for Fort St. John and Dawson Creek.
Environment Canada GENIE Database	Parameters monitored and reported in database limited to basic water quality monitoring data. No chemical data for BC available.
Environment Canada OpenData	Contains various air and water quality data. No relevant water quality information identified. Some Province-wide ambient air data, but no region-specific information.
FlowWorks	On line subscription based data sharing services for industry and government. Invitation-basis only. Seems to be related to data submission.
Geoscience BC	Water quality studies, primarily for ground water in shale gas producing areas. Parameters monitored relevant to hydrogeology, but not to human health.
Northern Health	NH regulates water supply systems. Data in EMS
Water Environment Hub	Presents a list of links to resources, many of them included in this summary Table.

[2] The BC EMS database includes the Land Resource Data Warehouse (LRDW), GeoBC Data Discovery, and BC Geographic Warehouse databases.

Table 1–2
Availability of Measured Background Data for the Identified Chemicals of Potential Concern

	Ambient Air Data	Ambient Surface Water Data	Drinking Water Data	Soil Data	Vegetation Data	Fish Tissue Data	Game Tissue Data
Sulphur Dioxide (SO_2)	**yes**	n/a	n/a	n/a	n/a	n/a	n/a
Nitrogen Dioxide (NO_2)	**yes**	n/a	n/a	n/a	n/a	n/a	n/a
Fine Particulate Matter ($PM_{2.5}$)	**yes**	n/a	n/a	n/a	n/a	n/a	n/a
Ozone	**yes**	n/a	n/a	n/a	n/a	n/a	n/a
Benzene	no	no	no	no	no	no	no
Toluene	no	no	no	no	no	no	no
Ethylbenzene	no	no	no	no	no	no	no
Xylenes	no	no	no	no	no	no	no
1,3-butadiene	no	no	no	no	no	no	no
Acrolein	no	no	no	no	no	no	no
Acetaldehyde	no	no	no	no	no	no	no
Cyclohexane	no	no	no	no	no	no	no
Formaldehyde	no	no	no	no	no	no	no
n-hexane	no	no	no	no	no	no	no
Hydrogen sulphide (H_2S)	**yes**	no	no	no	no	no	no
Isopropylbenzene (cumene)	no	no	no	no	no	no	no
Naphthalene	no	no	no	no	no	no	no
n-pentane	no	no	no	no	no	no	no
Trimethylbenzenes	no	no	no	no	no	no	no
Benzo(a)pyrene and other Carcinogenic PAHs	no	no	no	**yes**	no	no	no

B2.0 DATA ANALYSIS

Summary statistics (*i.e.*, average and standard deviation) were calculated for datasets when there were sufficient data. When the proportion of non-detectable results exceeded 60 to 80% of the data, Helsel (2005) suggests that any statistical analysis is likely to result in unacceptably high error rates. As a result, the average was not calculated when sample sizes were less than 10; or when greater than 80% of the chemical concentrations were non-detect (*i.e.*, less than 20% were detected above the method detection limit (MDL)).

B2.1 Air

A limited amount of ambient air monitoring data for the COPC was identified for locations within the study area. When available, data for the COPC were obtained from the:

- BC EMS database (Government of BC 2014),
- BC MOE (2014) Air Quality Data Archives,
- Environment Canada (2014) NAPS database.

The data collected from monitoring within the study area varied widely in terms of sample size, year(s) sampled and the frequency of sampling. In some cases the averaging period of the measured data was also not clearly presented. Ambient air concentrations prior to 2000 were not considered relevant to the current assessment and have not been included in this appendix. As such, data were taken from the most recent available years following 2000.

In addition to the above data sources, the communities of Tomslake, Groundbirch, Rolla, Farmington and Kelly Lake were monitored for a limited period of time during 2010 and 2011 via the BC MOE Mobile Air Monitoring Laboratory (MAML). These MAML locations provided ambient monitoring for NO_2, $PM_{2.5}$, SO_2 and O_3.

Historic monitoring data were available for the town of Taylor for both NO_2 and O_3, however, monitoring for these COPC appears to have been discontinued at this location in 2002. Current ambient monitoring data is available from two locations within Taylor for SO_2 and from one location for H_2S. Additionally, current ambient monitoring data were available from two locations within Chetwynd for both SO_2 and H_2S. Historic ambient $PM_{2.5}$ data is available for Fort St. John prior to 2004, Hudson's Hope prior to 2007, and Dawson Creek up to 2013. A very limited dataset of ambient H_2S air data for Fort St. John is also available from 2013. Tables 2–1 through Tables 2–5 present the summary statistics of the available data for NO_2, $PM_{2.5}$, SO_2, and O_3, respectively.

Table 2–1
Measured Hourly Ambient NO_2 Concentrations at Locations Within the HHRA Study Area

Location	Farmington MAML	Groundbirch MAML	Kelly Lake MAML	Rolla MAML	Taylor Townsite	Tomslake MAML
Years measured	**2010**	**2010**	**2011**	**2010**	**2000-2002**	**2010**
Average	4.3	0.9	4.8	6.8	16.0	3.4
Stdev	3.7	0.8	4.3	7.9	17.6	3.5
Min	0.0	0.0	0.4	0.4	0.0	0.0
Max	27.3	5.3	47.2	51.9	272.8	19.6
Sample Size	650	562	1,321	470	17,134	626

Table 2–2
Measured Hourly Ambient $PM_{2.5}$ Concentrations at Locations Within the HHRA Study Area

Location	Farmington MAML	Groundbirch MAML	Kelly Lake MAML	Rolla MAML	Tomslake MAML	Fort St. John[1]	Dawson Creek[1]	Hudson's Hope[1]
Years measured	**2010**	**2010**	**2011**	**2010**	**2010**	**2001-2003**	**2011-2013**	**2003-2006**
Average	6.7	2.3	4.6	7.5	4.6	6.9	5.0	6.9
Stdev	6.7	2.3	4.6	7.5	4.6	4.6	3.9	5.5
Min	0.3	0.2	0.0	0.5	0.4	0	0.9	2
Max	80.6	10.2	38.9	60.2	21.9	53	27.7	46
Sample Size	684	612	1,221	533	676	588	230	329

Notes:
[1] The averaging period over which samples at these locations were collected is not clearly presented in the database from which the values were obtained.

Table 2–3
Measured Hourly Ambient SO_2 Concentrations at Locations Within the HHRA Study Area

Location	Farmington MAML	Groundbirch MAML	Kelly Lake MAML	Chetwynd (Pine River Gas Plant)	Chetwynd (Pine River Hasler)	Rolla MAML	Taylor South	Taylor Townsite	Tomslake MAML
Years measured	**2010**	**2010**	**2011**	**2010-2013**	**2010-2013**	**2010**	**2010-2013**	**2010-2013**	**2010**
Average	0.7	0.6	1.8	16.5	1.0	0.8	1.8	4.3	0.8
Stdev	1.0	1.1	1.9	33.9	1.5	0.8	4.3	12.5	0.7
Min	0	0	0	0	0	0	0	0	0
Max	18.3	9.4	15.5	765.4	76.5	6.0	203.9	317.1	6.8
Sample Size	652	563	1,290	32,115	32,719	472	32,951	33,304	636

Table 2–4
Measured Hourly Ambient O_3 Concentrations at Locations Within the HHRA Study Area

Location	Farmington MAML	Groundbirch MAML	Kelly Lake MAML	Rolla MAML	Taylor Townsite	Tomslake MAML
Years measured	**2010**	**2010**	**2011**	**2010**	**2000-2002**	**2010**
Average	35.2	44.2	77.7	48.0	39.8	52.5
Stdev	20.5	15.3	15.9	23.9	29.4	28.0
Min	0.6	8.2	23.2	1.2	0.0	1.4
Max	78.7	79.3	113.7	137.6	131.5	100.7
Sample Size	650	563	1,299	470	16,723	649

Table 2–5
Measured Hourly Ambient H_2S Concentrations at Locations Within the Study Area

Location	Fort St. John (Bessborough 237 Road)	Chetwynd (Pine River Gas Plant)	Chetwynd (Pine River Hasler)	Taylor South
Years measured	**2013**	**2010-2013**	**2010-2013**	**2010-2013**
Average	0.6	0.7	0.7	0.2
Stdev	3.0	0.9	0.8	0.2
Min	--	0	0	0
Max	63.8	43.9	22.0	8.4
Sample Size	649	31,020	33,017	25,129

Notes:
-- = The dataset presented several negative values. No interpretation was provided.

B2.2 Soil

A limited amount of soil sample data was available for locations within the study area. Soil sample data was available for three locations from the Government of BC EMS (2014) – Fort St. John, Dawson Creek and Tumbler Ridge. However, sample sizes were not adequate to calculate summary statistics (*i.e.*, average, standard deviation, 95UCLM) for each of the locations. Furthermore, it was not considered appropriate to combine the samples for statistical analysis due to the differences in sampling locations. Therefore, due to the lack of available and representative soil data, soil concentrations were predicted in the multiple pathways exposure assessment. Table 2–6 presents the COPC concentrations in soil measured at three locations under varying depths.

Table 2–6
COPC concentrations measured in soil [mg/kg]

Location	Depth	Chemical	Min	Max	Count	#Non-Detect	% Non-Detect
Fort St. John	0.0 - 0.1	Benz(a)anthracene	<0.001	<0.001	2	2	100%
		Benzo(a)pyrene	<0.001	<0.001	2	2	100%
		Benzo(b)fluoranthene	<0.001	<0.001	2	2	100%
		Benzo(k)fluoranthene	<0.001	<0.001	2	2	100%
		Chrysene	<0.001	0.001	2	1	50%
		Fluoranthene	<0.001	0.002	2	1	50%
		Indeno(1,2,3-c,d)pyrene	<0.001	<0.001	2	2	100%
		Phenanthrene	0.002	0.01	2	0	0%
	0.5 - 0.6	Benz(a)anthracene	<0.001	<0.001	1	1	100%
		Benzo(a)pyrene	<0.001	<0.001	1	1	100%
		Benzo(b)fluoranthene	<0.001	<0.001	1	1	100%
		Benzo(k)fluoranthene	<0.001	<0.001	1	1	100%
		Chrysene	<0.001	<0.001	1	1	100%
		Fluoranthene	<0.001	<0.001	1	1	100%
		Indeno(1,2,3-c,d)pyrene	<0.001	<0.001	1	1	100%
		Phenanthrene	<0.001	<0.001	1	1	100%

Location	Depth	Chemical	Min	Max	Count	#Non-Detect	% Non-Detect
Dawson Creek	0.0 - 0.1	Benz(a)anthracene	<0.001	<0.001	1	1	100%
		Benzo(a)pyrene	<0.001	<0.001	1	1	100%
		Benzo(b)fluoranthene	<0.001	<0.001	1	1	100%
		Benzo(k)fluoranthene	<0.001	<0.001	1	1	100%
		Chrysene	<0.001	<0.001	1	1	100%
		Fluoranthene	<0.001	<0.001	1	1	100%
		Indeno(1,2,3-c,d)pyrene	<0.001	<0.001	1	1	100%
		Phenanthrene	--	0.005	1	0	0%
	0.5 - 0.6	Benz(a)anthracene	--	0.003	1	0	0%
		Benzo(a)pyrene	--	0.002	1	0	0%
		Benzo(b)fluoranthene	--	0.019	1	0	0%
		Benzo(k)fluoranthene	<0.001	<0.001	1	1	100%
		Chrysene	--	0.002	1	0	0%
		Fluoranthene	--	0.004	1	0	0%
		Indeno(1,2,3-c,d)pyrene	--	0.004	1	0	0%
		Phenanthrene	--	0.044	1	0	0%
Tumbler Ridge	0.0 - 0.1	Benz(a)anthracene	<0.001	<0.001	3	3	100%
		Benzo(a)pyrene	<0.001	<0.001	3	3	100%
		Benzo(b)fluoranthene	<0.001	0.002	3	2	67%
		Benzo(k)fluoranthene	<0.001	<0.001	3	3	100%
		Chrysene	<0.001	<0.001	3	3	100%
		Fluoranthene	<0.001	<0.001	3	3	100%
		Indeno(1,2,3-c,d)pyrene	<0.001	<0.001	3	3	100%
		Phenanthrene	0.006	0.014	3	1	33%

Notes:
-- not available

B3.0 OTHER ENVIRONMENTAL MEDIA

Relevant data for other environmental media were not available (*i.e.*, berries, Labrador tea, root, and game meat). A limited amount of data was available for metals in fish and animal tissue. However, due to the nature of the emission sources included in the HHRA, metals were not evaluated. As a result, particular focus was not given to the metals database in biota.

Some drinking water data was available from the various communities in the area and Northern Health. However, no data was identified for the COPC included in the detailed HHRA. Overall, the available data appeared to be limited to aesthetic and microbial parameters, and metals.

Very limited ambient groundwater data for the NE region is available with respect to chemical parameters. In the absence of an established, consistent database of ground water quality, it is difficult to evaluate the potential impacts of oil and gas development.

Similarly, very limited surface water quality data was identified for the COPC included in the HHRA from the sources consulted. The available data was limited to aesthetic and microbial parameters, and some metals.

B4.0 REFERENCES

BC MOE (British Columbia Ministry of the Environment). 2014. British Columbia Air Data Archive Database. Available at: http://envistaweb.env.gov.bc.ca/. Accessed: May 2014.

Environment Canada. 2014. National Air Pollutant Surveillance Database. Available at: http://www.ec.gc.ca/rnspa-naps/default.asp?lang=En&n=8BA86647-1.

Government of BC (Government of British Columbia). 2014. British Columbia Environmental Monitoring System (EMS). Available at: http://www.env.gov.bc.ca/emswr/ Accessed: May 2014.

Helsel, D.R. 2005. Nondetects and Data Analysis: Statistics for Censored Environmental Data. Wiley-Interscience, Denver, CO. pp.77-78.

Appendix C – Toxicity Profiles

TABLE OF CONTENTS

LIST OF TABLES

Glossary

<	less than
>	greater than
%	percent
√	square root
µg/kg bw/d	microgram per kilogram of body weight per day
µg/m³	micrograms per cubic metre
µmol/L	micromoles per litre
AAQO	ambient air quality objective
AAQG	ambient air quality guideline
ACGIH	American Conference of Governmental Industrial Hygienists
ADI	allowable daily intakes
ADJ	adjusted
AEGL	acute exposure guideline level(s)
AMML	acute myelogenous and monocytic leukemia
ANCOVA	analysis of covariance
A or a	animal
AQG	air quality guideline
AQO	air quality objective
ATSDR	Agency for Toxic Substances and Disease Registry
BaP	benzo(a)pyrene
BC MOE	British Columbia Ministry of the Environment
BMC	benchmark concentration
BMCL	benchmark concentration level
BMD	benchmark dose
BMDL	benchmark dose level
bw/d	body weight per day
CAC	criteria air contaminant
CAP	compound action potential
CARB	California Air Resources Board
CCME	Canadian Council of Ministers of the Environment
CEPA/FPAC	Canadian Environmental Protection Act and Federal Provincial Advisory Committee
CES	critical effect size
CI	confidence interval
CNS	central nervous system
CO	carbon monoxide
COHb	carboxyhemoglobin
COPC	chemical(s) of potenical concern
CR	carcinogenic risk
CR_{inhal}	carcinogenic risk via air
CR_{oral}	carcinogenic risk via intake
DAF	dosimetric adjustment factor
DPOAE	distortion product otoacoustic emissions
e.g.	Latin “for example”

EC_{50}	effective concentration (i.e., 50% of the population is affected)
ESL	effects screening level
ESRD	Alberta's Environment and Sustainable Resource Development
et al.	Latin "and other authors"
etc.	Latin *et cetera* "and other"
ET	extrathoracic
EU	European Union
FEL	frank effect level
FEV	forced expiratory volume
GD	gestational day(s)
H or h	human
$H_{b/g}$	ratio of blood:gas partition coefficient
HEC	human equivalent concentration
HEI	Health Effects Institute
HHRA	human health risk assessment
i.e.	Latin "such as" or "that is"
ILCR	incremental lifetime cancer risk
IPCS	International Program on Chemical Safety
IRIS	Integrated Risk Information System
kg	kilogram
kHz	kilohertz
L	libre
LEC	limit of exposure concentration
LOAL	lowest-observed-affect level
LOAEL	lowest-observed-adverse-effect level
m^3/day	cubic metres per day
mg/kg	milligram per kilogram
mg/m^3	milligram per cubic metre
mg/mL	milligram per millilitre
MA DEP	Massachusetts Department of Environmental Protection
MAL	maximum acceptable level
MDL	maximum desirable level
MRL	minimal risk level
MTL	maximum tolerable level
n	number
ng/m^3	nanograms per cubic metre
n/a	not applicable
NAAQS	National Ambient Air Quality Standards
NO_2	nitrogen dioxide
NO_X	nitrogen oxides
NOAEL	no-observed-adverse-effect level
NTP	National Toxicology Program
OEHHA	California Office of Environmental Health Hazard Assessment
OMOE	Ontario Ministry of the Environment
PAH	polycyclic aromatic hydrocarbon
PCO	Pollution Control Objectives

PEF	potency equivalence factor(s)
pH	power of hydrogen
PHC	petroleum hydrocarbon
$PM_{2.5}$	fine particulate matter (less than 2.5 micrometres in diameter)
POD	point of departure
ppb	parts per billion
ppm	parts per million
PPRTVs	Provisional Peer Review Toxicity Values
REL	reference exposure levle
ReV	reference values
RfC	reference concentration
RfD	reference dose
RGDR	regional gas dose ratio
RIVM	Netherlands National Institute of Public Health and the Environment
RsC	risk-specific concentration
RsD	risk-specific dose
SA	surface area
SD	standard deviation
SF	slope factor(s)
SO_2	sulphur dioxide
STEL	short-term exposure limit(s)
TC_{01} or TC_{05}	tumorigenic concentration representing a 1 or 5% excess probability of cancer (mg/m^3)
TC	tolerable concentration
TCA	tolerable concentrations in air
TCEQ	Texas Commission on Environmental Quality
TDI	tolerable daily intake
TEF	toxic equivalency factor(s)
TEQ	toxic equivalency quotient
TLV-TWA	threshold limit value-time weighted average
TPHCWG	Total Petroleum Hydrocarbon Criteria Working Group
TRV	toxicological reference values
TWA	time weighted average
UCL	upper confidence limit
UF	uncertainty factor
URE	unit risk estimate
US EPA	United States Environmental Protection Agency
VE	volume
WHO	World Health Organization

C1.0 INTRODUCTION

This Appendix describes the scientific basis for the acute (short-term) and chronic (long-term) exposure limits used in the quantitative HHRA to assess potential human health risks associated with the chemicals of potential concern (COPC). An overview of the general process used to evaluate and select exposure limits or toxicity reference values for use in the human health risk assessment (HHRA) is provided. As well, this appendix presents a series of individual profiles for the COPC, wherein the available values are summarized and information regarding the selected exposure limits is provided.

C1.1 Background

In general, chemicals may be categorized into two groups based on the nature of their toxic response – threshold chemicals and non-threshold chemicals. Threshold chemicals make up the largest category and consist of virtually all types of toxic responses and chemicals. For threshold chemicals, a minimum or 'threshold' dose must be exceeded for a toxic response to be observed, and the severity or magnitude of the toxic response is generally assumed to increase with increasing dose. Non-threshold chemicals are a select group of substances that can potentially produce cancer through mechanisms that do not involve a threshold response, and a dose-response relationship is not always apparent.

Typically, exposure limits are differentiated on the basis of the duration of exposure in recognition of the variability in toxic responses that may be seen with the same chemical following an acute vs. chronic exposure. For the purposes of this assessment, exposure limits selected to evaluate acute and chronic exposures were based on the following definitions:

- **Acute –** single or intermittent exposures lasting up to 24-hours; and
- **Chronic –** repeated, exposures over longer term periods that are conservatively assumed to take place over a lifetime.

Differing terminology may also be assigned to exposure limits depending on the source of exposure (*e.g.*, air, water, food) and the regulatory jurisdiction involved. Often, generic terminology will apply, with the following terms and descriptions used:

- **Reference Concentration (RfC)** - refers to the safe levels of air-borne threshold chemicals where the primary route of exposure is through inhalation. The RfC is expressed as a concentration of the chemical in air (*i.e.*, micrograms per cubic metre - µg/m³).
- **Reference Dose (RfD) -** refers to the safe levels of threshold chemicals to which exposure occurs through multiple pathways, both primary and secondary (*i.e.*, oral, dermal). It is most commonly expressed as the daily dose of the chemical per unit body weight of the receptor (*i.e.*, micrograms per kilogram of body weight per day - µg/kg•bw/d).
- **Risk-specific Concentration (RsC) –** reserved for non-threshold carcinogens, the RsC refers to the concentration via inhalation that corresponds to a 'socially acceptable' incremental increase in the incidence of cancer, typically of one case in a population of 100,000 people. The RsC is expressed as a concentration in air (*i.e.*, µg/m³).
- **Risk-specific Dose (RsD) –** same as the RsC except that it refers to the dose from multiple pathways that corresponds to a 'socially acceptable' incremental increase in the

incidence of cancer (one in 100,000), often expressed as the daily dose of the chemical per unit body weight of the receptor (*e.g.*, µg/kg•bw/d).

For non-carcinogens, exposure limits are often derived based on the identification of a no-observed-adverse-effect level (NOAEL) – the dose at which no adverse effects are observed. Alternatively, exposure limits may be based upon a lowest-observed-adverse-effect level (LOAEL) or a benchmark dose/concentration (BMD/BMC). A NOAEL, LOAEL or BMD/BMC can then be used to derive an exposure limit or 'safe' level of exposure through the application of 'uncertainty' or safety factors that provide an added level of protection. The exposure limit refers to the dose of the chemical that is without effect on even the most sensitive subjects and is calculated as follows, using a NOAEL as an example:

Exposure Limit = NOAEL/Uncertainty Factor

The uncertainty factor can vary from 3 (or √10) to over 1,000 in order to ensure adequate protection of any exposed population. The most common forms of uncertainty factors are listed in Table 1-1.

The need for these uncertainty factors is dictated largely by the practical constraints that apply to conventional toxicological research (*i.e.*, the study of the harmful effects of chemicals). Most of the available information for some chemicals is limited to studies in laboratory rodents (*e.g.*, rats, mice, guinea pigs, rabbits), owing largely to their availability in large numbers, their low cost, and the ease with which they can be housed and handled.

It is considered to be common practice to apply an uncertainty factor of 10 to account for possible differences in sensitivity between species (*i.e.*, interspecies differences, such as those that might exist between rodents and humans) and an additional uncertainty factor of 10 to accommodate differences in sensitivity between individuals within the same species (*i.e.*, intraspecies differences). Some other uncertainty factors that are often applied include an uncertainty factor of 10 to adjust from subchronic to chronic exposure and a factor of 10 to account for the uncertainty associated with the use of a LOAEL instead of a NOAEL. Where the toxicity database is very limited, an additional uncertainty factor can be applied to account for uncertainties in the database.

In some instances, the uncertainty factors may be less than 10, based on the chemical-specific information reviewed by an agency or organization in the derivation of the value. For example, values of 3 or √10 are used when the available information does not support the use of a factor of 10.

Table 1-1
Examples of Commonly Used Uncertainty Factors

Nature of Uncertainty[1]	Magnitude of Factor	Comments
Differences in sensitivity between species	3 or √10, 10	Used to accommodate the uncertainty around the use of laboratory animal data to predict potential human responses.
Differences in sensitivity within a species	3 or √10, 10	Used to account for individuals within the human population that may be more sensitive to a chemical than the average person.
Subchronic to chronic exposure duration	3 or √10, 10	Used to account for the uncertainty surrounding the use of data involving shorter exposure periods to predict the responses that might occur over longer periods of exposure. Subchronic data is used when exposures are expected to occur for long periods and defensible chronic toxicity data is not available.
LOAEL to a NOAEL	3 or √10, 10	Used to account for the uncertainty surrounding the use of a LOAEL when a NOAEL is not available for the most sensitive test species.
Database uncertainty	3 or √10, 10	Used to account for a lack of toxicological information for one or more endpoints.

(1) Uncertainty factors are not applied in the derivation of non-threshold carcinogenic exposure limits.

Some chemicals are capable of producing cancer through a number of possible mechanisms (*e.g.*, mutagenicity, cytotoxicity, inhibition of programmed cell death, mitogenesis (uncontrolled cell proliferation) and immune suppression) that, in theory, do not require the exceedance of a threshold (US EPA 2005). These compounds are refered to as non-threshold carcinogens.

In general, tumorigenicity data from animals or human epidemiological studies are evaluated to gain a better understanding of cause and effect, and the potential mechanisms of action. In studies where animal data was used, it is becoming more common that toxicokinetic models or dose-scaling methods be used to estimate human equivalent doses. The exposure concentrations in carcinogenicity studies vary, and may be higher than the range of concentrations that would be protective of human health. As a result, various mathematical techniques are often used to extrapolate the dose-response curve based on observed data to lower-dose concentrations. For example, mathematical transformations may be completed on the data in order to a fit a curve to the data set, from which an effect dose (*e.g.*, 5% or 10% increase in tumour incidence) can be determined. From this point, low-dose extrapolation may be completed using a linear approach, non-linear approach, or both. The choice of these extrapolation methods is dependent in part on the toxicological mode of action for the chemical under study. In general, when linear methods are used, the slope of the line is an estimate of the extra cancer risk per unit dose (*e.g.*, per mg/kg or (mg/kg)-1). This slope factor can be used directly in risk assessments, or transformed into Unit Risk Estimates (URE) which represent the cancer risk per unit of media (*e.g.*, per µg/m^3 or per µg/L. Another expression of cancer risks is the Risk-specific Dose (RsD) or Risk-specific Concentration, where the cancer risk is expressed in terms of the risk per 100,000 people. Slope factors, URE and RsD/RsC can all be inter-converted, as they are all based on the same data. No uncertainty factors are applied to these values (TERA 2014; US EPA 2005).

When a nonlinear method is used to extrapolate tumour data (*i.e.*, for a threshold acting carcinogen), the value generated is an effect-level that is more comparable to an RfC or RfD-based approach. Various uncertainty factors may be applied to non-linear-based values (TERA 2014; US EPA 2005).

Regulatory agencies such as Health Canada and the US EPA assume that any level of long-term exposure to carcinogenic chemicals is associated with some 'hypothetical cancer risk'. As a result, Health Canada has specified an incremental lifetime cancer risk (ILCR) (*i.e.*, over and above background) of 1.0 in 100,000 to be acceptable, tolerable or essentially negligible (Health Canada 2012). The regulatory benchmark of an acceptable cancer risk is policy-based and its interpretation by various regulatory agencies differs (CCME 2006).

C1.2 Exposure Limit Selection

A tiered approach was used in the review and selection of available exposure limits for each of the COPC. 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.

To ensure that the most defensible and appropriate exposure limit was selected for each chemical in the HHRA, consideration was given only to exposure limits meeting the following criteria:

- Established or recommended by reputable scientific authorities;
- Protective of the health of the general public based on the current scientific understanding of the health effects known to be associated with exposures to the COPC;
- Protective of sensitive individuals through the use of appropriate uncertainty factors; and
- Supported by adequate and available documentation.

All supporting documents were critically evaluated to identify the most appropriate and defensible value for use in the HHRA. In the case that the above criteria were supported by more than one standard, guideline or objective, the most scientifically defensible limit was selected and the rationale for the decision is provided in the toxicity profile.

The process and resources used in selecting exposure limits varied slightly between the acute inhalation, chronic inhalation and chronic oral sections, due to the types of information available for these values. For all three categories of exposure limits, a tiered process of limit review and selection was utilized.

Two 'Tiers' of sources for exposure limits have been identified. The resources in Tier 1 represent reputable governmental agencies or established organizations, generally have supporting documentation available, and are generally recognized by governmental agencies. In the event that a defensible value with available supporting documentation was not available from Tier 1, the search for exposure limits was extended to include the agencies and organizations listed as Tier 2.

For some chemicals, the approach for Tier 1 can vary slightly due to the nature of the information available. Some notable examples are below.

Criteria Air Contaminants (CACs)

Many jurisdictions have specific air quality guidleines for CACs and various other chemicals, such as formaldehyde and lead. As a result, the search strategy for these substance is slightly different.

For $PM_{2.5}$, in addition to the standard Tier 1 list, values from the CCME, the California Air Resources Board (CARB), United States Environmental Protection Agency (US EPA) values are considered.

For nitrogen dioxide (NO_2) and sulphur dioxide (SO_2), as no other US EPA values are available, and recent National Ambient Air Quality Standards (NAAQSs) from the US EPA are available for these two substances, consideration is given to the 1-hour Standards for both NO_2 and SO_2 as well as the appropriate statistics.

Carbon monoxide (CO) is considered only on an acute basis, using the standard Tier 1. No limit is selected for the chronic inhalation section due to the toxicological characteristics of CO.

C1.3 Acute Inhalation Exposure Limits

The Tier 1 sources for acute inhalation exposure limits are as follows:

- Alberta's Environment and Sustainable Resource Development (ESRD) – Ambient Air Quality Objectives (1-hour, 8-hour, 24-hour);
- Agency for Toxic Substances and Disease Registry (ATSDR) – Minimal Risk Levels (MRLs), Acute inhalation;
- British Columbia Ministry of the Environment (BC MOE) – Ambient Air Quality Objectives;
- California Office of Environmental Health Hazard Assessment (OEHHA) – Acute Reference Exposure Levels (RELs);
- Ontario Ministry of the Environment (OMOE) – Air Quality Standards and Guidelines (1-hour, 24-hour guidelines);
- Texas Commission for Environmental Quality (TCEQ) – Acute Reference Values (ReV);
- United States Environmental Protection Agency (US EPA). Integrated Risk Information System (IRIS) – Acute Reference Concentrations;
- World Health Organization (WHO) – Air Quality Guidelines for Europe.

In the event that a defensible value with adequate supporting documentation could not be identified from the Tier 1 sources, the search for acute exposure limits was expanded to include the following Tier 2 sources:

- American Conference for Governmental and Industrial Hygienists (ACGIH). Only Short-term Exposure Limits (STELs) and ceiling values should be considered as potential Tier 2 acute values;
- US EPA Acute Exposure Guideline Levels (AEGLs) Level 1 (*i.e.*, AEGL-1 values).

C1.4 Chronic Inhalation Exposure Limits

The search for chronic inhalation exposure limits involved the consideration of both cancer-based and non-cancer based exposure limits, when applicable. For COPCs with

defensible cancer and non-cancer limits, both types of assessment (carcinogenic and non-carcinogenic) assessments were completed.

The classification of carcinogenicity for PAHs varies among different regulatory agencies. In this assessment, Health Canada's Potency Equivalence Factors (PEFs) were selected as the source for determination of a PAH's carcinogenic potential. Based on this approach, a PAH is considered a carcinogen only if it is identified with a PEF relative to benzo(a)pyrene. Health Canada provides a list of PAHs considered being carcinogenic with a PEF identified.

The Tier 1 sources used to identify chronic inhalation exposure limits for the HHRA included:

- ESRD – Alberta Ambient Air Quality Objectives (annual);
- ATSDR – Minimal Risk Levels (Chronic inhalation);
- British Columbia Ministry of the Environment (BC MOE) – Ambient Air Quality Objectives and Pollution Control Objectives;
- BC MOE Contaminated Sites Regulation 375/96 of the Environmental Management Act - Generic Numerical Vapour Standards;
- Health Canada - Federal Contaminated Sites document and Existing Substances Division Tolerable Concentrations and Tumourigenic Concentrations); or the Health Based Guidance Values for Substances on the Second Priority Substances List;
- OEHHA – Chronic RELs, Chronic inhalation RsC (or unit risk estimates (URE) or slope factors (SF) converted to RsC) from the Cancer Potency Factors document;
- Health Institute of the Netherlands (RIVM) – Chronic Tolerable Concentrations in Air (TCA) or excess carcinogenic risk via air (CR_{inhal});
- TCEQ – Chronic ReVs and Chronic Linear ESLs;
- US EPA– Chronic RfCs, Chronic RsCs (or URE or SF converted to RsC);
- WHO – Annual Air Quality Guidelines

In the event that a defensible chronic value with adequate supporting documentation was not available from these sources, the search was expanded to include the following Tier 2 sources:

- ATSDR MRLs, intermediate (sub-chronic) inhalation;
- ACGIH TLV-TWA; and,
- PPRTVs from the US EPA.

C1.5 Chronic Oral Exposure Limits

The selection of chronic oral exposure limits for use in the multiple pathway assessment also considered two Tiers of values. Similar to the chronic inhalation assessment, consideration was given to both cancer and non-cancer based values, where applicable.

The Tier 1 sources consulted for chronic oral exposure limits included:

- ATSDR – Minimal Risk Levels (Chronic oral);
- Health Canada – Federal Contaminated Sites Document, Existing Substances Division Tolerable Daily Intakes (TDI) and Tumorigenic Concentrations, and TDI or Allowable Daily Intakes (ADI) that serve as the basis for the Canadian Drinking Water Quality Guidelines;
- OEHHA – chronic oral slope factors converted to RsD from the Cancer Potency Factors document;
- RIVM – oral TDIs or excess carcinogenic risk via intake (CR_{oral});
- US EPA – Chronic Oral RfDs (or SF converted to RsD); and,

- WHO – TDIs or ADIs that are the basis of the World Health Organization drinking water guidelines.

In the event that a defensible value with adequate documentation could not be identified for a COPC, the following Tier 2 sources were consulted:

- ATSDR MRLs, intermediate (subchronic) oral; and,
- Provisional Peer Review Toxicity Values (PPRTVs) from the US EPA.

C2.0 1,3-BUTADIENE

C2.1 Acute Inhalation Exposure Limits

Table 2-1
Acute Inhalation Exposure Limits for 1,3-Butadiene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR			ATSDR 2013, 2012
ESRD	–	–	ESRD 2013
OEHHA	1-hour REL	660	OEHHA 2014
OMOE	–	–	OMOE 2012
TCEQ	1-hour ReV	3,700	TCEQ 2013, 2008
US EPA	24-hour RfC	15	US EPA 2014, 2002
WHO	–	–	WHO 2000

– = Not available

The US EPA (2014, 2002) presents a 24-hour acute RfC of 15 µg/m³ based on decreased fetal body weights in males. Pregnant CD-1 mice were administered 0, 40, 200 and 1,000 ppm 1,3-butadiene via inhalation for 6 hours/day on gestational days (GDs) 6 to 15. Dams were weighed prior to mating and on GDs 0, 6, 11, 16 and 18. They were sacrificed on GD 18. The study examined a number of reproductive and developmental outcomes. The reproductive outcomes included the number of implants, resorptions, and live/dead fetuses, while fetal weights and observation of external, visceral, and/or skeletal abnormalities observed as the developmental outcomes (Hackett *et al.* 1987). Hackett *et al.* (1987) reported a statistically significant reduction in male fetal body weights at all exposure concentrations relative to the controls. On GD 20, male fetal body weights were 5, 18 and 23% lower than controls in the 40, 200 and 1,000 ppm groups, respectively. As such, a LOAEL of 40 ppm was identified for fetal effects (decreased body weight in males). The US EPA (2002) selected this endpoint for further investigation, and conducted several iterations of benchmark dose modelling (generating Effect Concentrations) and various approaches for evaluating and transforming data. Of the approaches used by the US EPA (2002), the most conservative estimate of a POD was the LEC_{05} of 2.9 ppm. An uncertainty factor of 400 was applied to account for interspecies differences (3), intraspecies differences (10), the use of an effect-level (4, similar to a LOAEL-to-NOAEL extrapolation factor), and database limitations (3). The result is an acute RfC of 7 ppb or 15 µg/m³ based on a reproductive and developmental endpoint. This value was selected for use as a 24-hour limit in the acute effects assessment.

The TCEQ (2013, 2008) has derived an acute ReV of 3,700 µg/m³ (1,700 ppb) also based on Hackett *et al.* (1987). In addition, the TCEQ (2008) presents a re-analysis of the Hackett *et al.* (1987) data based on indications that the apparent significant decrease in male fetal body weight in the 40 ppm groups was the erroneous result of the statistical analysis used (Christian 1996; Green 2003). The Green (2003) re-analysis using analysis of covariance (ANCOVA) on the average pup weight adjusted for covariates in combination with the Dunnett-Hsu test to compare the mean weight for each of the exposed groups to the mean weight for the control group indicates that the lowest exposure concentration of 40 ppm (88 mg/m³) should be considered the study NOAEL for decreased male fetal body weights (TCEQ 2008), and not an effect level as reported by Hackett *et al.* (1987). Green's conclusions were corroborated by

Sielken *et al.* (2007) following review of the Hackett *et al.* (1987) study and the Green (2003) re-analysis (TCEQ 2008). The analysis by Green (2003) was not available at the time of the US EPA analysis and derivation of the acute RfC. The US EPA appears to have selected fetal body weights as the toxicological endpoint of interest (without completing benchmark dose modelling, *etc.* for any other endpoints) based on the original statistics presented in Hackett *et al.* (1987).

Benchmark dose modelling was completed by the TCEQ based on Hackett *et al.* (1987) data for reduction in extragestational weight gain and fetal weight gain (TCEQ 2008). BMCL and Critical Effect Size (CES – similar to the BMC in concept, but is intended for continuous data) values were calculated for both decreased male fetal body weights and maternal extragestational weight gain. The $BMCL_{1\ SD}$ for the most sensitive endpoint, reduction in extragestational weight gain, was calculated as 51.3 ppm (in comparison to the $BMCL_{05}$ of 55 ppm for decreased fetal body weights). Given that the $BMCL_{1\ SD}$ was derived from a developmental endpoint (the TCEQ notes that the maternal effects observed are correlated with fetal effects in the literature, thus are considered to be developmental in nature), the exposure duration was not adjusted to 1 hour due to potential sensitive windows of exposure (TCEQ 2008). The TCEQ (2008) applied dosimetry adjustments from animal-to-human exposure to the POD, calculating a $BMCL_{1SD}$ of 51.3 ppm for extragestational weight gain. The TCEQ (2008) applied an uncertainty factor of 30 to the $(BMCL_{1\ SD})_{HEC}$ to account for interspecies variability (3) and intraspecies variability (10). This results in a 6-hour acute ReV value of 3,700 µg/m³ (1.71 ppm). Although it is recognized that the TCEQ (2008) assessment is more recent and takes additional information into account, the acute TCEQ ReV was not selected for use in the acute effects assessment. The approaches used by the two agencies (US EPA and TCEQ) are different enough that there is uncertainty as to which agency-derived value is the most protective of human health. Given this uncertainty, the more conservative US EPA value was selected for use in the acute effects assessment.

The OEHHA (2014) has developed an acute REL of 660 µg/m³, also based on the Hackett (1987) study described above for the US EPA value and the Green *et al.* (2003) re-analysis cited by the TCEQ. Based on the Green *et al.* (2003) re-analysis, the OEHHA derived a $BMCL_{05}$ of 17.7 ppm (40,000 µg/m³). This $BMCL_{05}$ was further adjusted to a HEC through the application of a dosimetric adjustment factor (DAF). A DAF of 1.68 was calculated by the OEHHA based on the ratio of human and animal blood/air partition coefficients for 1,3-butadiene estimated from mouse and human PBPK models. The HEC was calculated to be approximately 66,000 µg/m³. This $BMDL_{05}$ was further adjusted through the application of an uncertainty factor of 100 to account for intraspecies toxicodynamic differences (10), intraspecies toxicokinetic variability (3), and interspecies toxicodynamic variability (3). The latter uncertainty factor was selected to be 3 by the OEHHA on the basis that mice more actively metabolize 1,3-butadiene to reactive epoxides. The approaches used by the two agencies (US EPA and OEHHA) are different enough that there is uncertainty as to which agency-derived value is the most protective of human health. Given this uncertainty, the more conservative US EPA value was selected for use in the acute effects assessment.

C2.2 Chronic Inhalation Exposure Limits

Table 2-2
Chronic Inhalation Exposure Limits for 1,3-Butadiene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013, 2012
BC MOE[1]	–	2	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	RsC	1.7	Health Canada 2010, 2004
OEHHA	RsC REL	0.06 2	OEHHA 2009 OEHHA 2014, 2013
RIVM	CR	0.3	RIVM 2009
TCEQ	ReV ESL - cancer	33 20	TCEQ 2013, 2008
US EPA	RfC RsC	2 0.3	US EPA 2014, 2002
WHO	–	–	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

The US EPA (2014, 2002) bases its inhalation unit risk of 3×10^{-5} per µg/m³ on the Health Canada (1998) analysis of the leukemia incidence rates in styrene-butadiene rubber workers (n = 15,000) from eight different facilities. The key study evaluated by Health Canada (1998) was a cohort study by Delzell *et al.* (1996). 1,3-butadiene exposures to individual workers were estimated to derive cumulative exposure estimates for each worker. The follow-up period with workers was about 49 years. Both Delzell *et al.* (1996) and Health Canada (1998) conducted dose-response modelling for leukemia incidence, and the Health Canada (1998) modelling served as the basis for the US EPA assessment. Adjustments to the data were made for benzene and styrene exposure, to focus the statistics on 1,3-butadiene. The occupational exposures from Delzell *et al.* (1996) were adjusted for continuous exposure (240/365 days × 10/20 m³/day), and potential risks up to age 85 were predicted for the workers. Age-specific mortality rates for all races and genders were used to distinguish leukemia deaths from all-cause mortality rates. The US EPA also applied a linear rate model and leukemia incidence rate date from 1994 to 1998 to estimate leukemia rates. From the incidence rate data, a 95% lower confidence limit of the exposure concentration associated with a 1% increased risk (LEC_{01}) was calculated to be 0.25 ppm. The US EPA then conducted low-dose linear extrapolation (assuming that zero exposure is associated with zero risk), resulting in a predicted unit risk estimate of 0.04 per ppm 1,3-butadiene. An adjustment factor of 2 was applied by the US EPA to account for the potential for tumours to occur at other sites in humans, and also to account for potential differences in sex-sensitivity to 1,3-butadiene carcinogenicity. The resulting value equates to an RsC of 0.3 µg/m³ based on incidence of leukemia. This value was selected for use in the carcinogenic assessment of 1,3-butadiene as it represents the most relevant value to human health out of those evaluated.

An RsC of 1.7 µg/m³ was developed by Health Canada (2004) from a tumorigenic concentration (TC_{01}) of 1.7 mg/m³ based on the incidence of leukemia in 15,649 workers in the same

epidemiological study (Delzell *et al.* 1996) considered by the US EPA in their derivation of the unit risk estimate. As the Health Canada value uses the same data set as the US EPA, but is less conservative than the US EPA RsC, the Health Canada RsC was not selected for use in the chronic effects assessment.

RIVM (2009) adopted the US EPA (2002) RsC value described above as the human chronic inhalation value.

The OEHHA (2009) derived a unit risk estimate of 0.00017 per µg/m³ for 1,3-butadiene based on the incidence of lung alveolar and bronchoalveolar tumours in female mice. This unit risk estimate equates to an RsC of 0.06 µg/m³. In NTP (1984), male and female mice were exposed to 0, 652 or 1,250 ppm of 1,3-butadiene for 6 hours/day, 5 days/week for a duration of 60 weeks (males) or 61 weeks (females). Mortality resulted from malignant neoplasms of the heart, lung, mammary gland, ovaries, forestomach and liver, as well as hematopoietic lymphoma. The majority of these tumours were observed in control and exposed animals. Differences in incidence rates were found to be significantly higher in exposed animals than in controls for tumours of the heart, lymphoma, lung, mammary, ovary and forestomach. In the Melnick *et al.* (1990) study male and female mice were exposed to 0, 6.25, 20, 62.5, 200 and 625 ppm 1,3-butadiene for 40 or 65 weeks. Significantly increased incidences of cardiac hemangiosarcomas, hematopoietic lymphomas, squamous cell neoplasms in the forestomach, alveolar-bronchiolar neoplasms and mammary gland adenocarcinomas. Similar to the NTP study, the majority of these tumours were observed in control and exposed animals at varying rates. Statistical significance comparisons between control and exposure groups were not provided by the OEHHA (2000). The OEHHA (2000) selected the incidence of lung alveolar and bronchoalveolar tumours in female mice as the critical effect. Given that the US EPA and Health Canada consider there to be sufficient human data available for the development of a human-based RsC, the OEHHA animal-based RsC was not used in the chronic effects assessment.

The US EPA (2014, 2002) has derived an RfC of 2 µg/m³ based on ovarian atrophy in a 2-year mouse inhalation study. In the key study (NTP 1993), male and female B6C3F_1 mice (70 per group per sex) exposed to 0, 6.25, 20, 62.5, or 200 ppm 1,3-butadiene, 6 hours per day, 5 days per week for up to 103 weeks. Two additional groups of mice (90 male, 90 female) also were exposed to 625 ppm on the same exposure schedule. Up to 10 animals from each group were examined after 9 and 15 months of exposure. Survival was significantly decreased in both sexes above 20 ppm, primarily due to tumours. After 9 months of exposure, adverse effects on blood clinical chemical parameters was evident in the two higher exposure groups in males and females (625 ppm and 200 ppm in females, 625 and 62.5 ppm in males). The NTP (1993) determined that the effects were the result of a macrocytic anemia in the bone marrow of these mice. Testicular atrophy was observed on at 625 ppm. In females, ovarian atrophy was observed at 625 and 200 ppm after 9 months. By 15 months of exposure, mice exposed to 20 ppm and above had ovarian atrophy. After 2 years of exposure, ovarian atrophy was evident at all exposure concentrations. The US EPA conducted benchmark dose modelling on the ovarian atrophy data, discarding the high-dose group due to a high rate of early mortality (625 ppm). A BMC_{10} and a $BMCL_{10}$ of 1.0 ppm (2,250 µg/m³) and 0.88 ppm (1,980 µg/m³) were derived, incorporating adjustment for continuous exposure (6/24 hour, 5/7 days per week). The US EPA applied an uncertainty factor of 3 for interspecies extrapolation to the $BMCL_{10}$, 10 for intrahuman variability, 3 for database deficiencies, and 10 for the use of 10% effect level (similar to a LOAEL to NOAEL factor). This value was selected for use in the chronic non-cancer assessment of 1,3-butadiene.

The OEHHA (2014, 2013) has derived a non-carcinogenic REL of 2 µg/m³, based on the same study as the US EPA (NTP 1993). From this study, benchmark dose modelling was completed using the ovarian atrophy data set. A $BMCL_{05}$ of 1.01 ppm was calculated, and then adjusted for continuous exposure to 0.180 ppm. A further adjustment was made using a DAF. A DAF of 1.68 was calculated by the OEHHA based on the ratio of human and animal blood/air partition coefficients for 1,3-butadiene estimated from mouse and human PBPK models. This DAF was applied to the adjusted $BMCL_{05}$ of 0.180 ppm, resulting in HEC of 0.302 ppm (668 µg/m³). The OEHHA applied an uncertainty factor of 100 to account for intraspecies differences in toxicokinetics (10), intraspecies variability in toxicodynamics (3), interspecies variability in toxicodynamics (10).

The TCEQ (2013, 2008) has developed a chronic ReV of 33 µg/m³ for 1,3-butadiene based on the NTP (1993) data set (same as the US EPA and OEHHA non-cancer values described above). Benchmark dose modelling was conducted using the ovarian atrophy data in females. The $BMCL_{05}$ was used as the POD, as the TCEQ notes that a benchmark response level of 5% severe effects such as ovarian atrophy and it is considered a conservative NOAEL surrogate (compared to the 10% excess risk approach used by the US EPA). A $BMCL_{05}$ of 0.462 ppm (1,040 µg/m³) was calculated from dose-response data that had already been adjusted for continuous exposure before analysis. A default RGDR value of 1 was applied to this value in the absence of substance-specific information. The TCEQ applied an uncertainty factor of 30 to account for interspecies differences (10) and intrahuman variability (3). This value was not selected in favour of the more conservative value from the US EPA. The difference between the two doses arises from the benchmark dose modelling approaches used.

The TCEQ (2013) also provides a linear effects screening level (ESL) of 20 µg/m³ based on a cancer endpoint. A thorough review of Denzell's findings by the Health Review Committee (HEI 2006) confirmed the exposure response relation between increasing cumulative exposures to butadiene and the linear increase in the relative rate of leukemia mortality. Sathiakumar and Delzell. (2007) conducted an exposure estimate validation study using updated butadiene exposure estimates, then dose-response modelling was conducted based on the updated studies (Cheng et al. 2007; Sielken *et al.* 2007). This value was not used, given the existence of a defensible and more conservative value.

The BC MOE (2014) has derived a vapour standard of 2 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not available; therefore, this value was not selected for use.

C2.3 Oral Exposure Limits

1,3-Butadiene was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, 1,3-butadiene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C2.4 Summary of Exposure Limits

Table 2-3
Summary of Exposure Limits Selected for 1,3-Butadiene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	24-hour	Inhalation	RfC	15	µg/m³	US EPA 2014, 2002	Reproductive / developmental effects
Chronic	Annual	Inhalation	RsC	0.3	µg/m³	US EPA 2014, 2002	Leukemia
			RfC	2	µg/m³	US EPA 2002	Reproductive / developmental effects
		Multiple Exposure Pathway	n/a	n/a	µg/kg/day	n/a	n/a

n/a = Not applicable

C2.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2012. Toxicological Profile for 1,3-butadiene. September 2012. US Department of Health and Human Services, Public Health Service.

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

Cheng, H., Sathiakumar, N., Graff, J., *et al.* 2007. 1,3-butadiene and leukemia among synthetic rubber industry workers: Exposure–response relationships. Chem Biol Inter 166:15-24. Cited in: TCEQ 2008.

Christian, M.S. 1996. Review of reproductive and developmental toxicity of 1,3-butadiene. Toxicology 113:137-143. Cited in: TCEQ 2008

Delzell, E, Sathiakumar, N. and Hovinga, M. 1996. A follow up study of synthetic rubber workers. Toxicology 113:182 189.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued February 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Green, J.W. 2003. Statistical analysis of butadiene mouse data from Hackett *et al.* (1987). Study completed for American Chemistry Council. Laboratory Project ID: Dont-13474. Sponsor Contract ID: OLF-114.0-BD-stat-DHL.

Hackett, P.L. Sikov, M.R., Mast, T.J., et. al. 1987. Inhalation developmental toxicology studies: Teratology study of 1,3 butadiene in mice. Final report No. NIH-401-ES-40131. PNL-6412, UC-48. Prepared for: National Institute of Environmental Health Sciences, National Toxicology Program.

Health Canada. 1998. Canadian Environmental Protection Act Priority Substances List Health Assessment: 1,3 Butadiene. Draft for second stage peer review. Ottawa. March 1998. Additional information was provided by Health Canada for calculation of the confidence intervals (personal communication from Michael Walker to Leslie Stayner, 18 June 1999).

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

HEI (Health Effects Institute). 2006. An updated study of mortality among North American synthetic rubber industry workers. Prepared by E. Delzell, N. Sathiakuman, J. Graff, *et al.* Health Effects Institute Research Report Number 132. Cited in: TCEQ 2008.

Melnick, R.L., Huff, J.E., Chou, B.J. and Miller, R.A. 1990. Carcinogenicity of 1,3 butadiene in C57BL/6 × C3HF1 mice at low exposure concentrations. Cancer Research 50:6592 6599. Cited in: US EPA 2002.

NTP (National Toxicology Program). 1984. Toxicology and Carcinogenesis Studies of 1,3 Butadiene (CAS No. 106 99 0) in B6C3F_1 Mice (inhalation studies), NTP TR 288. NIH Publication No. 84 2544. National Toxicology Program, National Institutes of Health, Washington, DC.

NTP (National Toxicology Program). 1993. NTP technical report on the toxicology and carcinogenesis studies of 1,3-butadiene (CAS No. 106-99-0) in B6C3F_1 mice (inhalational studies), NTP TR434, NIH Publication No. 93-3165, US Department of Health and Human Services Public Health Service. National Institute of Health, Research Triangle Park, NC.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Determination of Noncancer Chronic Reference Exposure Levels Chronic Toxicity Summary. 1,3 Butadiene (page 48). Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OEHHA (California Office of Environmental Health Hazard Assessment). 2013. 1,3-Butadiene Reference Exposure Levels. CAS 106-99-0. Final. July 2013. CA. Available at: http://www.oehha.ca.gov/air/chronic_rels/pdf/072613bentCREL.pdf

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2009. Environmental Risk Limits for 1,3-butadiene. Letter Report 601782014/2009.

Sathiakumar, N. and Delzell. E. 2007. A follow-up study of women in the synthetic rubber industry: Study methods. Chem Biol Inter 166: 25-28. Cited in: TCEQ 2008

Sielken, R.L., Valdez-Flores, C., Gargas, M.L., *et al.* 2007. Cancer risk assessment for 1,3-butadiene: Dose response modeling from an epidemiological perspective. Chem Biol Inter 166: 140-49. Cited in: TCEQ 2008.

TCEQ (Texas Commission on Environmental Quality). 2008. 1,3 Butadiene (CAS Registry Number: 106 99 0). Development Support Document, Final, August 2008. Prepared by: Roberta L. Grant, 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/butadiene,_1-3-_106-99-0_final.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2002. Health Assessment of 1,3-butadiene. National Center for Environmental Assessment-Washington Office. Office of Research and Development. Washington, DC. October 2002. EPA/600/P-98/001F

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

C3.0 ACETALDEHYDE

C3.1 Acute Inhalation Exposure Limits

Table 3-1
Acute Inhalation Exposure Limits for Acetaldehyde

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO	90	ESRD 2013
OEHHA	1-hour REL 8-hour REL	470 300	OEHHA 2014, 2008
OMOE	24-hour Standard	500	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OEHHA (2014, 2008) derived two acute RELs (1-hour and 8-hour). The acute 1-hour REL is based on a study by Prieto (2000), the purpose of which was to establish the concentration at which a 20% decrease in forced expiratory volume (FEV_1) occurred in 1 second – an endpoint selected by the authors as being of interest with respect to the acute effects of acetaldehyde inhalation (OEHHA 2008). Subjects were exposed via mouth inhalation to air concentrations ranging from 150 to 1,200 mg/m³, with a geometric mean of 527 mg/m^3, and a lower 95% confidence interval of about 142 mg/m³. This concentration was selected as the LOAEL for effects on expiratory volume in asthmatics, and this value was used as the basis of the acute REL. Two follow-up studies (Prieto *et al.* 2002a, b) were conducted and considered in the development of the REL. Prieto *et al.* (2002a) compared the respiratory response to acetaldehyde with known bronchoconstricting compounds (methacholine and adenosine-5'-monophosphate), and the repeatability of the respiratory response to acetaldehyde. Prieto *et al.* (2002b) also incorporated a healthy subject group, and subjects with allergic rhinitis or asthma. Subjects with allergic rhinitis and asthma both demonstrated significant differences from the healthy subject group with respect to the occurrence of FEV_1 decreases of 20%. The geometric mean exposure concentrations associated with significant bronchoconstriction in the rhinitis group and asthmatic group were determined to be 2,166 mg/m³ and 1,136 mg/m³, respectively. A study by Silverman *et al.* (1946) was cited by OEHHA (2008) as a supporting study to the acute REL. In this study, twelve people were exposed to acetaldehyde for 15 minutes at concentrations ranging from 25 to above 200 ppm (not specified). Nose and throat irritation were reported at 200 ppm and above, evidence of eye irritation was not apparent at 200 ppm.

The LOAEL of 142 mg/m³ observed in Prieto *et al.* (2000) was selected by OEHHA (2008). A cumulative uncertainty factor of 300 was applied to this LOAEL to account for the use of a LOAEL instead of a NOAEL (10), and to account for intraspecies variability (30). The factor of 30 was applied to account for the potential for exacerbation of asthma in children (as the subjects examined were all adults) and the potential for hyper-responsiveness to methacholine (OEHHA 2008). The result is an REL of 470 µg/m³. Although the exposure duration in the key

study was 2 to 4 minutes, the OEHHA (2008) did not convert the REL using Haber's law. Instead it states that the REL represents a level at which intermittent 1-hour exposures are not expected to result in adverse health effects. As such, the REL of 470 µg/m³ was used in the assessment as a 1-hour exposure limit, based upon nasal and respiratory effects. The OEHHA (2008) states that this REL also is protective against the effects of eye irritation.

The OEHHA (2008) also provides an 8-hour REL of 300 µg/m³ to be protective of repeated 8-hour exposures to acetaldehyde. This value was based on a 4-week study in Wistar rats exposed to 0, 273, 728, 910, 1,820, 4,004, or 9,100 mg/m³ acetaldehyde for 6 hours/day, 5 days/week. Significant degeneration of the olfactory epithelium was observed at concentrations of 728 mg/m³ and above (the study LOAEL). As such, 273 mg/m³ was identified as the NOAEL. Benchmark dose modelling was completed on the study data, and the BMC_{05} was identified as being 178 mg/m³. The BMC_{05} was further converted to a human equivalent concentration of 242 mg/m³ using pharmacokinetic modelling specific to the study species and acetaldehyde. Adjustments were made to account for continuous exposure (6/24 hours × 5/7 days) resulting in an adjusted BMC_{05} of 86.5 mg/m³. A cumulative uncertainty factor of 300 was applied to account for subchronic exposure (√10), interspecies differences (√10), inter-individual variation (√10), and potential for exacerbation of asthma in children (10). The result is the 8-hour REL of 300 µg/m³. This value was not used in the acute effects assessment, given that it is based on animal rather than human data, and involves repeated dose exposures as opposed to instantaneous effects (which are well documented in support of the OEHHA 1-hour REL).

The ESRD (2013) presents a 1-hour AAQO for acetaldehyde of 90 µg/m³. This objective, however, was adopted from the TCEQ, which developed its short-term ESL based on odour perception. Given that the ESRD AAQO is not health-based, it was not used to evaluate the potential short-term health risks associated with acetaldehyde.

The OMOE (2012) has derived a 24-hour standard of 500 µg/m³; however, adequate supporting documentation is not available. As a result, the study team is unable to comment on the scientific merit of this standard and it was not used in the acute effects assessment.

C3.2 Chronic Inhalation Exposure Limits

Table 3-2
Chronic Inhalation Exposure Limits for Acetaldehyde

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE[1]	–	4.5	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC_{05} TC	17.2 390	Health Canada 2010, 2004
OEHHA	RsC REL	3.7 140	OEHHA 2009 OEHHA 2014, 2008
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	RfC RsC	9 5	US EPA 2014, 1991a US EPA 2014, 1991b
WHO	–	–	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

Health Canada (2004) derived a TC_{05} of 86 mg/m³, which is associated with a 5% increase in nasal tumours in rats. In the key study (Woutersen *et al.* 1986), male and female Wistar rats were exposed to 0, 750, 1,500, or 3,000 ppm (equivalent to 0, 1,350, 2,700, or 5,400 mg/m³) of acetaldehyde for 6 hours/day, 5 days/week for 28 months. Squamous cell carcinomas and adenocarcinomas were observed in olfactory and respiratory epithelia in the nasal cavities of exposed animals. No lung tumours were observed (Woutersen *et al.* 1986). The TC_{05} of 86 mg/m³ was derived using a multistage model, with adjustment for intermittent to continuous exposure (6/24 hours × 5/7 days). Based on a risk level of one in 100,000, the TC_{05} equates to a risk specific concentration of 17.2 µg/m³, based on incidence of nasal carcinoma. The adjusted value of 17.2 µg/m³ was used in the chronic carcinogenic inhalation assessment of acetaldehyde.

The OEHHA (2009) derived a URE of 2.7E-06 $(\mu g/m^3)^{-1}$ for acetaldehyde (equivalent to an RsC of 3.7 µg/m³) based on the same study used by Health Canada (Woutersen *et al.* 1986), described above. The OEHHA (2009) adjusted exposure estimates for intermittent exposure. Linearized multistage modelling was conducted, and the 95% upper confidence limit was determined. Exposures were then scaled based on body weight by the OEHHA. The OEHHA (2009) value was not used in the chronic inhalation effects assessment as the tumours observed in the study animals appear to be in tissues that have first contact with inhaled acetaldehyde, making the dose-scaling adjustments based on body weight less relevant.

The US EPA (2014, 1991b) also presents a quantitative estimate of carcinogenic risk from inhalation exposure. Its inhalation unit risk of 2.2E-06 per µg/m³ equates to an RsC of 5 µg/m³ (based on a risk level of one in 100,000). The US EPA inhalation unit risk was not used in the current assessment for the following reasons:

- The US EPA last reviewed its limit in 1991, while the Health Canada value is more recent (published in 2004). The Health Canada and US EPA limits are based on studies conducted by the same researchers.
- The Health Canada limit is based on a 1986 study by Woutersen *et al.*, which is more recent than the work completed by Woutersen and Appelman in 1984, upon which the US EPA limit is based.
- The scientific rationale for the Health Canada limit is considerably more detailed than what the US EPA provides in support of its limit.

Health Canada (2004) presents a non-cancer TC of 390 µg/m³ based on the incidence of olfactory lesions after 4 weeks of exposure. Health Canada applied a benchmark dose model and calculated a BMC_{05} based on tumour incidence data from Appelman *et al.* (1982, 1986). Health Canada (2000) states that "although the data were derived from short-term studies, the incidence of degenerative changes in olfactory epithelium was not dissimilar to that observed in the same strain of rats in the long-term carcinogenesis bioassay at similar concentrations, conducted by Woutersen *et al.* (1986)." Health Canada used the THRESH program to calculate a BMC_{05} (the concentration associated with a 5% increase in the incidence of nasal olfactory epithelial lesions) for male Wistar rats of 357 mg/m^3, and a lower 95% confidence limit ($BMCL_{05}$) of 218 mg/m^3. Health Canada then calculated a Tolerable Concentration (TC) for non-neoplastic lesions using the following formula:

$$TC = \frac{218\ mg/m^3}{100} \times \frac{6}{24} \times \frac{5}{7}$$

$$TC = 0.390\ mg/m^3$$

Where 100 is the cumulative uncertainty factor to account for interspecies (10) and intraspecies variability (10). The Health Canada TC of 390 µg/m³ based on nasal olfactory lesions was used in the chronic non-carcinogenic inhalation assessment.

The US EPA (2014, 1991a) derived an RfC of 9 µg/m³ based on the degeneration of olfactory epithelium in rats exposed to 0, 150, or 500 ppm of acetaldehyde for a duration of 4 weeks using the same studies Health Canada relied on. The study authors identified 150 ppm (273,000 µg/m³) as the NOAEL. The NOAEL was further adjusted for time (273,000 µg/m³ × 6/24 × 5 days/week = 48,750 µg/m³) and human equivalency (48,750 µg/m³ × RGDR of 0.178 = 8,700 µg/m³). A cumulative uncertainty factor of 1,000 was applied to the $NOAEL_{(HEC)}$ to account for intraspecies sensitivity (10), use of a subchronic study (10) and interspecies differences (10). The resultant RfC of 9 µg/m³ based on the degeneration of olfactory epithelium was not used as a chronic inhalation limit for the non-carcinogenic assessment of acetaldehyde for the following reasons:

- The US EPA applied a 10-fold uncertainty factor for the use of subchronic data. However, Health Canada (2000) considered it inappropriate to include an uncertainty factor for the use of a subchronic study, as there is no indication that the severity of the critical effect increases with duration of exposure.
- The US EPA last reviewed its limit in 1991, while the Health Canada value is more recent (published in 2004). The Health Canada and US EPA limits are based on studies conducted by the same researchers.
- The US EPA's confidence in the inhalation RfC for acetaldehyde is low.
- Health Canada used a benchmark concentration model (as opposed to a NOAEL) to derive its exposure limit.

The OEHHA (2014, 2008) also derived a chronic REL of 140 µg/m³ based on the same Appelman *et al.* (1982, 1986) studies that Health Canada and the US EPA relied on for the

derivation of their chronic exposure limits. Like Health Canada, the OEHHA developed its limit based in part on benchmark concentration modelling. However, the OEHHA applied an uncertainty factor of √10 for the use of subchronic data, which Health Canada considers inappropriate. For this reason, the OEHHA REL was not used in the current assessment.

The BC MOE (2014) developed a vapour standard of 4.5 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, this value was not selected for use, given that supporting documentation was not available.

C3.3 Oral Exposure Limits

Table 3-3
Chronic Oral Exposure Limits for Acetaldehyde

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	– –	– –	Health Canada 2010 Health Canada 2013
OEHHA	RsD –	1 –	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2014

– = Not available

The OEHHA (2009) presents an oral slope factor of 1E-02 (mg/kg bw/d)$^{-1}$ (equivalent to an RsD of 1 µg/kg bw/d). However, this value is based on the chronic inhalation study by Woutersen *et al.* (1986) described above. Given the uncertainty associated with route-to-route extrapolation for a non-systemic effect such as nasal tumours, this value was not selected for use in the assessment.

As no defensible chronic oral limit was available from the sources listed above, the search was expanded to include PPRTVs from the US EPA (2013b). Still no chronic oral limit could be identified. As a result, acetaldehyde could not be assessed in the chronic multiple pathway assessment.

C3.4 Summary of Exposure Limits

Table 3-4
Summary of Exposure Limits Selected for Acetaldehyde

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	REL	470	µg/m³	OEHHA 2014, 2008	Eye, nasal and respiratory irritants
Chronic	Annual	Inhalation	RsC	17.2	µg/m³	Health Canada 2010, 2004	Nasal tumours
			RfC	390	µg/m³	Health Canada 2010, 2004	Nasal irritants
		Multiple Exposure Pathway	RsD	–	µg/kg bw/d	–	–
			RfD	–	µg/kg bw/d	–	–

– = Not available

C3.5 References

Appelman, L.M., R.A. Woutersen, and V.J. Feron 1982. Inhalation toxicity of acetaldehyde in rats. I. Acute and subacute studies. Toxicology 23: 293-307.

Appelman, L.M., R.A. Woutersen, V.J. Feron, R.N. Hoofman and W.R.F. Notten 1986. Effect of variable versus fixed exposure levels on the toxicity of acetaldehyde in rats. J Appl Toxicol 6: 331-336.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada 2000. Priority Substances List Assessment Report: Acetaldehyde. ISBN 0-662-28654-5.

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. TSD for Noncancer RELs. Acetaldehyde Reference Exposure Levels. Appendix D1. December 2008. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=5

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Prieto, L., F. Sanchez-Toril, B. Brotons, S. Soriano, R. Casan and J.L. Belenguer. 2000. Airway responsiveness to acetaldehyde in patients with asthma: relationship to methacholine responsiveness and peak expiratory flow variation. Clin Exp Allergy 30(1): 71-78. Cited in: OEHHA 2008b

Prieto, L., F. Sanchez-Toril, V. Gutierrez and M.J. Marin. 2002b. Airway responsiveness to inhaled acetaldehyde in subjects with allergic rhinitis: Relationship to methacholine responsiveness. Respiration 69(2): 129-135.

Prieto, L., V. Gutierrez, A. Cervera, and J. Linana. 2002a. Airway obstruction induced by inhaled acetaldehyde in asthma: repeatability and relationship to adenosine 5'-monophosphate responsiveness. J Invest Allergol Clin Innunol. 12(2):91-98

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

Silverman, L., H.F. Schulte and M.W. First. 1946. Further studies on sensory response to certain industrial solvent vapours. J Ind Hyg Toxicol 28(6):262-266.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1991a. IRIS Summary of Acetaldehyde (75-07-7). Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0290.htm#refinhal

US EPA (United States Environmental Protection Agency). 1991b. IRIS Summary of Acetaldehyde (75-07-7). Carcinogenicity Assessment for Lifetime Exposure. Available at: http://www.epa.gov/ncea/iris/subst/0290.htm#carc

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

Woutersen, R.A. and L.M. Appelman. 1984. Lifespan inhalation carcinogenicity study of acetaldehyde in rats. III. Recovery after 52 weeks of exposure. Report No. V84.288/190172. CIVO-Institutes TNO, The Netherlands. Cited in: US EPA 1991b.

Woutersen, R.A., L.M. Appelman, A. Van Garderen-Hoetmer and V.J. Feron. 1986. Inhalation toxicity of acetaldehyde in rats. III. Carcinogenicity study. Toxicology. 41: 213-231.

C4.0 ACROLEIN

C4.1 Acute Inhalation Exposure Limits

Table 4-1
Acute Inhalation Exposure Limits for Acrolein

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	1-hour MRL	6.9	ATSDR 2013, 2007
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	1-hour REL 8-hour REL	2.5 0.7	OEHHA 2014, 2008
OMOE	1-hour Standard 24-hour Standard	4.5 0.4	OMOE 2012
TCEQ	1-hour ReV	11	TCEQ 2014a,b
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OEHHA (2014, 2008) has derived a 1-hour acute REL of 2.5 µg/m³ based on the geometric mean of two acute REL values developed from two acute exposure studies employing human subjects – Darley *et al.* (1960) and Weber-Tschopp *et al.* (1977). Darley *et al.* (1960) exposed 36 healthy people to 0, 0.06, 1.3 to 1.6, or 2.0 to 2.3 ppm acrolein for 5 minutes. Acrolein was dissolved in water and administered via face masks equipped with respirators such that only the eyes were exposed to acrolein. Subjects rated the degree of eye irritation every 30 seconds during exposure. A LOAEL of 0.06 ppm (~0.14 mg/m³) was identified. A cumulative uncertainty factor of 60 was applied to account for the use of a LOAEL instead of a NOAEL for a relatively mild adverse effect (6), and intraspecies variability (to protect against the exacerbation of asthma in children - 10). The result is an acute 1-hour REL of 2.3 µg/m³.

In a chamber study by Weber-Tschopp *et al.* (1977), 54 healthy human volunteers were exposed to increasing concentrations (0 to 0.6 ppm, equivalent to 0 to 1.4 mg/m³) of acrolein for 40 minutes, while 46 healthy human volunteers were continuously exposed to 0.3 ppm (equivalent to 0.68 mg/m³) for 60 minutes. Another group of individuals (n=42) were exposed to various acrolein concentrations (not specified) for 90 seconds. Subjective eye and nasal irritation were reported and eye-blink and respiratory rates were measured during the exposures. For the exposure group with increasing levels of acrolein, significantly higher eye irritation and nasal irritation relative to the control group were reported at 0.07 ppm and 0.26 ppm, respectively. As well, respiratory rates decreased with increasing acrolein concentrations, with changes being significant between 0.09 and 0.30 ppm acrolein. For the continuous exposure group, subjective eye and nasal irritation increased quickly during the initial 20 minutes of exposure and plateaued by 40 minutes. Respiratory rates decreased by 10% and eye blink rates doubled after 10 minutes of exposure. On this basis, the OEHHA (2008) identified a LOAEL of 0.07 ppm for subjective ocular irritation. A cumulative uncertainty factor of 60 was applied to the LOAEL to account for the use of a LOAEL instead of a NOAEL for a relatively mild adverse effect (6), and intraspecies variability (10 – to protect against the exacerbation of asthma in children). The result is an acute 1-hour REL of 2.7 µg/m³.

The OEHHA (2008) calculated the geometric mean of the two acute RELs to derive the acute 1-hour REL for acrolein of 2.5 µg/m³. Although no conversion was made for a 5-minute to a 1-hour exposure, the OEHHA (2008) states that the acute REL is intended to be protective of intermittent 1-hour exposures. The acute REL of 2.5 µg/m³ based on eye and nasal irritation, respiratory irritation was selected for use in the acute effects assessment.

The OEHHA (2008) also developed an 8-hour REL based on a 65-day study in which Fischer 344 rats were exposed to 0.02 to 1.8 ppm for 6 hours/day, 5 days/week over the 65-day period. The 8-hour REL was not used in the acute effects assessment as it is based on subchronic exposure data in animals, as opposed to acute human data used in the 1-hour limit.

The OMOE (2012) presents a 1-hour value of 4.5 µg/m³, and a 24-hour standard of 0.4 µg/m³ for acrolein based on health. However, as no supporting documentation for the 24-hour standard are available, these values were not considered for the acute inhalation assessment.

The TCEQ (2014a,b) has derived an acute 1-hour ReV for acrolein of 11 µg/m³, based on the Weber-Tschopp *et al.* (1977) study. The TCEQ (2010) states that the 40-minute exposure group experienced the highest degree of irritation, as reported in questionnaires completed by subjects every 5 minutes. Eye irritation was reported at 0.3 ppm, throat irritation at 0.4 ppm, and significantly decreased respiratory rates were recorded at 0.6 ppm. From this 40-minute exposure group, the exposure concentration of 0.3 ppm was identified by the TCEQ as a LOAEL. No adjustments for continuous exposure were applied. An uncertainty factor of 63 was applied to account for the use of a LOAEL for a mild effect (6.3) and intraspecies differences (10). The TCEQ value was not selected for use in the assessment, primarily because the OEHHA (2008) selected a lower LOAEL value than the TCEQ for the Weber-Tschopp *et al.* (1977) study. The analysis by the OEHHA appears to be more representative of the findings of the study, as effects were reported below 0.3 ppm. It is possible that the TCEQ value may not be adequately conservative as a result of the LOAEL selected.

The ATSDR (2013, 2007) has derived an acute MRL of 0.003 ppm (0.0069 mg/m³) based on decreased respiratory rate and nose and throat irritation reported in the Weber-Tschopp *et al.* (1977) study. Forty-six volunteers were exposed to a gradually increasing concentration of acrolein for 40 minutes. Participants subjectively scored irritancy at 5 minute intervals as the concentrations increased from 0 to 0.6 ppm (0 to 1.3 mg/m³). The ATSDR identified a LOAEL for nose irritation of 0.26 ppm (0.60 mg/m³) and then applied an uncertainty factor of 100 to the LOAEL to account for the use of a LOAEL instead of a NOAEL (10) and intraspecies variability (10). Because the OEHHA developed a lower exposure limit based on the same study, ATSDR's MRL of 6.9 µg/m³ was not used in the current assessment.

C4.2 Chronic Inhalation Exposure Limits

Table 4-2
Chronic Inhalation Exposure Limits for Acrolein

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE[1]	–	2	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	0.4	Health Canada 2004 Government of Canada 2000
OEHHA	REL	0.35	OEHHA 2014, 2008
RIVM	–	–	RIVM 2009, 2001
TCEQ	ReV	2.7	TCEQ 2014, 2010
US EPA	RfC	0.02	US EPA 2014, 2003
WHO	–	–	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

The OEHHA (2014, 2008) has derived a chronic REL of 0.35 µg/m³ based on the incidence of nasal lesions in a subchronic rat inhalation study by Dorman *et al.* (2008). Groups of 12 adult male F344 rats were exposed to 0, 0.02, 0.06, 0.2, 0.6 or 1.8 ppm acrolein (approximately 0, 0.05, 0.14, 0.5, 1.4, or 4.1 mg/m³) acrolein *via* inhalation for 6 hours/day, 5 days/week for up to 13 weeks. Some animals were sacrificed after 4, 14, 30 and 65 days of exposure, and respiratory tract histopathology was examined at these intervals. A number of rats were included in a recovery group that was sacrificed 60-days post-exposure. A statistically significant decrease in body weight was observed in all acrolein exposed animals after 13 weeks of exposure, although the body weight effects were less pronounced and slower to develop at the lower dose levels. Mild hyperplasia of respiratory epithelia was observed at concentrations of 0.6 ppm and above after four days or more of exposure. The hyperplasia became more severe at 1.8 ppm, and squamous metaplasia also was observed. The most sensitive site within the nasal cavity was observed to be the lateral wall, although lesions were observed at other sites as well. Immunohistochemical analysis of epithelial cells indicated that immunoreactivity was not observed at 0.2 ppm, but was present at 0.6 ppm and 1.8 ppm. An exposure-related effect on olfactory epithelium also was observed in animals exposed to 1.8 ppm acrolein for four or more days. After four days, animals in the 1.8 ppm group displayed moderately severe olfactory neuronal degeneration and atrophy, and in some instances, marked olfactory neuron loss was observed. Effects became more severe with increasing exposure duration. Only partial recovery of the olfactory epithelium was observed after 65 days post-exposure. At 1.8 ppm, mild squamous metaplasia also was observed in the larynx and trachea, but no exposure-related effects were observed in the lungs. A NOAEL for nasal epithelial lesions was determined to be 0.2 ppm. Given the subchronic nature of the exposure, and that the toxicological endpoint did not involve trigeminal nerve irritation, this NOAEL was adjusted to account for intermittent exposure (*i.e.*, 6/24 hours × 5/7 days), resulting in a $NOAEL_{ADJ}$ of 0.036 ppm. A human equivalent concentration ($NOAEL_{HEC}$) was calculated by multiplying the duration-adjusted NOAEL by a dosimetric adjustment factor (DAF) of 0.85

derived by the OEHHA (2008). This DAF represents the ratio of the gas flux across olfactory epithelium in rats relative to humans, based on modeling conducted by Kimbell *et al.* (2001).

The $NOAEL_{HEC}$ was calculated to be 0.03 ppm (70 µg/m³). A cumulative uncertainty factor of 200 was applied to this $NOAEL_{HEC}$ to account for interspecies variability (a default value of √10 for potential toxicodynamic differences, and a value of 2 for toxicokinetic differences), subchronic to chronic extrapolation (√10), and intraspecies differences (10) to account for the potential for asthma exacerbation in children. The result is a chronic REL of 0.35 µg/m³ based on effects to the nasal epithelium. This value was selected for use in the chronic assessment of acrolein.

Health Canada (2004) has developed a tolerable concentration of 0.4 µg/m³ based on the lower benchmark concentration of 0.14 mg/m³ associated with a 5% increase in non-neoplastic lesions in the nasal respiratory epithelium of rats (Health Canada 2004; Government of Canada 2000; Cassee *et al.* 1996). A cumulative uncertainty factor of 100 was incorporated to account for interspecies variation (10) and intraspecies variation (10). The limit was further adjusted by Health Canada to account for continuous exposure (*i.e.*, rats were exposed intermittently for 6 hours/day so the limit was multiplied by 6/24 hours). Given that Health Canada's tolerable concentration is based on acute exposure (3 days) it was not used in the current chronic assessment of acrolein.

The TCEQ (2014a,b) has derived a chronic ReV of 2.7 µg/m³ on the same Dorman *et al.* (2008) study as the OEHHA REL. Information regarding study design is provided above in association with the OEHHA value. The TCEQ identified 0.2 ppm as a NOAEL for nasal epithelial hyperplasia and squamous metaplasia, and a LOAEL of 0.6 ppm for hyperplasia of the nasal cavity, septum and larynx. The TCEQ also identified a NOAEL of 0.6 ppm and a LOAEL of 1.8 ppm for olfactory epithelial inflammation and atrophy. However, the lowest NOAEL of 0.2 ppm was selected as the POD. Benchmark dose modelling was considered, but could not be used as a result of the response rates at the various concentrations. An adjustment to account for continuous exposure was conducted (0.2 ppm × 6/24 hours × 5/7 days), resulting in a $NOAEL_{ADJ}$ of 0.035 ppm. To convert this $NOAEL_{ADJ}$ to a $NOAEL_{HEC}$, the TCEQ applied an RGDR value of 1.0 as a default. 1The result of this conversion was a $NOAEL_{HEC}$ of 0.036 ppm. An uncertainty factor of 30 was applied to account for interspecies differences (3), and intraspecies variability (10). The TCEQ value was not used in the assessment, due to the availability of a more conservative value based on the same study by the OEHHA. The only differences between the OEHHA REL and the TCEQ ReV are the adjustment for toxicokinetic differences and the application of uncertainty factors – the selected key study and NOAEL are the same. The OEHHA REL was selected over the TCEQ due to the more specific approach used to evaluate differences in olfactory sensitivity between humans and rodents (the OEHHA used a DAF, while the TCEQ assumed a default ratio of 1 between animals and humans, resulting in no actual adjustment being made).

The US EPA (2014, 2003) has derived an inhalation RfC of 0.02 µg/m³ based on nasal lesions observed in a subchronic rat inhalation study conducted by Feron *et al.* (1978). Six Wistar rats, ten Syrian golden hamsters and two Dutch rabbits were administered 0, 0.4, 1.4, or 4.9 ppm acrolein in a whole-body exposure chamber for five days/week for 13 weeks. Histopathologic changes described as "slightly affected" were observed in the nasal cavity of one of the 12 rats exposed to 0.4 ppm (0.9 mg/m³) (US EPA 2003). Severity increased at the higher levels of exposure in all species, most clearly so in the rat. No nasal lesions were reported in other species at 0.4 ppm (0.9 mg/m³). Based on the concentration-related severity of lesions, the rat was identified as the most sensitive species. The US EPA identified a LOAEL of 0.4 ppm

(0.9 mg/m³) and adjusted the LOAEL to continuous exposure (*i.e.*, 6/24 hours × 5/7 days), resulting in a $LOAEL_{ADJ}$ of 0.16 mg/m³. In addition, the US EPA (2003) calculated the $LOAEL_{HEC}$ using the RGDR approach, where the duration-adjusted LOAEL for the rat was then multiplied by the $RGDR_{ET}$ to yield a $LOAEL_{HEC}$ of 0.02 mg/m³. The US EPA (2003) applied an uncertainty factor of 1,000 to the $LOAEL_{HEC}$ to account for extrapolation from rats to humans (3), intraspecies variability (10), adjustment from a subchronic to chronic study (10), and use of a minimal LOAEL (3). An uncertainty factor of 3 was used for interspecies variability because dosimetric adjustments were already made through the use of the RGDR methodology. This value was not selected for use, as the OEHHA (2008) value is based on more recent and robust study data and incorporates dosimetry modelling data instead of the RGDR approach.

The ATSDR (2013) has derived an intermediate inhalation MRL of 0.00004 ppm (0.09 µg/m³) based on the same Feron *et al.* (1978) study as the US EPA (2003). The end point identified for derivation of the MRL was nasal epithelial metaplasia in rats, based on a LOAEL of 0.4 ppm. As chronic inhalation exposure limits were available from other regulatory agencies, the ATSDR intermediate value was not used in the assessment.

The BC MOE (2014) has developed a vapour standard of 2 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not available. As such, this value was not selected for use.

C4.3 Oral Exposure Limits

Table 4-3
Chronic Oral Exposure Limits for Acrolein

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	–	–	Health Canada 2010
	–	–	Health Canada 2013
OEHHA	–	–	OEHHA 2009
	–	–	OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
US EPA	RfD	0.5	US EPA 2014, 2003
WHO	–	–	WHO 2014

– = Not available

The US EPA (2014, 2003) presents an RfD of 0.5 µg/kg bw/d for acrolein. This RfD was derived from a chronic oral rat study that reported a NOAEL of 0.05 mg/kg bw/day for decreased survival. Groups of male and female Sprague-Dawley rats received daily doses via gavage of 0, 0.05, 0.5 or 2.5 mg/kg acrolein in water. A suite of clinical, hematological and urinary parameters were recorded at 3, 6, 12 and 18 months and upon termination. Ten rats from each group (70/sex per group) were terminated after one year, while the remaining rats were terminated after two years. No effects on food consumption, body weight, or any of the clinical, hematological and urinary parameters were reported. The only exception was a statistically significant depression of creatinine phosphokinase at all dose levels and at most time intervals.

A significant reduction in survival was reported in the 2.5 mg/kg/dose groups after one year ($p<0.05$). Survival was marginally reduced in the 0.5 mg/kg/dose groups (p value not reported), and no differences in survival were reported between the 0.05 mg/kg/dose groups and the control groups. Significant associations between dosing and survival were also observed in the 2.5 mg/kg/dose and 0.5 mg/kg/dose groups after two years, but only among the female rats. Based on the dose-dependent increase in mortality, a frank effect level (FEL) of 0.5 mg/kg/day and a NOAEL of 0.05 mg/kg/day were identified. According to the US EPA (2014), a FEL is "a level of exposure or dose which produces irreversible, adverse effects at a statistically or biologically significant increase in frequency or severity between those exposed and those not exposed". The US EPA applied a cumulative uncertainty factor of 100 to the NOAEL to account for interspecies (10) and intraspecies variability (10), resulting in the RfD of 0.0005 mg/kg bw/day (0.5 µg/kg bw/day) for acrolein. This RfD was used as the chronic oral exposure limit in the multiple pathway assessment for acrolein.

C4.4 Summary of Exposure Limits

Table 4-4
Summary of Exposure Limits Selected for Acrolein

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	REL	2.5	µg/m³	OEHHA 2014, 2008	Eye, nasal, respiratory irritation
Chronic	Annual	Inhalation	RsC	–	µg/m³	–	–
			RfC	0.35	µg/m³	OEHHA 2014, 2008	Nasal irritation
		Multiple Exposure Pathway	RsD	–	µg/kg bw/d	–	–
			RfD	0.5	µg/kg bw/d	US EPA 2014, 2003	Decreased survival

– = not available

C4.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2007. Toxicological Profile for Acrolein. US Department of Health and Human Services, Public Health Service. Available at: http://www.atsdr.cdc.gov/ToxProfiles/tp124.pdf

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Cassee, F., J. Groten and V. Feron. 1996. Changes in the nasal epithelium of rats exposed by inhalation to mixtures of formaldehyde, acetaldehyde and acrolein. Fundamentals of Applied Toxicology 29: 208-218.

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

Dorman, D.C., M.F. Struve, B.A. Wong, M.W. Marshall, E.A. Gross and G.A. Wilson. 2008. Respiratory tract response in male rats following subchronic acrolein inhalation. Inhalation Toxicology 20:205-216.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Feron, V.J., A. Kryusse, H.P. Till and H.R. Immel. 1978. Repeated exposure to acrolein vapour: subacute studies in hamsters, rats and rabbits. Toxicology 9: 47-57.

Government of Canada. 2000. Canadian Environmental Protection Act Priority Substances List Assessment Report. Acrolein. ISBN 0-662-282575-1 Cat. No. En40-215/48E

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

Kimbell, J.S., J.H. Overton, R.P. Subramaniam, P.M. Schlosser, K.T. Morgan, R.B. Conolly and F.J. Miller. 2001. Dosimetry modeling of inhaled formaldehyde: binning nasal flux predictions for quantitative risk assessment. Toxicological Sciences 64:111-121.

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. TSD for Noncancer RELs. Appendix D. Individual acute, 8-hour, and chronic reference exposure levels. *Acrolein (page 42)*. 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

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2014a. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

TCEQ (Texas Commission on Environmental Quality). 2014b. Acrolein. Development Support Document. CAS Registry Number 107-02-8. March 14, 2014.

US EPA (United States Environmental Protection Agency). 2003. Toxicological Review of Acrolein (CAS No. 107-02-8). May 2003. Available at: http://www.epa.gov/ncea/iris/toxreviews/0364tr.pdf

US EPA (United States Environmental Protection Agency). 2014. 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

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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C5.0 ALIPHATIC C_5-C_8 GROUP

C5.1 Acute Inhalation Exposure Limits

Table 5-1
Acute Inhalation Exposure Limits for Aliphatic C_5-C_8 Group

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	AAQO	21,000 (n-hexane)	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Standard	2,500 (n-hexane mixture) 6,100 (cyclohexane) 11,000 (heptane) 50,000 (1-octene)	OMOE 2012, 2005a OMOE 2012, 2005b OMOE 2012 OMOE 2012
TCEQ	1-hour ReV	200,000 (n-pentane)	TCEQ 2013, 2011
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The aliphatic C_5-C_8 group was created for comparison against limits recommended by the CCME (2008), MA DEP (2003), RIVM (2001) and TPHCWG (1997) for the petroleum hydrocarbon (PHC) fractions. These agencies, however, have only developed chronic limits, and not acute limits, for the PHC fractions. As a result, the search for acute limits was necessarily expanded to include limits for the individual constituents of the aliphatic C_5-C_8 group.

The TCEQ (2013, 2011) has derived a 1-hour ReV of 200,000 µg/m³ for n-pentane. In the key study by Lammers *et al.* (2011), two acute experiments were conducted. In the first experiment, male WAG/RijCHBR rats (8 per group) were exposed to 0, 2,000, 6,500, or 20,000 mg/m^3 of n-pentane for 8 hours per day for 3 consecutive days. An assessment of motor activity and neurobehavioral functions was conducted using a standardized functional observational battery of tests. No significant adverse neurological effects were observed in any of the exposure groups.

In the second experiment, male WAG/RijCHBR rats (8 per group) were exposed to the same concentrations of n-pentane for the same amount of time, with tests for cognitive performance being conducted after exposure. Mild, reversible changes in performance speed were observed in the two lowest exposure groups, but not in the high-exposure group. Tests conducted one day post-exposure revealed no adverse effects due to n-pentane exposure. The TCEQ (2013) identified 20,000 mg/m^3 (19,872 mg/m^3 average measured concentration) as a free-standing NOAEL. The recommended default RGDR of one (TCEQ 2006) was applied to account for the ratio of the blood: gas coefficients of rats to humans being less than one, resulting in a POD of 19,872 mg/m^3 (equivalent to the NOAEL). An uncertainty factor of 100 was applied to the POD to account for interspecies differences (3, due to the use of an RGDR), intraspecies differences (10), and database deficiencies (3). The resulting 1-hour ReV of 200,000 µg/m³ was used to assess the aliphatic C_5-C_8 group in the acute inhalation assessment.

Although the TCEQ (2013) provides supporting documents for hexane and pentene isomers, no acute ReVs have been derived for these chemicals due to a lack of sufficient information (TCEQ 2007a,b).

ESRD (2013) presents a 1-hour AAQO for n-hexane of 21,000 µg/m³, and indicates that this value is based on a 24-hour California air quality objective. However, a search of the OEHHA (2014) did not reveal a 24-hour value for n-hexane, only a chronic value. As a result, this value was not considered further.

The OMOE (2012, 2005a) developed a 24-hour standard of 2,500 µg/m³ for an n-hexane mixture. This standard was developed from a LOAEL of 58 ppm (204 mg/m³) for polyneuropathy in humans (Sanagi *et al.* 1980). Workers were exposed to low concentrations of n-hexane and acetone in a tungsten carbide alloys facility for an average of 6.2 years. This value is based on chronic exposures that are not relevant to acute assessment. As such, this value was not considered suitable as an acute exposure limit.

In addition, the OMOE (2012, 2005b) has established a 24-hour standard of 6,100 µg/m³ for cyclohexane based on a NOAEL of 6,886 mg/m³ for reduced pup weights in the F1 and F2 generations in a reproductive and developmental inhalation study (Kreckmann *et al.* 2000; OMOE 2005b). The NOAEL was revised to an HEC of 1,722 mg/m³ and the lower confidence of the benchmark concentration (BMCL) was then derived (1,822 mg/m³). An uncertainty factor of 300 was applied to the BMCL to account for intraspecies variability (10), interspecies variability (3), and database deficiencies due to the lack of chronic studies specifically examining developmental neurotoxicity and hepatic effects (10) (OMOE 2005b). Due to the long-term study duration and the uncertainty factor applied for subchronic exposure, this value was not selected for use.

C5.2 Chronic Inhalation Limits

Table 5-2
Chronic Inhalation Exposure Limits for Aliphatic C_5-C_8 Group

Regulatory Agency	Type	Value (µg/m³)	Reference
BC MOE	–	–	BC MOE 2013
CCME	RfC	18,400	CCME 2008
MA DEP	RfC	200	MA DEP 2003
RIVM	TCA	18,400	RIVM 2001
TPHCWG	RfC	18,400	TPHCWG 1997

The CCME (2008) and RIVM (2001) both provide an RfC of 18,400 µg/m³ for the C_5-C_8 aliphatic group based on the neurotoxic endpoint of commercial hexane. This exposure limit was adopted from the TPHCWG (1997), and was developed from the NOAEL of 10,307 mg/m³ for two (rat and mice) chronic bioassays involving lifetime exposure. The NOAEL was adjusted for continuous exposure (6/24 hours × 5/7 days) to a concentration of 1,840 mg/m³. The TPHCWG (1997) applied an uncertainty factor of 100 to account for interspecies variability (10) and intraspecies variability (10). The TPHCWG (1997) recommends using the RfC derived for commercial hexane over an RfC specific to n-hexane (as is the case of the MA DEP RfC) as it is more representative of the aliphatic fraction. According to the TPHCWG (1997), using n-hexane alone results in an overestimation of the toxicity of the fraction because n-hexane is the most

toxic of the group's constituents, it is uniquely toxic and its interaction with other petroleum compounds influences its toxicity. The RfC of 18,400 µg/m³ for commercial hexane was used to evaluate the risks associated with the aliphatic C_5-C_8 group.

The MA DEP (2003) RfC of 200 µg/m³ was derived from toxicity data specific to n-hexane, which is considered overly conservative when characterizing the toxicity of the aliphatic C_5-C_8 group as a whole. As n-hexane has unique neurotoxic characteristics, it is not representative of the toxicity of the aliphatic C_5-C_8 group. As such, the MA DEP value was not selected.

C5.3 Oral Exposure Limits

The aliphatic C_5-C_8 group was not included in the multiple pathway exposure assessment. Thus, a chronic oral exposure limit was not required for the aliphatic C_5-C_8 group.

C5.4 Summary of Exposure Limits

Table 5-3
Summary of Exposure Limits Selected for Aliphatic C_5-C_8 Group

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	ReV	200,000	µg/m³	TCEQ 2013, 2011	–
Chronic	Annual	Inhalation	RfC	18,400	µg/m³	CCME 2008, RIVM 2001, TPHCWG 1997	Neurological effects
		Multiple Exposure Pathway	n/a	n/a	n/a	n/a	n/a

– = Not available
n/a = Not applicable

C5.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

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

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued February 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Kreckmann, K.H., J.K. Baldwin, L.G. Roberts, R.J. Staab, D.P. Kelly and J.E. Saik. 2000. Inhalation developmental toxicity and reproduction studies with cyclohexane. Drug Chem. Toxicol. 23(4):555-573.

Lammers, J.H.C.M., H. Muijser, D.E. Owen, B.M. Kulig and R.H. McKee. 2011. Neurobehavioral effects of acute exposure to normal (n-) paraffins. International Journal of Toxicology 30(1):47-58.

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.

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2005a. Ontario Air Standards for n-Hexane. Standards Development Branch. Ontario Ministry of the Environment. June 2005.

OMOE (Ontario Ministry of the Environment). 2005b. Ontario Air Standards for Cyclohexane. Standards Development Branch. Ontario Ministry of the Environment. June 2005.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

Sanagi, S., Y. Seki, K. Sugimoto and M. Hirata. 1980. Peripheral nervous system functions of workers exposed to n-hexane at a low level. International Archives of Occupational and Environmental Health 47: 69-79.

TCEQ (Texas Commission on Environmental Quality). 2006. Guidelines to develop effects screening levels, reference values, and unit risk factors. Chief Engineer's Office. RG-442. Available from: http://www.tceq.state.tx.us/implementation/tox/esl/guidelines/about.html

TCEQ (Texas Commission on Environmental Quality). 2007a. n-Hexane (CAS Registry Numbers: 100-54-3). Developmental Support Document, Final, October 15, 2007. Prepared by: Valerie Meyers, Ph.D., Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2007b. Pentenes (CAS Registry Numbers: 1-pentene: 109-67-1, c-2-pentene: 627-20-3, t-2-pentene: 647-04-8). Developmental Support Document, Final, October 15, 2007. Prepared by: Bernard J. Kadlubar, M.S., Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2011. Pentane, All Isomers (CAS Registry Numbers: n-Pentane: 109-66-0, Isopentane: 78-78-4, Neopentane: 463-82-1. Developmental Support Document, Final, July 29, 2011. Prepared by: Jong-Song Lee, Ph.D. Toxicology Division, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

TPHCWG (Total Petroleum Hydrocarbon Criteria Working Group). 1997. Vol. 4. Development of Fraction Specific Reference Doses (RfDs) and Reference Specific Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH). Amherst Scientific Publishers. Amherst, Massachusetts.

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C6.0 AROMATIC C_9-C_{16} GROUP

C6.1 Acute Inhalation Exposure Limits

Table 6-1
Acute Inhalation Exposure Limits for Aromatic C_9-C_{16} Group

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Guideline	220 (trimethylbenzene) 400 (isopropylbenzene)	OMOE 2012 OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The aromatic C_9-C_{16} group was created for comparison against limits recommended by the CCME (2008), MA DEP (2003), RIVM (2001) and TPHCWG (1997) for the petroleum hydrocarbon (PHC) fractions. These agencies, however, have only developed chronic limits, and not acute limits, for the PHC fractions. As a result, the search for acute limits was necessarily expanded to include limits for the individual constituents of the aromatic C_9-C_{16} group.

The OMOE (2012) presents 24-hour values of 220 µg/m³ for trimethylbenzenes and 400 µg/m³ for isopropylbenzene; however, as no supporting documentation is available, these values were not considered in the acute assessment.

As no acute exposure limits with adequate supporting documentation were identified from the above listed sources for the individual constituents of the aromatic C_9-C_{16} group, the search for was further expanded to include STEL and Ceiling values from the ACGIH (2013) and AEGL-1 values from the US EPA (2013a).

The ACGIH (2013, 1992) has derived a STEL for naphthalene based on eye irritation (please refer to the toxicity profile for naphthalene for additional details). The 15-minute STEL was adjusted to an equivalent 1-hour concentration of 2,000 µg/m³ and used as a 1-hour exposure limit in the acute assessment of the aromatic C_9-C_{16} group, as it represents the most conservative limit with available supporting rationale.

The US EPA (2007a) has derived a 1-hour AEGL-1 of 250 mg/m^3 for isopropylbenzene (cumene). This value is based on what appears to be an anecdotal report from an occupational environment (Dow 1948) that was published but has since been withdrawn, according to the US EPA (2007a) reference list. This value was not considered in the acute assessment as the supporting information could not be verified.

The US EPA (2007b) has derived a 1-hour AEGL-1 for all isomers of trimethylbenzene of 140 ppm (690,000 µg/m³). Due to a lack of available human data for acute trimethylbenzene exposure, the AEGL-1 was derived from an analysis of several animal studies. Korsak and Rydzynski (1996) conducted a study involving acute (4-hour) exposure to 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene and 1,2,3-trimethylbenzene at concentrations ranging from 250 to 2,000 ppm (individual doses not specified) within a controlled chamber. Concentration-related changes were observed in rotarod performance in the exposed rats (male only). EC_{50} values for each isomer based on disturbances in rotarod function were determined to be: 4,693 mg/m³ (95% CI 3,891 to 5,493 mg/m³) for 1,2,4-trimethylbenzene; 4,738 mg/m³ (95% CI 3,675 to 5,453 mg/m³) for 1,3,5-trimethylbenzene; and 3,779 mg/m³ (95% CI 2,832 to 4,615 mg/m³) for 1,2,3-trimethylbenzene. Changes in pain sensitivity also were observed for the three isomers in the acute study. EC_{50} values for pain sensitivity (demonstrated by the paw lick response) were determined to be the following: 5,682 mg/m³ (95% CI 2,715 to 7,596 mg/m³) for 1,2,4-trimethylbenzene; 5,938 mg/m³ (95% CI of 5,194 to 6,512 mg/m³) for 1,3,5-trimethylbenzene; and 4,155 mg/m³ (3,400 to 4,811 mg/m³ for 1,2,3-trimethylbenzene. Of the two endpoints, rotarod disturbance seems to be the more sensitive effect. Korzack and Rydzynski (1996) note that the 1,2,3-trimethylbenzene isomer appeared to demonstrate more neurotoxic potential than the other two isomers.

Also cited as a key study by US EPA (2007c), Korsak *et al.* (1995) conducted a similar study with only 1,2,4-trimethylbenzene in male rats. Rats were exposed for a duration of 4 hours to 250 to 2,000 ppm (individual dose levels not specified) within a controlled chamber. Altered rotorod activity indicative of neurotoxicity, altered pain response and decreased respiratory rate were observed in association with concentration-dependent responses. EC_{50} values for rotorod performance, pain sensitivity and respiratory depression were determined to be 4,693 mg/m³ (95% CI 3,891 to 5,493 mg/m³), 5,682 mg/m³ (95% CI 2,715 to 7,596 mg/m³) and 2,840 mg/m³ (95%,CI 1,500 to 3,900 mg/m³), respectively. Although it is not clear how the US EPA calculated the value, an average of 900 ppm was calculated to be the average EC_{50} for neurological effects from the animal data, and served as the point of departure for the derivation of the AEGL. The Haber's Law approach was used by the US EPA (2007c) to convert the 4-hour concentration to a 1-hour concentration of 1,429 mg/m³. A total uncertainty factor of 10 was applied to account for interspecies differences (3), and intraspecies differences (3), to result in the 1-hour AEGL of 690 mg/m³ (690,000 µg/m³). This value was not selected for use in the assessment, as it is much higher than the adjusted STEL for naphthalene.

C6.2 Chronic Inhalation Exposure Limits

Table 6-2
Chronic Inhalation Exposure Limits for Aromatic C_9-C_{16} Group

Regulatory Agency	Type	Value (µg/m³)	Reference
BC MOE	–	–	BC MOE 2013
CCME	RfC	200	CCME 2008
MA DEP	RfC	50	MA DEP 2003
RIVM	TCA	200	RIVM 2001
TPHCWG	RfC	200	TPHCWG 1997

– = Not available

The MA DEP (2003) has developed an RfC of 50 µg/m³ based on a study by Clark *et al.* (1989). The chronic RfC is based on increased liver and kidney weights in male rats exposed to high flash aromatic naphtha, which is primarily composed of 9-carbon aromatic compounds. Rats were administered 0, 450, 900 or 1,800 mg/m³ of a mixture of C_9 aromatics for 6 hours/day, 5 days/week for 12 months (Clark *et al.* 1989). A NOAEL of 900 mg/m³ was identified for liver and kidney effects and converted to continuous exposure (6/24 hours × 5/7 days) resulting in a NOAEL of 160 mg/m³. After applying an uncertainty factor of 1,000 to account for the interspecies variability (10), intraspecies variability (10) and use of a subchronic study (10), the MA DEP (2003) also applied an additional uncertainty factor of 3 to account for database deficiencies, which are detailed within MA DEP (2003). This partial uncertainty factor was applied to account for the lack of toxicity information on non-PAH compounds in the C_9-C_{16} aromatic fraction range (MA DEP 2003). The resulting value of 50 µg/m³ based on liver and kidney effects was selected for use in the chronic effects assessment of the aromatic C_9-C_{16} group.

The CCME (2008) has adopted its chronic RfC for C_9-C_{16} aromatics of 200 µg/m³ from the TPHCWG (1997). The TPHCWG limit also was based on the 1989 study by Clark *et al.* The TPHCWG (1997) applied an uncertainty factor of 1,000 to the duration-adjusted NOAEL of 160 mg/m³ to account for the interspecies variability (10), intraspecies variability (10) and use of a subchronic study (10). The CCME/TPHCWG RfC of 200 µg/m³ was not used in the chronic inhalation effects assessment, as the MA DEP (2003) RfC represents a more conservative limit.

The RIVM (2001) TCA has been adopted from the TPHCWG (1997), and also was rejected in favour of the more conservative value from MA DEP.

C6.3 Oral Exposure Limits

The aromatic C_9-C_{16} group was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, the aromatic C_9-C_{16} group Name was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C6.4 Summary of Exposure Limits

Table 6-3
Summary of Exposure Limits Selected for Aromatic C_9-C_{16} Group

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	STEL	2,000	µg/m³	ACGIH 2013, 1992	Eye irritation
Chronic	Annual	Inhalation	RfC	50	µg/m³	MA DEP 2003	Liver and kidney effects
		Multiple Exposure Pathway	RfD	n/a	n/a	–	–

– = Not available
n/a = Not applicable

C6.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

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

Clark, D.G., S.T. Butterworth, J.G. Martin, H.R. Roderick and M.G. Bird. 1989. Inhalation toxicity of high flash aromatic naphtha. Toxicol Ind. Health 5(3):415-428.

Dow (Dow Chemical Company). 1948. Toxicology and hygiene – isopropylbenzene. July 6, 1948. Study authors were E.M. Adams and V.K. Rowe. Previously released to NTIS/OTS #0206685, but since withdrawn. Provided 9/2004 courtesy of S. Ripple, Dow Corporation. Cited in: US EPA 2007a.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Korsak, Z. and K. Rydzynski. 1996. Neurotoxic effects of acute and subchronic inhalation exposure to trimethylbenzene isomers (pseudocumene, mesitylene, hemimellitene) in rats. Int J Occup Med Environ Health 9(4): 341-349.

Korsak, Z., S. Radoslaw and K. Rydzynski. 1995. Toxic effects of acute inhalation exposure to 1,2,4-trimethylbenzene (pseudocumene) in experimental animals. Int J Occup Med Environ Health 8(4):331-337

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.

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

TPHCWG (Total Petroleum Hydrocarbon Criteria Working Group). 1997. Vol.4. Development of Fraction Specific Reference Doses (RfDs) and Reference Specific Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH). Amherst Scientific Publishers. Amherst, Massachusetts.

US EPA (United States Environmental Protection Agency). 1998. Toxicological Review of Naphthalene (CAS No. 91-20-3). August 1998. Available at: http://www.epa.gov/ncea/iris/toxreviews/0436tr.pdf

US EPA (United States Environmental Protection Agency). 2007a. Interim. Acute Exposure Guideline Levels (AEGLs) for Cumene (CAS Reg. No. 98-82-8). Available at: http://www.epa.gov/opptintr/aegl/pubs/cumene_tsd_interimversion1_8_28_07.pdf

US EPA (United States Environmental Protection Agency). 2007b. Interim. Acute Exposure Guideline Levels (AEGLs) for 1,3,5-Trimethylbenzene (CAS Reg. No. 108-67-8), 1,2,4-Trimethylbenzene (CAS Reg. No. 95-63-6), 1,2,3-Trimethylbenzene (CAS Reg. No. 526-73-8). November 2007. Available at: http://www.epa.gov/opptintr/aegl/pubs/123_124_135_trimethylbenzenes_interim_11_2007.v1.pdf

US EPA (United States Environmental Protection Agency). 2007c. Provisional Peer-Reviewed Toxicity Values for 1,2,4-trimethylbenzene. Superfund Health Risk Assessment Technical Center, National Center for Environmental Assessment. CASRN 95-63-6.

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C7.0 AROMATIC C_{17}-C_{34} GROUP

C7.1 Acute Inhalation Exposure Limits

Table 7-1
Acute Inhalation Exposure Limits for the Aromatic C_{17}-C_{34} Group

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	–	–	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The aromatic C_{17}-C_{34} group was created for comparison against limits recommended by the CCME (2008), MA DEP (2003), RIVM (2001) and TPHCWG (1997) for the petroleum hydrocarbon (PHC) fractions. These agencies, however, have only developed chronic limits, and not acute limits, for the PHC fractions. As a result, the search for acute limits was necessarily expanded to include limits for the individual constituents of the aromatic C_{17}-C_{34} group.

Acute exposure limits for the constituents of the aromatic C_{17}-C_{34} group were not available from any of the regulatory agencies listed above, therefore the search was expanded to include short-term occupational limit values (*i.e.*, STEL and Ceiling) developed by the ACGIH (2013), as well as AEGLs-1 (2013b) developed by the US EPA. As acute limits were not available from these additional sources, the aromatic C_{17}-C_{34} group was not evaluated on an acute basis.

C7.2 Chronic Inhalation Exposure Limits

Table 7-2
Chronic Inhalation Exposure Limits for the Aromatic C_{17}-C_{34} Group

Regulatory Agency	Type	Value (µg/m³)	Reference
BC MOE	–	–	BC MOE 2013
CCME	–	–	CCME 2008
MA DEP	–	–	MA DEP 2003
RIVM	–	–	RIVM 2001
TPHCWG	–	–	TPHCWG 1997

– = Not available

The regulatory agencies typically searched for exposure limits for the aromatic C_{17}-C_{34} group did not provide any values, nor did the search for the individual constituents of the aromatic C_{17}-C_{34}

group reveal any exposure limits. As a result, the aromatic C_{17}-C_{34} group was not evaluated in the chronic inhalation assessment.

C7.3 Oral Exposure Limits

Table 7-3
Chronic Oral Exposure Limits for the Aromatic C_{17}-C_{34} Group

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
CCME	RfD	30	CCME 2008
MA DEP	RfD	30	MA DEP 2003
RIVM	TDI	30	RIVM 2001
TPHCWG	RfD	30	TPHCWG 1997

The TPHCWG (1997) has derived an oral RfD of 30 µg/kg bw/d for the aromatic C_{17}-C_{34} fraction based on the US EPA's oral RfD for pyrene (US EPA 1993). The US EPA RfD for pyrene was derived from a NOAEL of 75 mg/kg bw/d reported in a chronic oral mouse study, in which male and female CD-1 mice (20/sex/group) were gavaged with 0, 75, 125, or 250 mg/kg/day pyrene in corn oil for 13 weeks (US EPA 1989). Kidney effects (changes in renal tubular pathology and reduced kidney weights) in the two highest dose groups determined the NOAEL of 75 mg/kg bw/d. An uncertainty factor of 1,000 was applied to the NOAEL to account for interspecies variability (10), intraspecies variability (10) and use of a subchronic study (10). A modifying factor of 3 was also applied due to the lack of adequate toxicity data. The resulting RfD of 30 µg/kg bw/d was used in the chronic multiple exposure pathway assessment of the aromatic C_{17}-C_{34} group.

The CCME (2008) and RIVM (2001) adopted the TPHCWG's value of 30 µg/kg bw/d as a chronic oral exposure limit for the aromatic C_{17}-C_{34} group.

The MA DEP (2003) also recommends an oral RfD of 30 µg/kg bw/d for the aromatic fraction of C_9-C_{32}. Their RfD is based on the US EPA (1993) RfD of 0.03 mg/kg bw/d for pyrene as well (described above).

C7.4 Summary of Exposure Limits

Table 7-4
Summary of Exposure Limits Selected for the Aromatic C_{17}-C_{34} Group

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	–	–	–	–	–
Chronic	Annual	Inhalation	–	–	–	–	–
		Multiple Exposure Pathway	RfD	30	µg/kg bw/d	CCME 2008, MA DEP 2003, RIVM 2001, TPHCWG 1997	Kidney effects

– = Not available

C7.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

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

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

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.

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

TPHCWG (Total Petroleum Hydrocarbon Criteria Working Group). 1997. Vol.4. Development of Fraction Specific Reference Doses (RfDs) and Reference Specific Concentrations (RfCs) for Total Petroleum Hydrocarbons (TPH). Amherst Scientific Publishers. Amherst, Massachusetts.

US EPA (United States Environmental Protection Agency). 1989. Mouse Oral Subchronic Toxicity of Pyrene. Study conducted by Toxicity Research Laboratories, Muskegon, MI for the Office of Solid Waste, Washington, DC. Cited in US EPA 1993

US EPA (United States Environmental Protection Agency). 1993. IRIS Summary for Pyrene (129-00-0). Reference Dose for Chronic Oral Exposure (RfD). Available at: http://www.epa.gov/ncea/iris/subst/0445.htm#reforal

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C8.0 BENZENE

C8.1 Acute Inhalation Exposure Limits

Table 8-1
Acute Inhalation Exposure Limits for Benzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	24-hour MRL	30	ATSDR 2013, 2007
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO	30	ESRD 2013
OEHHA	6-hour REL	1,300	OEHHA 2014, 2008
OMOE	24-hour	2.3	OMOE 2012
TCEQ	1-hour ReV	580	TCEQ 2013, 2007
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The TCEQ (2013, 2007) has derived an acute ReV of 580 µg/m³ for benzene. Review of the supporting documentation for this value indicates that TCEQ used the same key study (Rozen *et al.* 1984) as the ATSDR. As well, the TCEQ identified the same LOAEL value of 10.2 ppm. The difference between the ATSDR and TCEQ values originates from the adjustment of the LOAEL for continuous exposure and the uncertainty factors applied.

The TCEQ (2007) established that the $LOAEL_{ADJ}$ for benzene in the Rozen *et al.* (1984) study was 18.5 ppm (59 mg/m³), using Haber's law and a default approach for converting exposures of more than one hour to a 1-hour exposure level from TCEQ (2007). The $LOAEL_{ADJ}$ was converted to a $LOAEL_{HEC}$ using a regional gas dose ratio (RGDR). In the case that the animal blood to gas partition coefficient is greater than the human blood to gas partition coefficient, a default value of 1 is used for the RGDR. Thus, the $LOAEL_{HEC}$ was calculated to be 18.5 ppm. A cumulative uncertainty factor of 100 was applied by the TCEQ (2007) to the $LOAEL_{HEC}$ to account for interspecies differences (3), intraspecies variability (10), and the use of a LOAEL (3). A factor of 3 was applied for extrapolation of animal data to humans since dosimetric adjustments were conducted to address toxicokinetic differences. In addition, studies indicate that benzene is metabolized along similar pathways in animals and humans and data suggests that mice are relatively sensitive in regards to hematotoxic effects of benzene (TCEQ 2007). A factor of 3 was applied for extrapolation from a LOAEL to a NOAEL on the basis that the LOAEL used to derive the acute ReV is lower than other LOAELs observed in animal and human studies, and the LOAEL is similar to NOAELs observed in mouse studies (TCEQ 2007). In addition, benchmark dose modelling of estimated lymphocyte count depression data produces a BMCL of 4 ppm, which supports a factor of 3 as being sufficiently conservative (TCEQ 2007). The TCEQ (2007) also states that lymphocyte count depression is a sensitive sentinel effect that is not a serious nature, and the reported decreased lymphocyte count at 10.2 ppm appears to be within the normal range. The result is an acute ReV of 580 µg/m³ based on immunological effects, which was used as a 1-hour limit in the acute effects assessment of benzene.

The ATSDR (2013, 2007) presents an acute MRL of 0.009 ppm (30 µg/m³) based on immunological effects. Male C57BL/6J mice (7 or 8 per concentration) were exposed to 0, 10.2,

31, 100, or 301 ppm (0, 32.6, 99, 320, or 960 mg/m³) benzene in whole-body dynamic inhalation chambers for 6 hours/day on six consecutive days (ATSDR 2007). The control group was exposed to filtered, conditioned air only. Significant depression of femoral lipopolysaccharide induced B-colony-forming ability was observed at the 10.2 ppm exposure level in the absence of a significant depression of total number of B cells. Peripheral lymphocyte counts were depressed at all exposure levels. The ATSDR (2007) adjusted a LOAEL of 10.2 ppm (32.6 mg/m³) from intermittent to continuous exposure (6/24 hours) to a concentration of 2.55 ppm (8.16 mg/m³). The duration-adjusted LOAEL ($LOAEL_{ADJ}$) was converted to a HEC ($LOAEL_{HEC}$) for a category 3 gas causing respiratory effects. The average ratio of the animal blood: air partition coefficient would be greater than 1; thus, a default value of 1 was used in calculating the HEC (ATSDR 2007). As a result, a $LOAEL_{HEC}$ of 2.55 ppm (8.16 mg/m³) was identified. The ATSDR (2007) applied a cumulative uncertainty factor of 300 to the $LOAEL_{HEC}$ to account for interspecies variability (3), intraspecies variability (10) and use of a LOAEL (10). A factor of 3 was applied for the extrapolation of laboratory animal data to humans since the calculation of a HEC addressed the pharmacokinetic aspects of the interspecies uncertainty factor. This value was not selected, as the time-adjustment process applied by TCEQ (2007) was more defensible given the dose-response and duration-related effects observed for benzene.

ESRD (2013) also provides a 1-hour AAQO of 30 µg/m³ for benzene based on hematological effects. However, detailed supporting documentation is lacking. As a result, it was not used in the acute effects assessment.

The OMOE (2012) presents a 24-hour criterion of 2.3 µg/m³, however, no supporting documentation for this value is available. As a result, this value was not selected for use in the assessment.

The OEHHA (2014, 2008) presents a 6-hour acute REL of 1,300 µg/m³, based on reproductive effects. The key study (Coate *et al.* 1984) involved the exposure of pregnant female rats (40 per group) to 0, 1, 10, 40 or 100 ppm (0, 3.2, 32, 130 or 324 mg/m³) for 6 hours/day on days 6 to 15 of gestation. Significantly decreased mean fetal weights were observed at the highest (100 ppm) exposure level. No fetotoxic, teratogenic or maternal toxicity was observed in the 40 ppm group. The study NOAEL was identified as 40 ppm for reduced fetal weight. An uncertainty factor of 100 was applied to account for interspecies differences (10) and intraspecies variability (10). The OEHHA (2008) notes that the NOAEL was not adjusted to a 1-hour exposure due to the uncertainty associated with extrapolating data from repeated exposures to a 1-hour concentration. As a result of this uncertainty, the 6-hour REL of 1,300 µg/m³ may be considered equivalent to a 1-hour REL. This value was not selected, as reproductive effects do not appear to be the most sensitive endpoint in association with acute benzene exposure.

C8.2 Chronic Inhalation Exposure Limits

Table 8-2
Chronic Inhalation Exposure Limits for Benzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	9.8	ATSDR 2013, 2007
BC MOE	–	–	BC MOE 2013
BC MOE[(1)]	–	1.5	BC MOE 2014
ESRD	RfC	30	ESRD 2013
Health Canada	RsC	3	Health Canada 2010
OEHHA	RsC	0.3	OEHHA 2009
	REL	60	OEHHA 2014, 2000
RIVM	CR	2	RIVM 2001
TCEQ	ReV	280	TCEQ 2013, 2007
	Linear ESL	4.5	
US EPA	RfC	30	US EPA 2014, 2003
	RsC	1.3 to 4.5	US EPA 2014, 2000
WHO	RsC	1.7	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

The US EPA (2014, 2000) presents a range of potential carcinogenic risks from inhalation of benzene. The key data sets employed in the US EPA cancer assessment were those by Rinsky *et al.* (1987, 1981), which were also critically analyzed by Paustenbach *et al.* (1993), Crump and Allen (1984), Crump (1994, 1992), and US EPA (1998). The Rinsky *et al.* (1987, 1981) studies examined the incidence of leukemia in exposed white male workers in the rubber hydrochloride department of a pliofilm plant. The more comprehensive follow up study (Rinsky *et al.* 1987) involved the evaluation of 1,165 workers who were exposed for at least 1 day between 1965 and 1981. Individual assessments of cumulative exposure were calculated by Rinsky *et al.* for each worker based on air sampling data. Inhalation unit risks of 2.2×10^{-6} to 7.8×10^{-6} per µg/m³ were extrapolated based on a low-dose linear model using maximum likelihood estimates for leukemia in humans (US EPA 2000). The inhalation unit risks equate to an RsC of 1.3 to 4.5 µg/m³ associated with a risk level of one in 100,000 (US EPA 2000). The RsC of 1.3 µg/m³ based on leukemia incidence was selected as the chronic inhalation limit for benzene in the carcinogenic assessment as it is the more conservative of the RsC values presented within this range.

The OEHHA (2009) derived a unit risk estimate of 2.9E-05 $(\mu g/m^3)^{-1}$ (equivalent to an RsC of 0.34 µg/m³) based on epidemiological studies of Chinese workers. Although it is not very clear, the basis of the OEHHA value seems to be the studies by Yin *et al.* (1996, 1994). The Chinese cohort studies that served as the basis of the OEHHA derivation were some of the studies determined by the US EPA to have methodological issues (poor exposure characterization, co-exposure to other agents, data quality) to the point where the study was not adequate for quantitative assessment. The US EPA RsC value, in contrast, is based on a study that has been critically analyzed by several other studies. As such, the OEHHA value was not used in the chronic inhalation assessment of benzene.

An RsC of 3 µg/m³ is reported by Health Canada (2010) based on an inhalation unit risk of 0.0033 per mg/m³. This value was derived from data in the Rinsky *et al.* (1987) study discussed above in the US EPA summary, and was calculated through the identification of a dose associated with a 5% increase in mortality from acute myelogenous leukemia. However, this RsC was not selected as the US EPA value is more conservative.

The WHO (2000) provides an RsC of 1.7 µg/m³, which is associated with an increased cancer risk of one in 100,000. Using multiplicative risk estimates and a cumulative exposure model, a unit risk for lifetime exposure of 1.4 to 1.5 × 10^{-5} per ppb was derived with the Paustenbach exposure matrix and 2.4 × 10^{-5} per ppb with the Crump and Allen exposure matrix (WHO 2000). These values equate to unit risks that range from 4.4 × 10^{-6} per µg/m³ to 7.5 × 10^{-6} per µg/m³. From these datasets, the WHO (2000) selected a representative unit risk of 6 × 10^{-6} per µg/m³. The WHO (2000) value was not chosen for the chronic inhalation assessment as the US EPA RsC value was slightly more conservative.

The TCEQ (2013, 2007) also provides a linear Effects Screening Level (ESL) value, using cancer potency estimates based on Crump and Allen (1984) to calculate the URF and ESL. Crump and Allen investigated the risk of leukemia from occupational exposure to benzene in Plioform workers, and determined that acute myelogenous and monocytic leukemia (AMML) was the only cancer response clearly related to benzene exposure. A linear multiplicative risk model fit the Plioform cohort data best, and cancer potency estimates for both cumulative and weighted cumulative exposure metrics were used. The 95th percentile upper confidence limits (UCLs) on the estimates were calculated, and then the occupational concentrations were converted to environmental concentrations. The best fitting linear model for AMML was based on cumulative exposure as the exposure metric, and the air concentration corresponding to an excess cancer risk of 1 in 100,000 was 2.3 ppb (95% UCL = 1.4 ppb, or 4.5 µg/m³). This value was not selected, as it is not based on as robust an analysis as the US EPA value.

The RIVM (2001) provides a CR_{inhal} of 20 µg/m³ for one in 10,000 excess lifetime cancer risk for inhalation exposure. The equivalent CR_{inhal} for one in 100,000 excess lifetime cancer risk is 2 µg/m³. The RIVM has chosen the lower end limit adopted from the EU Working Group (EU 1999) cancer risk estimate range of 20 µg/m³ to 36 µg/m³. As the RIVM (2001) value is not as conservative as the US EPA RsC value, this limit was not selected for use in the assessment.

The ATSDR (2013, 2007) has derived a chronic inhalation MRL of 0.003 ppm (9.8 µg/m³) based on a study by Lan *et al.* 2004. The cross-sectional human study by Lan *et al.* (2004) studied 250 benzene-exposed workers at two shoe manufacturing factories in China. A control group of 140 workers from clothing manufacturing facilities was selected, and matched for age and sex with the exposed worker group. The average age of the study subjects was 29.9 years, and the average duration of exposure in the benzene-exposed group was 6.1 years. Exposure to benzene and toluene was monitored at least 5 times over a 16-month period using individual exposure monitors. Exposed subjects were grouped according to exposure concentrations measured over a 1-month period before the collection of blood samples: <1 ppm (109 subjects), 1 to <10 ppm (110 subjects), and ⩾10 ppm (31 subjects). Blood samples were analyzed for a number of parameters, including: WBC, granulocytes, lymphocytes, CD4+ and CD8+ T-cell counts, B cells, natural killer cells, platelets and hemoglobin. Details regarding potential susceptibility to the effects of benzene were examined through genotyping, with attention being given to four single-nucleotide polymorphisms that are known to impact benzene metabolism. Significant decreases in all WBC parameters were observed in subject exposed to less than 1 ppm. Significant decreases in hemoglobin were observed only in the high benzene exposure group (> 10 ppm). Linear trend analyses were also conducted by Lan *et al.*, and significant

effects on platelets and all WBC (with the exception of monocyte and CD8+ counts) were observed at all exposure levels. Further analysis was completed taking into account potential confounding factors and past exposures, exposure concentrations and a significant relationship remained between benzene exposure (< 1 ppm and above) on platelets and certain WBC parameters. Lan *et al.* 2004 concluded that benzene exposures of less than 1 ppm are associated with adverse effects on progenitor cell colony formation, and suggest that early progenitor cells are more sensitive than mature cells to the effects of benzene. Individuals with two of the four examined potentially sensitive genotypes were found to have a significant increase in the relative severity of the WBC reductions compared to the other genotype groups. Benchmark dose modelling was completed, and a $BMDL_{0.25sd}$ of 0.1 ppm (320 µg/m³) was calculated. The ATSDR adjusted the 8-hour exposure to a continuous exposure (0.1 × 8/24 hours × 6/7 days). An uncertainty factor of 10 was applied to the adjusted $BMDL_{0.25sd}$ of 0.03 ppm (98 µg/m³) to account for human variability. The resulting MRL of 9.8 µg/m³ based on hematological effects was used as the chronic non-cancer inhalation limit for benzene in the non-carcinogenic assessment, as this value is based upon detailed analysis that takes mechanism of effect and genetic susceptibility into account.

The US EPA (2014, 2003) has derived a non-carcinogenic RfC of 30 µg/m³ based on a cross-sectional occupational study where decreased lymphocyte counts were observed in exposed workers (Rothman *et al.* 1996). In the key study (Rothman *et al.* 1996), a study population of 44 exposed workers (21 female, 23 male) from adhesive, paint, and rubber factories was selected. These individuals were matched based on age and sex with 44 unexposed subjects (21 female, 23 male). Individuals with a history of cancer, radiation or chemotherapy, or who were pregnant were excluded from the study. All subjects in the exposed group had been exposed to benzene for at least 6 months, with no known exposure to other chemicals that could affect bone marrow. Interviews were used to collect lifestyle and employment related information that could influence benzene exposures. The mean number of years that subjects had been exposed was 6.3 years, with a range of 0.7 to 16 years. Passive dosimetry badges were used to collect benzene exposure information over a 1 to 2 week period before blood samples were collected. Blood samples were analyzed for total white blood cell count, absolute lymphocyte count, hematocrit, red blood cell and platelet counts, and mean corpuscular volume. In addition, urine samples were analyzed for the presence of benzene metabolites. The median 8-hour TWA exposure within the exposed population was determined to be 31 ppm (99,000 µg/m³). The exposed group was further divided into two groups – those with exposures greater than the median (with a group median of 92 ppm or 294,000 µg/m³) and those less than the median (with a group median of 13.6 ppm or 43,000 µg/m³). The less-than-median group was further subdivided, and a group of individuals with median exposures of 7.6 ppm (25,000 µg/m³). Urinary concentrations of metabolites were found to have a significant correlation with benzene exposure. In the high benzene exposure group (>31 ppm), all six blood parameters were significantly different relative to blood samples from unexposed subjects. In the lower benzene exposure group (<31 ppm), ALC, RBC and platelets were significantly lower than unexposed subjects. In the lowest exposure group (<7.6 ppm), ALC were significantly lower than controls. The US EPA (2002) concluded that ALC is a sensitive, sentinel effect associated with chronic benzene exposure, and selected it as the toxicological endpoint for the basis of the RfC derivation. Benchmark dose modelling was conducted based on the study data, and an 8-hour TWA BMC of 13.7 ppm and an 8-hour TWA BMCL of 7.2 ppm (23,000 µg/m³). The TWA BMCL was adjusted to a continuous exposure concentration of 8,200 µg/m³ (23,000 µg/m³ × 10/20 m³/day × 5/7 days per week). The US EPA applied an uncertainty factor of 300 to the adjusted BMCL to account for intraspecies differences (10), the use of subchronic data (3), and database deficiencies (3), resulting in an RfC of 30 µg/m³. The US EPA notes that there is some uncertainty surrounding the dose-response relationship for

ALC in the low dose range of the curve. This value was not selected, as the Lan *et al.* (2004) study that is the basis of the ATSDR value examined lower exposure concentrations.

The OEHHA (2014, 2000) also derived a non-cancer based value of 60 µg/m³ based on a study involving exposed refinery workers (Tsai *et al.* 1983). A total of 303 male workers exposed to benzene for a duration ranging from 1 to 21 years (average 7.4 years) served as the study population. Exposures to benzene were measured using personal monitors, and the median benzene exposure concentration was determined to be 0.53 ppm. Blood samples were taken over a period of 20 years. The median exposure concentration of 0.53 ppm was identified by the OEHHA as a free-standing NOAEL concentration for the purposes of the derivation of the REL. This NOAEL was adjusted to a $NOAEL_{HEC}$ of 0.19 ppm through adjustment for continuous exposure (10/20 m³/day × 5/7 days per week). This limit was not used in the assessment due to the existence of the more conservative (lower) ATSDR MRL that is based on a more robust derivation approach (*i.e.* benchmark dose modelling.

The TCEQ (2013, 2007) derived a chronic ReV of 280 µg/m³ based on the immunological effects reported in Rothman *et al.* (1996) (the basis of the US EPA value) and Lan *et al.* (2004) (see the ATSDR summary above). Using the information from these two studies, the TCEQ conducted benchmark dose modelling for each and identified two BMCLs (7.2 ppm and 0.26 ppm, respectively). These values were converted to POD_{HEC} through the adjustment for continuous exposure (10/20 m^3/day × 5 or 6 days per week). The resulting POD_{HEC} values for the Rothman and Lan data sets were 2.6 ppm and 0.11 ppm, respectively. The Rothman POD_{HEC} was selected by the TCEQ on the basis that the adverse effects observed in this study are more supported by the general weight of evidence than the findings of Lan *et al.* 2004. The TCEQ applied an uncertainty factor of 30 to account for human variability (10) and database uncertainties (3). This value was not selected, as it is less conservative that both the ATSDR and US EPA values, despite evidence of potential low-dose effects of benzene.

The ESRD (2013) presents a chronic inhalation value of 3 µg/m³ based on the incidence of hematological effects. A limited supporting information is provided for this value, it was not selected for use in the chronic assessment of benzene.

The BC MOE (2014) has derived a vapour standard of 1.5 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, this value was not selected for use, as supporting documentation was not available.

C8.3 Oral Exposure Limits

Benzene was not incorporated into the multiple pathway exposure assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, benzene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C8.4 Summary of Exposure Limits

Table 8-3
Summary of Exposure Limits Selected for Benzene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	ReV	580	µg/m³	TCEQ 2013, 2007	Immunological effects
Chronic	Annual	Inhalation	RsC	1.3	µg/m³	US EPA 2014, 2000	Leukemia
			RfC	9.8	µg/m³	ATSDR 2013, 2007	Immunological, hematological effects
		Multiple Exposure Pathway	n/a	n/a	n/a	n/a	n/a

n/a = Not applicable

C8.5 References

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

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Coate, W.B., A.M. Hoberman and R.S. Durloo. 1984. Inhalation teratology study of benzene in rats. In: MacFarland, H.N., editor. Advances in modern environmental toxicology, Vol VI. Applied toxicology of petroleum hydrocarbons. Princeton (NJ). Princeton Scientific Publishers, Inc. p. 197-198. Cited in: OEHHA 2008.

Crump, K.S. 1992. Exposure-response analyses of Pliofilm cohort. Unpublished. Cited in: US EPA 2000.

Crump, K.S. 1994. Risk of benzene-induced leukemia: a sensitivity analysis of the Pliofilm cohort with additional follow-up and new exposure estimates. J Toxicol Environ Health 42:219-242.

Crump, K.S. and B.C. Allen. 1984. Quantitative estimates of risk of leukemia from occupational exposure to benzene. Prepared for the Occupational Safety and Health Administration by Science Research Systems, Inc., Ruston, LA. Unpublished. Cited in: US EPA 2000

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

EU (European Union). 1999. Benzene: Risk Assessment – Chapter 2. Commission of European Communities, Council Directive on Ambient Air Quality Assessment and Management, Working Group on Benzene, January 1999. Cited in: RIVM 2001.

Health Canada. 2010. 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. September 2010.

Lan, Q., L. Zhang, G. Li, *et al.* 2004. Hematotoxicity in workers exposed to low levels of benzene. Science 306:1774-1776.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Determination of Noncancer Chronic Reference Exposure Levels. Benzene. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=24

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. Acute Toxicity Summary Benzene. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=18

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Paustenbach, D.J., R.D. Bass and P. Price. 1993. Benzene toxicity and risk assessment, 1972-1992: implications for future regulation. Environ Health Perspect 101 (Suppl 6):177-200.

Rinsky, R.A., A.B. Smith, R. Hornung, T.G. Filloon, *et al.* 1987. Benzene and leukemia: an epidemiologic risk assessment. N Engl J Med 316:1044-1050.

Rinsky, R.A.; R.J. Young and A.B. Smith. 1981. Leukemia in benzene workers. Am J Ind Med 2:217-245.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

Rothman, N., G.-L. Li, M. Dosemeci, W.E. Bechtold, G.E. Marti, Y.-Z. Wang, M. Linet, L.-Q. Xi, W. Lu, M.T. Smith, N. Titenko-Holland, L.-P. Zhang, W. Blot, S.-N. Yin and R.B. Hayes. 1996. Hematotoxocity among Chinese workers heavily exposed to benzene. American Journal of Industrial Medicine 29:236-246.

Rozen, M.G., C.A. Snyder and R.E. Albert. 1984. Depressions in B- and T-lymphocyte mitogen-induced blastogenesis in mice exposed to low concentrations of benzene. Toxicol Lett 20: 343-349.

TCEQ (Texas Commission on Environmental Quality). 2007. Benzene (CAS Registry Number: 71-43-2). Developmental Support Document, Final, October 2007. Prepared by: Haney, Joseph T. Jr. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

Tsai, S.P., C.P. Wen, N.S. Weiss, O. Wong, W.A. McClellan and R.L. Gibson. 1983. Retrospective mortality and medical surveillance studies of workers in benzene areas of refineries. J Occup Med. 25(9):685-692.

US EPA (United States Environmental Protection Agency). 1998, Carcinogenic effects of benzene: an update. Prepared by the National Center for Environmental Health, Office of Research and Development. Washington, DC. EPA/600/P-97/001F.

US EPA (United States Environmental Protection Agency). 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 (United States Environmental Protection Agency). 2003. IRIS Summary of Benzene (CASRN 71-43-2). Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0276.htm#refinhal

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

Yin, S-N, R.B. Hayes, M.S. Linet, G-L. Li, M. Dosemeci, L.B. Travis, *et al.* 1996. A cohort study of cancer among benzene-exposed workers in China: Overall results. Am J Ind Med 29: 227-235.

Yin, S-N., M.S. Linet, R.B. Hayes, G-L. Li, M. Dosemeci, Y-Z. Wang, *et al.* 1994. Cohort study among workers exposed to benzene in China: I. General methods and resources. Am J Ind med 26: 383-400.

C9.0 BENZO(A)PYRENE

C9.1 Acute Inhalation Exposure Limits

Table 9-1
Acute Inhalation Exposure Limits for Benzo(a)pyrene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Guideline	0.0011	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

Benzo(a)pyrene is the only individual carcinogenic polycyclic aromatic hydrocarbon (PAH) with an acute exposure limit established by one of the above agencies. The OMOE (2012) has developed a 24-hour guideline of 0.0011 µg/m³ based on the carcinogenic potential for benzo(a)pyrene. The limit was derived from an annual exposure limit of 0.00022 µg/m³ for protection against carcinogenic effects using a simple extrapolation factor generally considered to be overly conservative. However, this limit was not used in the acute effects assessment for benzo(a)pyrene or the benzo(a)pyrene group because it did not account for the influence of duration of exposure on the carcinogenic action of a chemical.

As acute inhalation exposure limits for benzo(a)pyrene are not provided by any of the other agencies listed above, the search for limits was extended to include intermediate inhalation MRLs from ATSDR, STEL and Ceiling values from the ACGIH (2013) and AEGL-1 values from the US EPA (2013a). No values for benzo(a)pyrene were identified, and therefore neither benzo(a)pyrene nor the benzo(a)pyrene group was assessed on an acute basis.

C9.2 Chronic Inhalation Exposure Limits

Benzo(a)pyrene and any other carcinogenic PAHs identified as chemicals of potential concern were evaluated in the chronic inhalation assessment using two different approaches.

In 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.

For 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 (TEQ) approach). The toxic equivalency of each PAH was determined using Potency Equivalency Factors (PEFs) that were assigned by Equilibrium and URS (2006), and later

adopted by Health Canada (2012). PAHs that did not have evidence of being directly carcinogenic or genotoxic were not assigned PEF values (*e.g.*, anthracene).[1]

The Toxic Equivalency Factors (TEFs) used in the current assessment of PAHs via the TEQ approach are shown in the following table.

Table 9-2
Relative Potency of Individual PAHs Compared with Benzo(a)pyrene

Compound	Toxicity Equivalency Factor [(a)]
Benz(a)anthracene	0.1
Benzo(a)pyrene	1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.1
Chrysene	0.01
Fluoranthene	0.001
Indeno(1,2,3-cd)pyrene	0.1
Phenanthrene	0.001

(a) Health Canada 2012

The TEQ approach 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 TEFs, (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 Tier 1 agencies were then searched to identify appropriate limits for use in each approach.

[1] Non-carcinogenic PAHs were evaluated on their own or as part of the appropriate aromatic hydrocarbon group.

Table 9-3
Chronic Inhalation Exposure Limits for Benzo(a)pyrene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	AAQO	0.0003	ESRD 2013
Health Canada	RsC	0.32	Health Canada 2010
OEHHA	RsC –	0.009 –	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	RsC	0.00012	WHO 2000

– = Not available

The WHO (2000) recommends an inhalation unit risk of 8.7E-05 per ng/m^3 (0.09 per µg/m³) based on epidemiological data from studies in coke-oven workers. The WHO (2000) identified an upper-bound individual lifetime unit risk estimate associated with continuous exposure to 1 µg/m³ of benzene-soluble compounds of coke-oven emissions in ambient air of 0.00062 (µg/m³)$^{-1}$ based on a linearized multistage model. Benzo(a)pyrene was selected as an indicator of general PAH mixtures from emissions of coke ovens and similar combustion processes in urban air. In the benzene-soluble fraction of coke oven emissions, 0.71% is reported to be benzo(a)pyrene. On this basis, the lifetime risk of lung cancer of 0.09 per µg/m³ was calculated (WHO 2000), which equates to an RsC of 0.00012 µg/m³ that is associated with an acceptable incremental lifetime cancer risk of one in 100,000. The WHO RsC of 0.00012 µg/m³ based on the incidence of lung cancer, was selected for use in the first approach of the chronic inhalation assessment of benzo(a)pyrene (Approach 1).

Health Canada (2010) derived an inhalation unit risk of 3.10E-02 per mg/m³, which equates to an RsC of 0.32 µg/m³. This RsC is associated with an acceptable incremental lifetime cancer risk of development of respiratory tumours of one in 100,000. The RsC was developed based on exposure to benzo(a)pyrene via multi-stage modelling of respiratory tract tumours in Syrian golden hamsters (Thyssen *et al.* 1981; Government of Canada 1994). In the key study, groups of 24 male Syrian golden hamsters were exposed by inhalation (nose only) to 0, 2.2, 9.5, or 45.6 mg/m³ benzo(a)pyrene for 4.5 hours/day, 7 days/week for the first 10 weeks, and for 3 hours/day for the rest of the exposure period (up to 96 weeks). A decrease in body weight gain in exposed animals was observed during the first 10 weeks of the study; however, with the exception of the high exposure group, the body weights of all surviving exposed animals were similar to those of the controls from the 10th to the 60th week. Mean survival decreased only in the highest exposure group.

The incidences of unspecified tumours of the respiratory tract (nasal cavity, larynx, and trachea) were:

- 0/27 for controls;
- 0/27 for the low-dose group;

- 9/26 (35%) for the mid-dose group; and
- 13/25 (52%) for the high-dose group (Thyssen *et al.* 1981).

Exposure related neoplasms (unspecified) were present in the pharynx (0, 0, 23, and 56% for control, low-, mid-, and high-dose, respectively), esophagus (0, 0, 0, and 8% for control, low-, mid-, and high-dose, respectively), and forestomach (0, 0, 4, and 4% for control, low-, mid-, and high-dose, respectively). Lung tumours were not observed (Thyssen *et al.* 1981). The Health Canada RsC of 0.32 µg/m³ based on the incidence of lung tumours was selected for the chronic inhalation assessment of benzo(a)pyrene using the TEQ approach (Approach 2).

As both of the benzo(a)pyrene limits selected are based on the same endpoint (lung cancer incidence), the higher of the two calculated risk estimates was added to the calculated risk estimate of any other chemicals sharing this endpoint (if any) in the mixture assessment.

The OEHHA (2009) presents an inhalation unit risk estimate of 1.1E-03 per µg/m³ (equivalent to an RsC of 0.009 µg/m³). This value was derived from the Thyssen *et al.* 1981 study (discussed above as the basis of the Health Canada value). Linearized multistage modelling was used to evaluate the respiratory tumour incidence data. The OEHHA applied a default body weight scaling method to account for differences in body surface area and body weight. According to the US EPA (2005) Cancer Risk Assessment guidance, for inhalation exposures, other approaches such as tract specific scaling are specified. The body weight scaling approach used by the OEHHA is consistent with the US EPA (2005) guidance for oral exposures, but not inhalation. In addition, the Government of Canada (1994) analysis of the tumourigenicity data is more substantial and technical than what is provided for the OEHHA (2009) value. On the basis that the Health Canada (2010) value represents the most defensible RsC for use in the chronic inhalation assessment based on the quality of the supporting documentation and methodologies used, the OEHHA (2009) value was not selected.

No supporting document for the ESRD 2013 value of 0.0003 µg/m³ was available. As a result, the ESRD value was not used in the assessment.

C9.3 Oral Exposure Limits

Table 9-4
Chronic Oral Exposure Limits for Benzo(a)pyrene

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	RsD –	0.0043 –	Health Canada 2010 Health Canada 2013
OEHHA	RsD –	0.001 –	OEHHA 2009 OEHHA 2014
RIVM	CR	0.05	RIVM 2001
US EPA	RsD	0.0014	US EPA 2014, 1994
WHO	–	–	WHO 2014, 2003

– = Not available

The US EPA (2014, 1994) provides an oral slope factor of 7.3 per mg/kg bw/d based on the geometric mean of four slope factors obtained by different modelling procedures and multiple

datasets from two different studies, including the Neal and Rigdon (1967) study that was used in the Health Canada (1988) assessment. The US EPA (1994) considered each of these datasets to be acceptable for the derivation of an oral slope factor for benzo(a)pyrene, but less-than-optimal. As a result, the use of a geometric mean of the four slope factors was preferred because it made use of more of the available data. The four slope factors were calculated as follows:

The Neal and Rigdon (1967) data was fit to a two-stage dose response model that included a term to permit the modelling of benzo(a)pyrene as its own promoter (modification of Moolgavkar-Venson-Knudson, generalized forms of two-stage model). In this model, the transition rates and the growth rate of pre-neoplastic cells were both considered to be exposure-dependent. In addition to the Neal and Rigdon (1967) control group, historical control stomach tumour data from a related, but not identical, mouse strain (SWR/J Swill) was used in the modelling (Rabstein *et al.* 1973). In the historical control data, the forestomach tumour incidence rate was 2/268 and 1/402 for males and females, respectively. The lifetime unit risk for humans was calculated based on the following standard assumptions: mouse food consumption was 13% of its body weight per day, human body weight was assumed to be 70 kg, and the assumed body weight of the mouse 0.034 kg (US EPA 1994). The standard assumption of surface area equivalence between mice and humans was the cube root of 70 kg/0.034 kg. A conditional upper-bound estimate was calculated to be 5.9 per mg/kg bw/d (US EPA 1994).

The same dataset as above was used to generate an upper-bound estimate extrapolated linearly from the 10% response point to the background of an empirically fitted dose-response curve (modification of Moolgavkar-Venson-Knudson, generalized forms of two-stage model). An upper-bound risk estimate was calculated to be 9.0 per mg/kg bw/d (US EPA 1994).

In order to reflect the partial lifetime exposure pattern over different parts of the animals' lifetimes, a generalized Weibull-type dose-response model was selected to assess the Neal and Rigdon (1967) data alone (*i.e.*, excluding the two additional control groups from Rabstein *et al.* 1973). An upper-bound was calculated to be 4.5 per mg/kg bw/d (US EPA 1994).

A linearized multistage procedure was used to calculate an upper bound estimate for humans from the Brune *et al.* (1981) rat dataset. Sprague-Dawley (rats/sex/group) were fed 0.15 mg/kg benzo(a)pyrene (reported to be 'highly pure') in the diet of either every 9th day or 5 days/week. These treatments resulted in annual average doses of 6 or 39 mg/kg, respectively. The control group contained 32 rats per sex. Treatment continued until the rats were moribund or dead; survival was similar in all groups. The combined incidence of tumours of the forestomach, esophagus and larynx was 3/64, 3/64 and 10/64 in the control group, the group fed benzo(a)pyrene every 9th day, and the group fed benzo(a)pyrene five times per week, respectively. A trend analysis showed a statistically significant tendency for the proportion of animals with tumours of the forestomach, esophagus or larynx to increase steadily with dose. An oral slope factor of 11.7 per mg/kg bw/d was calculated (US EPA 1994).

Because the US EPA considered (i) different modelling procedures, (ii) multiple datasets from two different studies, and (iii) both sexes of more than one strain of mice and species of out bred rodents, the US EPA RsD of 0.0014 µg/kg bw/d based on gastrointestinal tumours was selected as the chronic oral limit for assessing the mixture of carcinogenic PAHs using the TEQ approach (Approach 2).

Health Canada (2010) presents an oral slope factor of 2.3 (mg/kg bw/d)$^{-1}$ (equivalent to an RsD of 0.004 µg/kg bw/d), based on the Canadian guidelines for drinking water (Health Canada 1988). The Canadian drinking water quality guideline for benzo(a)pyrene took into consideration the increased incidence of stomach tumours (squamous cell papillomas and some carcinomas) (Health Canada 1988; Neal and Rigdon 1967). In the key study, male and female CFW-Swiss mice were fed concentrations of 0 ppm, 1 ppm, 10 ppm, 20 ppm, 40 ppm, 45 ppm, 50 ppm, 100 ppm or 250 ppm benzo(a)pyrene in the diet (purity was not reported). The control group contained 289 mice (number of mice/sex was not specified). No forestomach tumours were reported in the 0 ppm, 1 ppm, or 10 ppm dose groups. The incidence of forestomach tumours in the 20 ppm, 40 ppm, 45 ppm, 50 ppm, 100 ppm or 250 ppm dose groups were 1/23, 0/37, 1/40, 4/40, 23/40, 19/23 and 66/73, respectively. Incorporating a surface area correction and using the robust linear extrapolation model, the unit lifetime risk associated with the ingestion of 1 µg/L benzo(a)pyrene in drinking water was estimated as 5 × 10-5. Using an adult body weight of 70.7 kg and an adult water ingestion rate of 1.5 L/day (Health Canada 2012), an oral slope factor of 2.3 per mg/kg bw/d was calculated. The US EPA value was used over this value as it took more studies into consideration than just the Neal and Rigdon data set.

The OEHHA (2009) has derived an oral slope factor of 11.5 (mg/kg bw/d)$^{-1}$ (equivalent to an RsD of 0.001 µg/kg bw/d) based on the Neal and Rigdon (1967) data. However the approaches used are not clear in the supporting document. As a result, this value was not used in the assessment.

The RIVM (2001) presents an oral RsD of 0.5 µg/kg bw/d associated with a one in 10,000 risk level (or 0.05 µg/kg bw/d for a one in 100,000 risk level). This value was derived from a study, in which rats were administered 0, 3, 10 or 30 mg/kg/d of benzo(a)pyrene via oral gavage, 5 days/week for a duration of 2 years. Tumours in the forestomach, liver, kidney, skin, intestine and auditory canal and sarcomas of the esophagus, skin, and mammary glands were observed. This value was not used in the assessment because the US EPA RsD is more conservative.

C9.4 Summary of Exposure Limits

Table 9-5
Summary of Exposure Limits Selected for Benzo(a)pyrene

Duration	Averaging-Time	Exposure Pathway	Chemical	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	n/a	Inhalation	n/a	RfC	n/a	µg/m³	–	–
Chronic	Annual	Inhalation	B(a)P (Approach 1)	RsC	0.00012	µg/m³	WHO 2000	Lung tumours
			B(a)P equivalent (Approach 2)	RsC	0.32	µg/m³	Health Canada 2010	Lung tumours
		Multiple Exposure Pathway	B(a)P equivalent	RsD	0.0014	µg/kg bw/d	US EPA 2014, 1994	Gastrointestinal tumours

– = Not available
n/a = Not applicable

C9.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 2013. TLVs® and BEIs® Based on the Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. ISBN: 978-1-607260-28-8. ACGIH®, Cincinnati. OH.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Brune, H., R.P. Deutsch-Wenzel, M. Habs, S. Ivankovic and D. Schmahl. 1981. Investigation of the tumorigenic response to benzo(a)pyrene in aqueous caffeine solution applied orally to Sprague-Dawley rats. J Cancer Res Clin Oncol 102(2): 153-157.

Equilibrium and URS (Equilibrium Environmental Inc. and URS Canada Inc.). 2006. Potency Equivalency Factors for Carcinogenic Polycyclic Aromatic Hydrocarbons. Prepared for: Health Canada, Contaminated Sites Division.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Government of Canada. 1994. Canadian Environmental Protection Agency. Priority Substances List Assessment Report - Polycyclic Aromatic Hydrocarbons. ISBN: 0-662-22209-1.

Health Canada. 1988. Guidelines for Canadian Drinking Water Quality - Supporting Document for Benzo(a)pyrene. Prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment. Ottawa, ON: Health Canada.

Health Canada. 2010. 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. September 2010.

Health Canada. 2012. 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.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http: //hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

Neal, J. and R.H. Rigdon. 1967. Gastric tumors in mice fed benzo[a]pyrene: a quantitative study. Tex. Rep. Biol. Med. 25(4): 553-557.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Rabstein, L.S., R.L. Peters and G.J. Spahn. 1973. Spontaneous tumours and pathologic lesions in SWR/J mice. J Natl Cancer Inst 50(3): 751-758.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

Thyssen, J., J. Althoff, G. Kimmerle and U. Mohr. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J Nat Cancer Inst 66(3): 575-577.

US EPA (United States Environmental Protection Agency). 1994. IRIS Summary of Benzo(a)pyrene (CASRN 50-32-8). Carcinogenicity Assessment for Lifetime Exposure. Available at: http://www.epa.gov/ncea/iris/subst/0136.htm#carc

US EPA (United States Environmental Protection Agency). 2002. A Review of the Reference Dose and Reference Concentration Processes. Final Report. Risk Assessment Forum, United States Environmental Protection Agency. Washington, DC. EPA/630/P02/002F.

US EPA (United States Environmental Protection Agency). 2005. Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum, Washington DC. EPA/630/P-03/001F. March 2005.

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2003. Polynuclear aromatic hydrocarbons in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/polyaromahydrocarbons.pdf

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C10.0 CARBON MONOXIDE

C10.1 Acute Inhalation Exposure Limits

Table 10-1
Acute Inhalation Exposure Limits for Carbon Monoxide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE[(1)]	1-hour Level A	14,300	BC MOE 2013
	1-hour Level B	28,000	
	1-hour Level C	35,000	
	8-hour Level A	5,500	
	8-hour Level B	11,000	
	8-hour Level C	14,300	
ESRD	1-hour AAQO	15,000	ESRD 2013
	8-hour AAQO	6,000	
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	1-hour REL	23,000	OEHHA 2014
OMOE	30-minute	6000	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	1-hour NAAQS	40,000	US EPA 2011, 2010
	8-hour NAAQS	10,000	
WHO	15-minute	100,000	WHO 2000
	30-minute	60,000	
	1-hour Guideline	30,000	
	8-hour Guideline	10,000	

– = Not available
(1) Values for CO from the BC MOE are PCO for Food-processing, Agriculturally Orientated, and Other Misc. Industries

The US EPA (2011) has developed two National Ambient Air Quality Standards (NAAQS) for carbon monoxide (CO): a 1-hour standard of 40,000 µg/m³ and an 8-hour standard of 10,000 µg/m³. These values are based on blood carboxyhemoglobin (COHb) concentrations ranging from 2.1 to 2.9%, representing the levels of concern identified by the US EPA from several controlled human studies.

Concentrations associated with this range of COHb represent about a 2.5% increase above baseline values. Overall, there is a lack of information regarding adverse effects and COHb concentrations below 2%. In the derivation of the 8-hour standard, the US EPA concluded that ambient CO concentrations equivalent to the 8-hour standard of 10,000 µg/m³ would be unlikely to increase COHb concentrations above 2.1% in non-smokers. It was further concluded that ambient air exposure (excluding indoor sources) of 10,000 µg/m³ is associated with a relatively low degree of potential risk to sensitive, non-smoking individuals. While specifics regarding the key studies that these two standards are based on are not clear, it is apparent that the US EPA has recently reviewed a substantial amount of information as part of the Integrated Science Assessment (US EPA 2010 that accompanies this Rule). An equation (Coburn Forster Kane) was used by the US EPA to take into account CO uptake and kinetics in the derivation and

review of the standards. The US EPA 1-hour and 8-hour NAAQS of 40,000 µg/m³ and 10,000 µg/m³ were selected for use in the assessment, as these values are associated with the most recent and thorough review of CO toxicity.

The BC MOE (2013) has derived Level A, B and C 1-hour and 8-hour Pollution Control Objectives of 14,300, 28,000, 35,000, 5,500, 11,000 and 14,300 µg/m³ respectively. These values have been derived for food-processing, agricultural orientation and other miscellaneous industries. The averaging periods are separated into Level A, B and C to reflect the different conditions under which criteria may be applied. Due to the lack of supporting documentation provided by the BC MOE and the intended purpose of the values (*i.e.*, agriculture and food), these values were not selected for use in the assessment.

ESRD (2013) provides a 1-hour AAQO of 15,000 µg/m³ and an 8-hour AAQO of 6,000 µg/m³ for CO. These AAQOs were adopted from the Canadian Environmental Protection Act and Federal Provincial Advisory Committee (CEPA/FPAC) Working Group on Air Quality Objectives and Guidelines, which recommends maximum desirable, acceptable and tolerable objectives for CO. The Alberta objectives are based on the maximum desirable levels (*i.e.*, the lowest objective). These objectives were developed to protect the subpopulation sensitive to cardio-respiratory effects (CEPA/FPAC 1994). Given that the US EPA 1-hour and 8-hour values are more thoroughly documented than the ESRD AAQOs for CO and have been reviewed more recently, the ESRD AAQOs for CO were not used in the assessment.

The OEHHA (2014) has derived a 1-hour REL of 23,000 µg/m³. This value is based on the observed aggravation of pre-existing angina and other cardiovascular conditions. Increased COHb concentrations in blood have been associated with CO toxicity. A COHb concentration as low as 2% has been associated with an aggravation of angina symptoms. The OEHHA (2014) cites a NOAEL based on COHb concentrations ranging from 1.1% to 1.3%, corresponding to a CO concentration of about 20 ppm (*i.e.*, 23,000 µg/m³). However, no information regarding the design features (duration of exposure, concentrations, number of subjects, *etc.*) were provided for the key study. As a result of the limited information provided in the supporting document, this value was not used in the assessment.

The OMOE (2012) provides a 30-minute average of 6,000 µg/m³ based on health effects. As the OMOE does not provide supporting documentation for the derivation of this 30-minute standard, this limit was not considered for the acute inhalation assessment.

The World Health Organization (WHO 2000) has derived 1-hour and 8-hour Ambient Air Quality Guidelines of 30,000 and 10,000 µg/m³, respectively. Values for 15-minute and 30-minute averaging times also were provided in WHO (2000).. The WHO values were derived to prevent blood COHb concentrations from exceeding 2.5%. The WHO (2000) notes that during pregnancy, endogenous maternal blood COHb increases and can range from 0.7 to 2.5%. Also, it is noted that blood concentrations between 2 and 10% have been associated with low fetal birth weights. The threshold of 2.5% appears to have been derived based on this information. The WHO (2000) states that the Coburn Forster Kane equation was applied to account for all potential routes of CO uptake in the derivation of the guidelines, although further details are not provided. It is not evident that any uncertainty factors were applied in the derivation of the guidelines, however, sensitive individuals (pregnant women and foetuses) have been accounted for. Given that the US EPA presents the most recent and comprehensive documentation in support of the 1-hour and 8-hour standards, the WHO values were not used in the assessment.

C10.2 Chronic Inhalation Exposure Limits

Table 10-2
Chronic Inhalation Exposure Limits for Carbon Monoxide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2011
WHO	–	–	WHO 2000

– = Not available

No regulatory exposure limits were available for chronic exposure to CO, and it was not assessed on a chronic basis. The critical effect of carbon monoxide exposure is the formation of COHb in blood. Given that COHb concentrations reach a steady-state after 6 to 8 hours of exposure, CO exposure for longer periods of time (*i.e.*, chronic exposure), is not expected to cause accumulation of COHb in the blood (WHO 2000). The recent US EPA (2010) Integrated Science Assessment for CO concluded that there is no association between long term exposure to CO and mortality.

C10.3 Oral Exposure Limits

Carbon monoxide is a gaseous criteria air contaminant. As such, it was not evaluated in the multiple pathway assessment.

C10.4 Summary of Exposure Limits

Table 10-3
Summary of Exposure Limits Selected for Carbon Monoxide

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	NAAQS	40,000	µg/m³	US EPA 2011, 2010	Hypoxia
	8-hour	Inhalation	NAAQS	10,000	µg/m³	US EPA 2011, 2010	Hypoxia
Chronic	Annual	Inhalation	RsC	n/a	µg/m³	n/a	n/a
			RfC	n/a	µg/m³	n/a	n/a
		Multiple Exposure Pathway	RsD	n/a	µg/kg bw/d	n/a	n/a
			RfD	n/a	µg/kg bw/d	n/a	n/a

n/a = Not applicable

C10.5 References

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

BC MOE (British Columbia Ministry of the Environment). 2013. B.C. Ambient Air Quality Objectives. http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

CEPA/FPAC (Canadian Environmental Protection Act/Federal-Provincial Advisory Committee). 1994. Working Group on Air Quality Objectives and Guidelines. National Ambient Air Quality Objectives for Carbon Monoxide. Desirable, Acceptable and Tolerable Levels. Canadian Environmental Protection Act. Ottawa, ON.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of

Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2010. Integrated Science Assessment for Carbon Monoxide. National Center for Environmental Assessment. EPA/600/R-09/019F.

US EPA (United States Environmental Protection Agency). 2011. National Ambient Air Quality Standards for Carbon Monoxide: Proposed Rule. http://edocket.access.gpo.gov/2011/pdf/2011-2404.pdf

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C11.0 CYCLOHEXANE

C11.1 Acute Inhalation Exposure Limits

Table 11-1
Acute Inhalation Exposure Limits for Cyclohexane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Standard	6,100	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OMOE (2012, 2005) has established a 24-hour standard of 6,100 µg/m³ for cyclohexane based on a NOAEL of 6,886 mg/m³ for reduced pup weights in the F1 and F2 generations in a reproductive and developmental inhalation study (Kreckmann *et al.* 2000). The NOAEL was revised to a HEC of 1,722 mg/m³ and the lower confidence of the benchmark concentration (BMCL) was then derived (1,822 mg/m³). An uncertainty factor of 300 was applied to the BMCL to account for intraspecies variability (10), interspecies variability (3), and database deficiencies due to the lack of chronic studies specifically examining developmental neurotoxicity and hepatic effects (10) (OMOE 2005). Due to the long-term study duration and the uncertainty factor applied by the OMOE for subchronic exposure, this value was not selected for use.

As acute inhalation exposure limits for cyclohexane were not available from any other agencies listed above, the toxicity search was expanded to include acute exposure limits from ACGIH (2013) and AEGL-1 values from the US EPA (2013a). The search did not identify any acute exposure limits from these agencies. As a result, cyclohexane was not evaluated on an individual basis in the acute inhalation assessment.

Due to its structural characteristics, cyclohexane was evaluated as a component of the aliphatic C_5-C_8 group. Please refer to the toxicity profile for the aliphatic C_5-C_8 group for details of the limit.

C11.2 Chronic Inhalation Exposure Limits

Table 11-2
Chronic Inhalation Exposure Limits for Cyclohexane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2014 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	RfC	6,000	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The US EPA (2014, 2003) has derived a chronic RfC of 6,000 µg/m³ based on a two-generational reproductive and developmental inhalation study. Male and female Crl:CD BR rats (30/sex/concentration) were exposed by whole body inhalation to 0, 500, 2,000, or 7,000 ppm (0, 1,721, 6,886, or 24,101 mg/m³) cyclohexane vapour for 6 hours/day, 5 days/week for 10 weeks (Kreckmann *et al.* 2000). Rats were bred with their respective treatment group and allowed to deliver and rear their offspring until weaning. Females were exposed daily after breeding throughout pregnancy and lactation, with the exception of gestation day 21 until day 4 of lactation when they were not exposed. Neonate rats were not directly exposed to cyclohexane. At weaning, F1 rats were randomly selected to produce the next generation and were treated to the same exposure schedule as the P1 generation. At least 11 weeks after weaning, the F1 rats were bred to produce the F2 litters.

The NOAEL of 6,886 mg/m³ for maternal toxicity (reduced body weights, altered response to stimuli) and reduced pup weights was duration-adjusted from an intermittent exposure to a continuous exposure (6/24 hours), resulting in a $NOAEL_{ADJ}$ of 1,720 mg/m³ for developmental effects. Benchmark dose modelling was conducted by the US EPA, and a BMC_{1sd} of 1,822 mg/m^3 was calculated from the dose-response data. The BMC_{1sd} was converted to a HEC for a category 3 gas causing respiratory effects. The average ratio of the animal-blood:air partition coefficient would be marginally greater than 1; thus, a default value of 1 was used in calculating the BMC_{HEC} of 1,822 mg/m³. A cumulative uncertainty factor of 300 was applied to the BMC to account for interspecies variability (3), intraspecies variability (10), and database deficiencies (10). A factor of 3 was applied for the extrapolation of laboratory animal data to humans since the calculation of a HEC addressed the pharmacokinetic aspects of the interspecies uncertainty factor. Accordingly, only the pharmacodynamic aspects of uncertainty remain as a partial factor for interspecies uncertainty (US EPA 2003). The US EPA RfC of 6,000 µg/m³ was selected as the chronic inhalation limit for cyclohexane.

Cyclohexane was assessed individually as well as a component of the aliphatic C_5-C_8 group.

C11.3 Oral Exposure Limits

Cyclohexane was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, cyclohexane was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C11.4 Summary of Exposure Limits

Table 11-3
Summary of Exposure Limits Selected for Cyclohexane

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	–	–	µg/m³	–	–
Chronic	Annual	Inhalation	RfC	6,000	µg/m³	US EPA 2014, 2003	Reproductive and developmental effects
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	n/a	n/a

– = Not available
n/a = Not applicable

C11.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

Kreckmann, K.H., J.K. Baldwin, L.G. Roberts, R.J. Staab, D.P. Kelly and J.E. Saik. 2000. Inhalation developmental toxicity and reproduction studies with cyclohexane. Drug Chem. Toxicol. 23(4):555-573.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2005. Ontario Air Standards for Cyclohexane. Standards Development Branch. Ontario Ministry of the Environment. June 2005.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Cyclohexane). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2003. Cyclohexane. Chronic Inhalation Reference Concentration. United States Environmental Protection Agency. Available at: http://www.epa.gov/ncea/iris/subst/1005.htm

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

C12.0 ETHYLBENZENE

C12.1 Acute Inhalation Exposure Limits

Table 12-1
Acute Inhalation Exposure Limits for Ethylbenzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	acute MRL	21,700	ATSDR 2013, 2010
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO	2,000	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Standard	1,000	OMOE 2013
TCEQ	acute ReV	86,000	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The ATSDR (2013, 2010) provides an acute inhalation MRL of 5 ppm (21,700 µg/m³) based on neurological effects in rats. Wag/Rij rats were exposed to 0, 300, 400, or 550 ppm (0, 1,302, 1,736, or 2,387 mg/m³) ethylbenzene (99% pure) for 8 hours/day for 5 days (Cappaert *et al.* 2000). Three to six weeks following cessation of exposure, Measurement of Distortion Product Otoacoustic Emissions (DPOAE), Compound Action Potential (CAP), and hair cell counts were conducted. Although Cappaert *et al.* (2000) only provided the results of the study graphically the ATSDR was able to obtain the individual animal data directly from Cappaert *et al.*, allowing for use of the BMD model approach. Benchmark dose modelling was completed using the CAP auditory threshold data, where the largest effects were observed in response to 8, 12 and 16 kHz stimuli. The BMD model estimated $BMDL_{1SD}$ values of 102.3, 89.47, and 81.10 µmol/L at 8, 12 and 16 kHz, respectively. The lowest $BMDL_{1SD}$ of 81.10 µmol/L was used as the POD for the acute inhalation MRL. A HEC of 154.26 ppm (669.49 mg/m³) was calculated using the human PBPK model, a human body weight of 70 kg, and the assumption of 14-day continuous exposure. A cumulative uncertainty factor of 30 was applied to the $BMDL_{HEC}$ to account for extrapolation from animals to humans with dosimetric adjustment (3) and for human variability (10). The result is an acute inhalation MRL of 21,700 µg/m³ which was used as a 1-hour exposure limit in the acute effects assessment of ethylbenzene.

The TCEQ (2013) provides an acute ReV of 86,000 µg/m³ based on the same key study as the ATSDR (*i.e.*, Cappaert *et al.* 2000). However, the TCEQ did not obtain the individual animal data directly from Cappaert *et al.* and thus used the NOAEL/LOAEL approach over the BMD model approach to determine the POD for the development of the acute ReV. A NOAEL of 300 ppm (1,302 mg/m³) and a LOAEL of 400 ppm (1,736 mg/m³) were identified for significant deterioration in CAP auditory thresholds and significant outer hair cell losses. The 8-hour NOAEL was adjusted to a 1-hour NOAEL using modified Haber's law.

$$C_{ADJ}^{n} \times T_{ADJ} = C^{n} \times T$$
$$C^{3} \times 1 \text{ hour} = (1{,}302 \text{ mg/m}^3)^{3} \times 8 \text{ hours}$$

Where:

C_{ADJ}	=	duration-adjusted concentration
T_{ADJ}	=	desired time of exposure (1 hour)
C	=	concentration of exposure (1,302 mg/m³)
T	=	time of exposure (8 hours)
n	=	chemical-specific modification factor designed to account for the toxicity of a chemical being concentration and duration dependent (3).

The HEC was calculated from the $NOAEL_{ADJ}$ of 600 ppm (2,604 mg/m³) using the recommended equation for category 3 gases. The TCEQ notes, however, that ethylbenzene is classified as a category 2 gas since it is relatively soluble in water and produces both local and systemic effects, but category 2 gases are still under review by the US EPA.

$$RGDR = (H_{b/g})_A / (H_{b/g})_H$$

Where:

RGDR	=	regional gas dosimetry ratio
$H_{b/g}$	=	ratio of blood:gas partition coefficient
A	=	animal
H	=	human

The TCEQ (2013) assumed an $H_{b/g}$ for rats of 42.7 and a mean $H_{b/g}$ for humans of 28.0. When the $(H_{b/g})_A/(H_{b/g})_H$ is greater than 1, a default value of 1 is used for the RGDR. The RGDR was then multiplied by the $NOAEL_{ADJ}$, resulting in a $NOAEL_{HEC}$ of 600 ppm (2,604 mg/m³). The TCEQ (2013) applied a cumulative uncertainty factor of 30 to the $NOAEL_{HEC}$ to account for interspecies variability with dosimetric adjustment (3) and intraspecies variability (10). The result is an acute ReV of 86,000 µg/m³ for ethylbenzene. The TCEQ acute ReV was not used in the acute effects assessment for ethylbenzene because: (a) the TCEQ did not provide sufficient evidence to justify the use of this less conservative (*i.e.*, higher) limit over the ATSDR acute MRL of 21,700 µg/m³ that is based on the same key study; and, (b) the ATSDR obtained the individual animal data, and applied the BMD and PBPK models in the development of its acute MRL.

The OMOE (2013) has established a health-based 24-hour standard of 1,000 µg/m³ for ethylbenzene. However, no scientific basis or supporting document is provided for this standard. As a result, this limit was not used in the acute effects assessment of ethylbenzene.

ESRD (2013) presents an AAQO of 2,000 µg/m³ for a 1-hour average exposure. This limit was adopted from the TCEQ based on odour perception, but no specific basis was provided. As well, the TCEQ (2013) recently revised its acute odour-based acute ESL to a value of 740 µg/m³. Given that this objective is not health-based and does not reflect TCEQ's most current odour-based acute ESL, the ESRD AAQO was not used in the acute effects assessment of ethylbenzene.

C12.2 Chronic Inhalation Exposure Limits

Table 12-2
Chronic Inhalation Exposure Limits for Ethylbenzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	260	ATSDR 2013, 2010
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	1,000	Health Canada 2010
OEHHA	RsC REL	4 2,000	OEHHA 2009 OEHHA 2014, 2000
RIVM	TCA	770	RIVM 2001
TCEQ	ReV	1,900	TCEQ 2013, 2010
US EPA	RfC	1,000	US EPA 2014, 1991
WHO	–	–	WHO 2000

– = Not available

The ATSDR (2013, 2010), OEHHA (2014, 2000) and TCEQ (2013) have each developed their respective limits using the same key study – NTP (1999). In NTP (1999), male and female F344/N rats and B6C3F$_1$ mice were exposed to 0, 75, 250 or 750 ppm ethylbenzene via inhalation 6 hours/day, 5 days/week for 103 to 104 weeks. Increased severity of nephropathy was statistically significant for the 750 ppm male exposure group and for all female exposure groups (*i.e.*, 75, 250 and 750 ppm). However, the TCEQ (2013) notes that, for the 75 ppm female group, the severity of nephropathy was minimal to mild, that clinical findings and survival were unaffected by treatment, and the severity of nephropathy was similar to the control group. On this basis, the TCEQ (2013) and OEHHA (2000) selected 75 ppm as the NOAEL for increased severity of nephropathy. When adjusted for intermittent exposure (6/24 hours × 5/7 days), the NOAEL$_{ADJ}$ was calculated to be about 13 ppm (58 mg/m³). The TCEQ (2013) and OEHHA (2000) concluded that the RGDR should be equal to 1; thus, the NOAEL$_{HEC}$ was assumed to be 13 ppm. A cumulative uncertainty factor of 30 was applied by both the TCEQ (2013) and OEHHA (2000) to account for interspecies differences (3) and intraspecies variability (10). The result is a TCEQ ReV of 1,900 µg/m³ and an OEHHA REL of 2,000 µg/m³.

The ATSDR (2013, 2010) selected 75 ppm as the LOAEL for increased severity of nephropathy. The human PBPK model was used to estimate the internal dose metrics and predict the HEC of 17.45 ppm (75.73 mg/m³). The ATSDR (2010) applied a cumulative uncertainty factor of 300 to account for use of a LOAEL (10), extrapolation from animals to humans with dosimetric adjustment differences (3), and human variability (10). The resulting MRL of 260 µg/m³ was used in the chronic inhalation effects assessment of ethylbenzene as it is based on the more conservative (*i.e.*, lower) effect level of 75 ppm for increased severity of kidney effects instead of a no effect level of 75 ppm, and incorporates dosimetry modelling data instead of the RGDR approach to partially account for the uncertainty associated with extrapolation from rats to humans.

The OEHHA (2009) also provides a unit risk estimate of 2.5E-06 $(\mu g/m^3)^{-1}$ (equivalent to an RsC of 4 µg/m³). This value is based on the incidence of renal tumours in exposed rats. However, in

the US EPA (2014, 1991) carcinogenicity assessment of ethylbenzene, it is stated that the metabolic pathways for ethylbenzene are different between rodents and humans, and that the mutagenic metabolites observed in rodents have not been observed in humans. The US EPA (2014) did not derive a chronic inhalation quantitative estimate for carcinogenic risk due to the lack of data available. As such, the carcinogenic RsC value from the OEHHA (2009) was not used in the chronic effects assessment, due to the existence of more biologically relevant values.

The US EPA (2014, 1991) assessment of ethylbenzene reports an RfC of 1,000 µg/m³ based on a NOAEL of 100 ppm (434 mg/m³) for developmental toxicity in rats and rabbits. Wistar rats and New Zealand white rabbits were exposed to concentrations of 0, 100 or 1,000 ppm (0, 434 or 4,342 mg/m³) for 6 to 7 hours/day, 7 days/week during days 1 to 19 and 1 to 24 of gestation, respectively. According to the US EPA (1991), a NOAEL based on developmental effects should not be adjusted for intermittent exposure. A $NOAEL_{HEC}$ was calculated assuming a default value of 1.0 since *b:a* lambda values are unknown for the experimental animal species (a) and humans (h) (US EPA 1991). A cumulative uncertainty factor of 300 was applied to the study $NOAEL_{HEC}$ to account for interspecies variability (3), intra-species variability (10), and the absence of multigenerational reproductive and chronic studies (10). An uncertainty factor of 3 for interspecies variability was considered appropriate by the US EPA (1991) since the HEC adjustment addresses the pharmacokinetic component of the extrapolation factor, leaving only the pharmacodynamic area of uncertainty. This study only involved two dose levels (100 and 1,000 ppm). Adverse effects were observed at 1,000 ppm, but due to the lack of dose levels between 100 and 1,000 ppm, the threshold of these effects is unknown. The TCEQ (2013) and OEHHA (2000) discuss the US EPA RfC and its basis relative to the scientific weight of evidence for subchronic and chronic ethylbenzene exposure. The US EPA evaluation incorporated an uncertainty factor of 10 for the lack of multigenerational reproductive and chronic studies; however, both of these study types have since become available. For these reasons, the US EPA RfC was not used in the chronic inhalation assessment of ethylbenzene.

The Health Canada (2010) inhalation TC of 1,000 µg/m³ was adopted from the US EPA (1991). Thus, based on the same rationale for the exclusion of the US EPA RfC, the Health Canada TC was not used in the chronic inhalation assessment of ethylbenzene.

The RIVM (2001) provides a TCA of 770 µg/m³ based on kidney and liver effects in rats and mice. The TCA value was derived from a NOAEL of 430 mg/m³ (100 ppm) identified in the 1992 subchronic NTP (1996) study. The RIVM (2001) adjusted the NOAEL for intermittent exposure (6/24 hours × 5/7 days) and applied an uncertainty factor of 100 to the duration-adjusted NOAEL of 77 mg/m³ to account for interspecies variability (10) and intraspecies variability (10). An uncertainty factor was not applied to the NOAEL by the RIVM (2001) for use of a subchronic study because a higher NOAEL of 1,075 mg/m³ was reported in a chronic NTP study. This TCA from RIVM was not used in the chronic inhalation effects assessment because it is based on subchronic instead of chronic exposure data.

C12.3 Oral Exposure Limits

Ethylbenzene was not incorporated in the multiple pathway exposure assessment because it did not meet the physical-chemical criteria used to define non-volatile chemicals. Thus, a chronic oral exposure limit was not required for ethylbenzene.

C12.4 Summary of Exposure Limits

Table 12-3
Summary of Exposure Limits Selected for Ethylbenzene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	MRL	21,700	µg/m³	ATSDR 2013, 2010	Neurological effects
Chronic	Annual	Inhalation	RsC	-	µg/m³	–	–
			RfC	260	µg/m³	ATSDR 2013, 2010	Kidney effects
		Multiple Exposure Pathway	RsD	n/a	µg/kg bw/d	n/a	n/a
			RfD	n/a	µg/kg bw/d	n/a	n/a

– = Not available
n/a = Not applicable

C12.5 References

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

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Cappaert, N.L.M., S.F,L. Klis, A.B. Baretta, H. Muijser and G.F. Smoorenburg. 2000. Ethyl benzene-induced ototoxicity in rats: A dose-dependent mid-frequency hearing loss. J Assoc Res Otolaryngol 1(4):292-299.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2010. 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. September 2010.

NTP (National Toxicology Program). 1996. Toxicology and Carcinogenesis studies of ethylbenzene in F344/N rats and B6CF$_1$ mice (inhalation studies). Technical Report Series no 466. Cited in: RIVM 2001.

NTP (National Toxicology Program). 1999. Toxicology and Carcinogenesis Studies of Ethylbenzene (CAS No. 100-41-4) in F344/N Rats and B6C3F$_1$ Mice (inhalation studies). NTP TR 466 NIH Publication No. 99-3956. U.S. Department of Health and Human Services, Public Health Service, National Institute of Health. Available at: http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr466.pdf

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 (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2013. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2013.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2010. 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.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1991. IRIS Summary of Ethylbenzene (CASRN 100-41-4). Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/iris/subst/0051.htm#refinhal

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C13.0 FLUORANTHENE

C13.1 Acute Inhalation Exposure Limits

Table 13-1
Acute Inhalation Exposure Limits for Fluoranthene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014 OEHHA 2009
OMOE	–	–	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

Acute inhalation exposure limits were not available for fluoranthene from the above listed jurisdictions. The search was expanded to include STEL and Ceiling values from the ACGIH (2013) and AEGL-1 values from the US EPA (2013a). No values were identified from these sources.

Due to the lack of available exposure limits, fluoranthene could not be included in the acute inhalation assessment.

C13.2 Chronic Inhalation Exposure Limits

Table 13-2
Chronic Inhalation Exposure Limits for Fluoranthene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2014 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

Chronic inhalation exposure limits were not available for fluoranthene from the above listed sources. The search was expanded to include TLV-TWA from the ACGIH (2013) and PPRTVs from the US EPA (2013b). No chronic values were identified. Due to a lack of available exposure limits, fluoranthene could not be evaluated on its own in the chronic inhalation assessment.

Fluoranthene was evaluated as part of the benzo(a)pyrene TEQ group for carcinogenicity. Please refer to the benzo(a)pyrene toxicological profile for additional information.

C13.3 Oral Exposure Limits

Table 13-3
Chronic Oral Exposure Limits for Fluoranthene

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	–	–	Health Canada 2010, 2004 Health Canada 2013
OEHHA	–	–	OEHHA 2014 OEHHA 2009
RIVM	CR	5	RIVM 2001
US EPA	RfD	40	US EPA 2014, 1993
WHO	–	–	WHO 2014

– = Not available

The US EPA (2014, 1993) has derived a chronic RfD of 40 µg/kg bw/d. Male and female CD-1 (ICR)BR mice were exposed to 0, 125, 250 or 500 mg/kg bw/d via oral gavage for a duration of 13 weeks. Significantly increased absolute and relative liver weights, nephropathy, and SGPT levels were observed in the 250 and 500 mg/kg bw/d groups. Increased food consumption and body weight was recorded in the high-dose group only. In addition, pigmented lesions were observed in the livers of some mice administered 250 and 500 mg/kg bw/d. The study NOAEL was identified as 125 mg/kg bw/d and the LOAEL at 500 mg/kg bw/d. An uncertainty factor of 3,000 was applied to the NOAEL by the US EPA to calculate the RfD, to account for interspecies differences (10), intraspecies variability (10), the use of a subchronic study (10), and lack of adequate data (3). The resulting oral RfD of 40 µg/kg bw/d was used in this assessment to evaluate the non-carcinogenic effects of fluoranthene.

The RIVM (2001) has derived an oral CR of 50 µg/kg bw/d based on the excess lifetime cancer risk of one in 10,000, which is equivalent to an oral CR of 5 µg/kg bw/d based on a one in 100,000 excess lifetime cancer risk. As the key studies involved various routes of exposure (*e.g.*, skin painting, intraperitoneal and subcutaneous injections) the RIVM (2001) value was not used in the assessment.

Fluoranthene was also evaluated as part of the benzo(a)pyrene TEQ carcinogenic assessment. Please refer to the toxicological profile for benzo(a)pyrene for additional information.

C13.4 Summary of Exposure Limits

Table 13-4
Summary of Exposure Limits Selected for Fluoranthene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	–	–	µg/m³		
Chronic	Annual	Inhalation	–	–	µg/m³		
		Multiple Exposure Pathway	RfD	40	µg/kg bw/d	US EPA 2014, 1993	Liver and kidney effects

– = Not available
n/a = Not applicable

C13.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of

Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Fluoranthene). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1993. Fluoranthene (CASRN 206-44-0). Reference Dose for Chronic Oral Exposure (RfD). Available at: http://www.epa.gov/iris/subst/0444.htm

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C14.0 FORMALDEHYDE

C14.1 Acute Inhalation Exposure Limits

Table 14-1
Acute Inhalation Exposure Limits for Formaldehyde

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	2-hour MRL	50	ATSDR 2013, 1999
BC MOE	1-hour Action Level 1-hour Episode Level	60 370	BC MOE 2013
ESRD	1-hour AAQO	65	ESRD 2013
OEHHA	1-hour REL 8-hour REL	55 9	OEHHA 2014, 2008
OMOE	24-hour Standard	65	OMOE 2012
TCEQ	1-hour ReV	50	TCEQ 2013, 2008
US EPA	–	–	US EPA 2014
WHO	30-minute AQG	100	WHO 2000

– = Not available

The ATSDR (2013, 1999) has developed an acute inhalation MRL of 50 µg/m³ (0.04 ppm) for formaldehyde based on a LOAEL of 0.4 ppm for nasal and eye irritation. Occupationally exposed patients with skin hypersensitivity to formaldehyde and unexposed (control) patients, all of whom were non-smokers, were separated into two groups. Group 1 included seven male and three female volunteers with skin hypersensitivity to formaldehyde and Group 2 included 11 healthy males with no history of allergic diseases. Nasal washings were performed in both groups immediately before and after a 2-hour exposure to 0 ppm (placebo) or 0.4 ppm (0.5 mg/m³) formaldehyde and again 4 and 18 hours after the exposure period. In both groups, the placebo did not result in any effects on nasal wash cellular contents or symptom score. Exposure to 0.4 ppm formaldehyde showed statistically significant increased average symptom scores compared with average placebo scores, in both groups. As well, eosinophil counts and albumin levels were elevated in both groups. After 18 hours, symptom scores, eosinophil counts and albumin levels were no longer elevated. A cumulative uncertainty factor of 10 was incorporated by the ATSDR (1999) to account for intraspecies variability (3) and to account for the use of a minimal LOAEL (3). An uncertainty factor of 3 was considered adequately protective of human variability as the symptoms of irritation were observed in a potentially sensitive group of subjects. This 2-hour MRL of 50 µg/m³ based on eye and nasal irritation was conservatively used as the 1-hour exposure limit in the acute effects assessment for formaldehyde as it represents the most conservative value that is supported by adequate documentation.

The TCEQ (2013, 2008) also developed an exposure limit of 50 µg/m³ for formaldehyde based on eye and nose irritation in human volunteers. The TCEQ (2008) derived the acute ReV based on the same study used by the ATSDR (Pazdrak *et al.* 1993) in addition to another study by Krakowiak *et al.* (1998), which also identified a LOAEL of 0.4 ppm. Similar to the ATSDR, the TCEQ (2008) applied a cumulative uncertainty factor of 10 to account for use of a minimal LOAEL (3) and for intraspecies variability (3). A factor of 3 for intraspecies variability was

considered sufficient given that the studies included potentially sensitive subpopulations (TCEQ 2008). The resulting ReV of 50 µg/m³ is the same as the ATSDR MRL.

ESRD (2013) has adopted the TCEQ ESL value of 65 µg/m³ for formaldehyde. As the TCEQ does not provide any supporting documentation for this value and more conservative (*i.e.*, lower), scientifically defensible limits are available, this AAQO was not considered further.

The OEHHA (2014, 2008) derived 1-hour and 8-hour RELs for formaldehyde. The acute 1-hour REL is based on a study involving 19 healthy non-smokers. People were exposed to 0.5 to 3 ppm formaldehyde for a single 3-hour period. A NOAEL of 0.5 ppm and a LOAEL of 1 ppm were determined from the study results for mild-moderate eye irritation. Benchmark dose modelling was conducted, and the $BMCL_{05}$ was determined to be about 0.44 ppm (530 µg/m³). The OEHHA (2008) applied an uncertainty factor of 10 to this value to account for intraspecies differences, resulting in the 1-hour REL of 55 µg/m³. This value was not used as the ATSDR value is slightly lower and is well supported by scientific rationale. The 8-hour REL derived by the OEHHA was based on long-term occupational studies with exposures ranging from 1 to 36 years. As the value is not based on acute exposures, it was not considered further.

The OMOE (2012) provides an acute exposure limit value of 65 µg/m³ as a 24-hour standard based on a health effect. As the OMOE does not provide supporting documentation for the derivation of this acute limit, it was not considered further.

WHO (2000) has established a guideline of 100 µg/m³ based on literature reporting that the lowest concentration associated with nose and throat irritation in humans after short-term exposure is 0.1 mg/m³. WHO recommends this air quality guideline is used as a 30-minute limit to prevent sensory irritation in the general population. This value was not selected as the ATSDR value has a more robust supporting document.

The BC MOE (2013) has derived an Action Level and Episode Level 1-hour ambient air quality objective of 60 and 370 µg/m³ respectively. These values were obtained from provincial air quality objectives. These values were not selected, as the ATSDR value is more conservative.

C14.2 Chronic Inhalation Exposure Limits

Table 14-2
Chronic Inhalation Exposure Limits for Formaldehyde

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	10	ATSDR 2013, 1999
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	RsC	1.9	Health Canada 2004
OEHHA	RsC REL	2 9	OEHHA 2009 OEHHA 2014, 2008
RIVM	–	–	RIVM 2009, 2001
TCEQ	ReV RsC	11 18	TCEQ 2013, 2008
US EPA	RsC	0.8	US EPA 2014, 1991
WHO	–	–	WHO 2000

– = Not available

The US EPA (2014, 1991) has derived an inhalation RsC of 0.8 µg/m³ based on an inhalation study by Kerns *et al.* (1983) that examined the incidence of nasal squamous cell carcinomas in rats exposed to formaldehyde. In the Kerns *et al.* (1983) study, Fischer 344 rats and B6C3F$_1$ mice were exposed to 0, 2, 5.6 or 14.3 ppm (equivalent to 0, 2.5, 7 or 17.6 mg/m³) for 6 hours/day, 5 days/week for a duration of 24 months. Five animals were sacrificed in each exposure group at 6 and 12 months, while 20 were sacrificed in each exposure group at 18 months (Kerns *et al.* 1983). Squamous cell carcinomas and polyploidy adenomas were seen in the nasal cavities male and female rats exposed to 14.3 ppm, and in male animals (polyploidy adenoma only) at 5.6 ppm. In the 5.6 ppm group, only one rat of each sex presented nasal carcinomas. In exposed mice, squamous cell carcinomas were seen in two males at 14.3 ppm. No significant lesions were observed. Using the linearized multistage procedure with additional risk the US EPA (1991) developed an inhalation unit risk of 1.3 × 10-5 $(\mu g/m^3)^{-1}$, which equates to an RsC of 0.8 µg/m³ (associated with a one in 100,000 excess cancer risk). This value was used in the carcinogenic chronic effects assessment for formaldehyde. The US EPA 1991 RSC is currently under review, but still represents the current US EPA value for formaldehyde carcinogenicity. It is acknowledged that the US EPA has produced at draft re-assessment of formaldehyde and has proposed RSC values, the methodologies used by the EPA are currently under peer-review and there is much debate over mechanisms of toxicity and target tissues (NAS 2011). At the time of the finalization of this HHRA, final formaldehyde values

The OEHHA (2009) presents an inhalation unit risk estimate of 6.0E-06 $(\mu g/m^3)^{-1}$ (equivalent to an RsC of 2 µg/m³). This value was derived based on the Kerns *et al.* (1983) study and the US EPA RsC described above.

Health Canada (2004) presents a tumorigenic concentration (TC_{05}) for formaldehyde of 9.5 mg/m³ (Government of Canada 2001). This TC_{05} represents the total intake associated with a 5% increase in incidence of nasal squamous tumours in rats exposed to formaldehyde for up to 24 months (Monticello *et al.* 1996). The TC_{05} corresponds to an RsC of 1.9 µg/m³ that is

associated with an increased cancer risk of one in 100,000. This value was not used in the chronic inhalation assessment, as the TCEQ specifically accounts for mechanistic data and considers the overall weight of evidence in the derivation of its chronic limit for formaldehyde.

The TCEQ (2013, 2008) has derived a cancer-based exposure limit for formaldehyde of 18 µg/m³ derived from a comprehensive analysis of three rodent tumourigenicity data sets by Schlosser *et al.* (2003). One of the three data sets was the Kerns *et al.* (1983) study on which the US EPA RsC is based. The pooled data modelled included 482 rats exposed to 0.7, 2.0, 6.0, 10.0 or 15.0 ppm and 122 controls. $BMCL_{01}$ values were calculated for the various modelling approaches and endpoints (tumours, cell proliferation). Schlosser *et al.* (2003) conducted benchmark dose analysis of the data, and also applied computational flux modelling to account for differences in nasal dosimetry and a pharmacokinetic model to predict DNA cross-link formation. The dose-response relationship in the data for tumour incidence and cell proliferation were both highly non-linear. The TCEQ selected the 95% $BMCL_{01}$ of 0.44 ppm based on cell proliferation as the point of departure for the derivation of a cancer-based ReV, as it represented the most conservative value derived from biologically-based modelling approaches. An uncertainty factor of 30 was applied to the $BMCL_{01}$ to account for intraspecies variability (10), and interspecies differences (3), due to the use of a pharmacokinetic-based biological model in the derivation of the $BMCL_{01}$.

The TCEQ (2013, 2008) has derived a non-carcinogenic chronic ReV of 11 µg/m³ based on the incidence of eye, nasal and respiratory irritation in exposed workers. In an occupational study by Wilhelmsson and Holmstrom (1992), workers were exposed to a mean formaldehyde concentration of 0.21 ppm (0.26 mg/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 0.07 ppm (0.09 mg/m³). Both groups of workers included atopic individuals with Type I hypersensitivity that were responsive 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 reference group. The study LOAEL was identified as 0.26 mg/m³ and the NOAEL as 0.09 mg/m³. Three other human studies were examined as supporting evidence for the Wilhelmsson and Holmstrom (1992) study, with similar LOAEL and NOAEL values reported. The TCEQ adjusted the NOAEL Of 0.09 mg/m³ for continuous exposure (10/20 m³/day × 5/7 days) to a $NOAEL_{HEC}$ of 0.032 mg/m³. An uncertainty factor of 3 was applied to account for intraspecies variability, given that the study included some sensitive individuals. This value was selected for use in the non-cancer assessment of formaldehyde.

The ATSDR (2013, 1999) derived a chronic MRL of 10 µg/m³. This value is based on histological changes in nasal mucosa in occupationally exposed workers (n = 70) in a formaldehyde and formaldehyde resins producing chemical plant; furniture factory workers (n = 100) who were exposed to particle boards and glue components; and a control group of non-exposed office workers (n = 36) (Holmstrom *et al.* 1989). Average employment duration time for the two groups were 10.4 years (range 1 to 36 years) for the chemical workers, and 9.0 years (range 1 to 30 years) for furniture workers. Air concentration estimates of workers' breathing zones were determined to be 0.04 to 0.4 ppm formaldehyde (median 0.24 ± 0.13 ppm) for the chemical workers, and from 0.16 to 0.4 ppm (median 0.20 ± 0.04 ppm) for the furniture workers. Nasal mucosal specimens were taken from the workers from the middle turbinate. A significant difference in the mean histological scores for the chemical workers but not for the furniture workers was observed relative to controls. Histological abnormalities observed in samples from exposed workers included: epithelial dysplasia, cilia loss, goblet cell hyperplasia, cuboidal and squamous cell metaplasia. In addition, exposed workers reported

mild eye irritation in the 0.04 to 0.4 ppm (mean 0.24 ppm) range of exposures. The study LOAEL was determined by the ATSDR to be 0.24 ppm. Although the workers were only exposed 8/24 hours/day, 5/7 days a week, adjustments for continuous exposure were not made by the ATSDR based on the rationale that the effects of formaldehyde exposure are more related to concentration than to duration. A total uncertainty factor of 30 was applied for use of a LOAEL (3), and for intraspecies variation (10). This value is similar to the TCEQ value (described above) in both magnitude and toxicological basis. The TCEQ value was selected on the basis of the benchmark dose and inhalation modelling methods used as part of the derivation, as more consideration is given to dosimetry and the dose response-relationship in the supporting documentation than the ATSDR value.

The OEHHA (2014, 2008) chronic REL of 9 µg/m³ is based on the same study as the TCEQ chronic ReV (Wilhelmsson and Holmstrom 1992). The same NOAEL (0.09 mg/m³) was identified by the OEHHA as the TCEQ. However, no adjustment was made for continuous exposure, although the rationale for not doing this is not clear. An uncertainty factor of 10 was applied to the NOAEL to account for intraspecies variability, resulting in the REL of 9 µg/m³. The TCEQ value was selected for the non-cancer assessment on the basis of the benchmark dose and inhalation modelling conducted as part of the derivation, as more consideration is given to dosimetry and the dose response-relationship in the supporting documentation than the OEHHA value.

C14.3 Oral Exposure Limits

Table 14-3
Chronic Oral Exposure Limits for Formaldehyde

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	MRL	200	ATSDR 2013
Health Canada	–	–	Health Canada 2010,
	TDI	150	Health Canada 2013, 2003
OEHHA	RsD	0.48	OEHHA 2009
	–	–	OEHHA 2014, 2008
RIVM	–	–	RIVM 2009, 2001
US EPA	RfD	200	US EPA 2014, 1990
WHO	TDI	55	WHO 2014, 2005

– = Not available

ATSDR (2013, 1999) and the US EPA (2014, 1990) both derived chronic oral exposure limits of 200 µg/kg/day for formaldehyde based on the same study by Til *et al.* (1989). Male and female Wistar rats were administered formaldehyde in drinking water at mean doses of 0, 1.1, 15 or 82 mg/kg/day (males) and 0, 1.8, 21 or 109 mg/kg/day (females) for a duration of 104 weeks (2 years). About 10 rats/sex/dose were sacrificed and evaluated after 12 to 18 months of exposure, and the remaining rats were evaluated at 24 months. Statistically significant urinary symptoms were observed in high dose animals, including decreased urine production, increased mean urine pH, and the presence of occult blood in urine. Increased urinary pH was also observed in males at 1.1 and 15 mg/kg/day, and the presence of occult blood was observed for all males exposed to 1.1 and 15 mg/kg and in females at 21 mg/kg (in addition to the high-dose animals of both sexes). Significant decreases in plasma alkaline phosphatase activity and total plasma protein were observed at 15 and 82 mg/kg in males and 21 and

109 mg/kg and females. Decreased total plasma protein and increased plasma urea was observed in males at 82 mg/kg. Increased cholesterol was reported in males exposed to 15 mg/kg and 82 mg/kg, and plasma potassium was elevated in high dose females. However, all of these clinical chemical observations were made during the study but were not apparent at the end of the 104-week exposure period. Reduced body weights were observed in males at week 1, and in females from week 24 through the rest of the exposure period. Absolute heart, liver, testes and kidney weights were all significantly decreased in males at 82 mg/kg. Increased relative kidney weights were significantly increased at 109 mg/kg after 53 weeks. Increased relative brain weights were observed in males at 82 mg/kg and females at 109 mg/kg after the first 53 weeks.

In male and female high-dose rats, significant histopathological changes in the gastrointestinal tract were observed after 52 weeks of exposure, including irregular mucosal thickenings in the fore stomach or glandular stomach, increased papillary epithelial hyperplasia, hyperkeratosis, focal ulceration, irregular cellular formations, and mucosal evidence of gastric inflammation. Necrotic changes in the kidneys of high-dose males and females also were observed, namely renal papillary necrosis, and scattered necrosis throughout other nephronic structures. Statistical significance for the observed chronic nephropathy was only observed in low dose males and females, but not the higher doses. Til *et al.* (1989) identified a NOAEL of 15 mg/kg in males and 21 mg/kg in females. The lower NOAEL of 15 mg/kg/day was selected by both the ATSDR and US EPA based on reduced body weights, and histopathological changes of the gastrointestinal tract and kidneys. Both agencies applied an uncertainty factor of 100 to account for interspecies differences (10) and intraspecies variability (10).

The Health Canada Drinking Water Quality Bureau (Health Canada 2013, 2003) derived an oral TDI of 150 µg/kg/day, also based on the Til *et al.* (1989) study (described above for the ATSDR and US EPA values). A NOAEL of 15 mg/kg/day was identified for pathological chances in the stomach and renal papillary necrosis in male rats. An uncertainty factor of 100 was applied to the NOAEL account for interspecies differences (10) and intraspecies variability (10). The Health Canada TDI is essentially the same as the ATSDR value – numerical rounding appears to be the only difference. As this value represents the more conservative of the two values (ATSDR/US EPA and Health Canada), the Health Canada TDI of 150 µg/kg/day based on kidney effects and changes to the gastrointestinal tract was selected for use in the chronic oral assessment.

The WHO (2013, 2005) presents a TDI of 55 µg/kg/day, also based on the data from Til *et al.* (1989). WHO (2005) cites a TDI derived by the WHO International Programme on Chemical Safety of 2.6 mg/mL formaldehyde. This value was based on a NOAEL of 260 mg/L for histopathological changes in the oral and gastric mucosa in rats, cited as being from the Til *et al.* (1989) study. The WHO IPCS applied an uncertainty factor of 100 to this concentration for inter- and intraspecies differences, resulting in a TDI of 2.6 mg/L. Assuming a drinking water ingestion rate of 1.5 L/day and a body weight of 70.7 kg, this value is equivalent to a TDI of 55 µg/kg/day. Based on the review of the Til *et al.* (1989) study and the WHO IPCS (2002) document, and the clear presentation within Til *et al.* (1989) of a NOAEL of 15 mg/kg for histological changes, it is not clear how WHO IPCS identified the NOAEL concentration of 260 mg/L (which, using a standard body weight of 70.7 and a drinking water consumption rate of 1.5 L/day, is equivalent to about 5.5 mg/day). Due to this uncertainty, and the level of detail presented within the Til *et al.* (1989) study, the WHO TDI was not used in the assessment.

The OEHHA (2009) has derived an oral slope factor of 0.021 $(mg/kg\ bw/d)^{-1}$ that equates to an RsD of 0.48 µg/kg bw/d (associated with an increased cancer risk of one in 100,000). This oral

slope factor is based on the same key study (Kerns *et al.* 1983) that was used in the US EPA's RsC derivation for formaldehyde (discussed above). The cancer risk estimate was developed for formaldehyde by the OEHHA (1992) by using two models (PBPK and linearized multistage) to derive upper confidence limits (UCLs) for excess cancer risk. Given that this oral slope factor was derived from inhalation exposure data associated with a local adverse health effect (nasal squamous cell carcinomas), the resulting RsD was not used in the chronic effects assessment for formaldehyde.

C14.4 Summary of Exposure Limits

Table 14-4
Summary of Exposure Limits Selected for Formaldehyde

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	MRL	50	µg/m³	ATSDR 2013, 1999	Eye and nasal irritation
Chronic	Annual	Inhalation	RsC	0.8	µg/m³	US EPA 2014, 1991	Nasal tumours
			RfC	11	µg/m³	TCEQ 2013, 2008	Eye, nasal, respiratory irritation
		Multiple Exposure Pathway	RfD	150	µg/kg/day	Health Canada 2013, 2003	Kidney effects and gastrointestinal effects

C14.5 References

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

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Government of Canada. 2001. Priority Substances List Assessment Report. Formaldehyde. ISBN 0-662-29447-5 Cat. No. En40-215/61E. Environment Canada, Health Canada, Canadian Environmental Protection Act, 1999.

Health Canada. 2003. Canadian Drinking Water Guidelines. Technical Supporting Document – Formaldehyde. Health Canada Drinking Water Quality Bureau.

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

Holmstrom, M., B. Wilhelmsson, H. Hellquist, *et al.* 1989. Histological changes in the nasal mucosa in persons occupationally exposed to formaldehyde alone and in combination with wood dust. Acta Otolaryngol (Stockh) 107:120-129. Cited in: ATSDR 1999.

Kerns, W.D., K.L. Pavkov, D.J. Donofrio, E.J. Gralla and J.A. Swenberg, J.A. 1983. Carcinogenicity of formaldehyde in rats and mice after long-term inhalation exposure. Cancer Res. 43: 4382-4392.

Krakowiak, A., P. Gorski, K. Pazdrak, *et al.* 1998. Airway response to formaldehyde inhalation in asthmatic subjects with suspected respiratory formaldehyde sensitization. American Journal of Industrial Medicine 33: 274-281. Cited in: TCEQ 2008.

Monticello, T.M., J.A. Swenberg, E.A. Gross, R.J. Leininger, J.S. Kimbell, S. Seilkop, T.B. Starr, J.E. Gibson and K.T. Morgan, K.T. 1996. Correlation of regional and nonlinear formaldehyde-induced nasal cancer with proliferating populations of cells. Cancer Research. 56:1012-1022.

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. Technical Support Document for Noncancer RELs Formaldehyde Reference Exposure Levels. December 2008 California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. December 2008. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=128

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Pazdrak, K., P. Gorski, A. Krakowiak and U. Ruta. 1993. Changes in nasal lavage fluid due to formaldehyde inhalation. Int Arch Occup Environ Health 64:515-519.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

Schlosser, P.M., P.D. Lilly, R.B. Conolly, D.B. Janszen and J.S. Kimbell. 2003. Benchmark dose risk assessment for formaldehyde using airflow modeling and a single-compartment, DNA-protein cross-link dosimetry model to estimate human equivalent doses. Risk Analysis 23: 473-487.

TCEQ (Texas Commission on Environmental Quality). 2008. Formaldehyde (CAS Registry Number: 50-00-0). Developmental Support Document, Final, August 2008. Prepared by: Haney, Joseph T. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

Til, H.P., R.A. Woutersen, V.J. Feron, V.H.M. Hollanders and H.E. Falke. 1989. Two-year drinking-water study of formaldehyde in rats. Fd Chem. Toxic 27(2):77-87.

US EPA (United States Environmental Protection Agency). 1990. IRIS Summary of Formaldehyde (CASRN 50-00-0) Reference Dose for Chronic Oral Exposure (RfD). Available at: http://www.epa.gov/ncea/iris/subst/0419.htm#reforal

US EPA (United States Environmental Protection Agency). 1991. IRIS Summary of Formaldehyde (CASRN 50-00-0) Carcinogenicity Assessment for Lifetime Exposure. Available at: http://www.epa.gov/ncea/iris/subst/0419.htm#carc

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2005. Formaldehyde in Drinking Water. Background Document for Development of WHO Guidelines for Drinking Water Quality. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/formaldehyde130605.pdf

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

WHO IPCS (World Health Organization and International Programme on Chemical Safety). 2002. Concise International Chemical Assessment Document. 40. Formaldehyde. World Health Organization, Geneva, Switzerland.

Wilhelmsson, B. and M. Holmstrom. 1992. Possible mechanisms of formaldehyde-induced discomfort in the upper airways. Scandinavian Journal of Work & Environmental Health 18 403-407. Cited in TCEQ 2008.

C15.0 HEXANE

C15.1 Acute Inhalation Exposure Limits

Table 15-1
Acute Inhalation Exposure Limits for Hexane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO 24-hour AAQO	21,000 7,000	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour standard	7,500	OMOE 2012, 2005
TCEQ	–	–	TCEQ 2013
US EPA			US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OMOE (2012, 2005) provides a 24-hour standard of 7,500 µg/m³ for n-hexane and n-hexane isomers. This standard was developed from a LOAEL of 58 ppm (204 mg/m³) for polyneuropathy in humans (Sanagi *et al.* 1980). Workers were exposed to a low concentration of n-hexane and acetone in a tungsten carbide alloys facility for an average of 6.2 years. Significant decreases in mean motor nerve conduction velocities and slowed residual latency of motor conduction of lower extremities were observed. This value was not given further consideration, as it is based on chronic exposure, which is not relevant to the acute effects assessment.

The ESRD (2013) adopted the chronic OEHHA value of 7,000 µg/m³ for n-hexane as a 24-hour AAQO, then derived a 1-hour AAQO of 21,000 µg/m³ from this 24-hour objective. The OEHHA based its chronic REL of 7,000 µg/m³ on a NOAEL of 100 ppm for nervous system effects in mice (ESRD 2013). However, as this value is based on chronic exposure data, it was not used in the acute assessment.

Acute guidelines for hexane have not been established by any other regulatory agencies listed above. Therefore, the search was expanded to include short-term occupational limit values (*i.e.*, STEL and Ceiling) developed by the ACGIH (2013), as well as AEGLs-1, (2013b) developed by the US EPA. However, defensible, acute exposure limits for hexane were not available from these sources, and for this reason hexane was not evaluated individually on an acute basis. Due its structural characteristics, hexane was evaluated as a component of the aliphatic C_5-C_8 group. Please refer to the toxicity profile for the aliphatic C_5-C_8 group for details.

C15.2 Chronic Inhalation Exposure Limits

Table 15-2
Chronic Inhalation Exposure Limits for Hexane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	2,100	ATSDR 2013, 1999
BC MOE	–	–	BC MOE 2013
BC MOE[1]	–	700	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	700	Health Canada 2010
OEHHA	REL	7,000	OEHHA 2014, 2000
OMOE	–	–	OMOE 2012
RIVM	–	–	RIVM 2001
TCEQ	ReV	670	TCEQ 2013, 2007
US EPA	RfC	700	US EPA 2014, 2005
WHO	–	–	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

The TCEQ (2013, 2007) has derived a chronic ReV of 670 µg/m³ based on human occupational data. In the key study by Chang *et al.* (1993), a group of workers in a printing factory were evaluated for potential neurological effects. Workers were exposed to hexane concentrations ranging from 80 to 210 ppm, with an average exposure concentration of 132 ppm. Workers were exposed for 12 hours/day, 6 days/week for a mean duration of 2.6 years. Approximately 40% of the workers evaluated demonstrated subclinical neuropathy. In addition, reduced sensory and action potentials, motor nerve conduction velocity and increased distal latency were reported for exposed workers. The average concentration of 132 ppm was identified as a LOAEL by the TCEQ. This LOAEL was adjusted by the TCEQ to account for continuous exposure (10/20 m³/day × 6/7 days), resulting in a $LOAEL_{HEC}$ of 57 ppm. An uncertainty factor of 300 was applied to account for the use of a LOAEL (10), intraspecies variability (10) and database uncertainties (3). The resulting ReV of 670 µg/m³ based on neurological effects was selected for use in the chronic assessment of hexane.

The US EPA (2014, 2005) developed a chronic RfC of 700 µg/m³ for neurotoxicity. This RfC is based on a benchmark concentration level (BMCL) of 430 mg/m³ for peripheral neuropathy (decreased mean conduction velocity at 12 weeks) in a rat subchronic inhalation study (Huang *et al.* 1989). Male Wistar rats were exposed to 0, 500, 1,200, or 3,000 ppm (equivalent to 0, 1,762, 4,230 or 10,574 mg/m³) of n-hexane for 12 hours/day, 7 days/week for a duration of 16 weeks. Statistically significant decreases in weight gain, and mean conduction velocity accompanied by neural demyelination and remyelination were observed in the middle and high dose groups. A study NOAEL of 50 ppm (1,762 mg/m³) was identified by the US EPA (2005). The incidence of decreased mean conduction velocity was selected as the endpoint of interest, and benchmark dose modeling was conducted. From the modeling, a BMC of 550 mg/m³ and a BMCL of 430 mg/m³ were identified. The BMCL was adjusted from intermittent to continuous exposure (12/24 hours) to a concentration of 215 mg/m³. The blood:gas (air) partition coefficient ($H_{b/g}$) value for n-hexane in humans is 0.8, whereas a value of 2.29 has been

reported in rats (US EPA 2005). The $BMCL_{HEC}$ is equal to 215 mg/m³. The US EPA (2005) applied an uncertainty factor of 300 to the $BMCL_{HEC}$ to account for interspecies variability (3), intraspecies variability (10), extrapolation to chronic exposure from data in a less-than lifetime study (3) and database deficiencies (3, due to the limited reproductive and developmental information available for n-hexane).

Health Canada also provides an acute TC (provisional) of 700 µg/m³, which was adopted from the US EPA and is based on the study by Huang *et al.* (1989) described above. As the Huang *et al.* (1989) study is based on rodent data, the limit of 700 µg/m³ was not selected for use in the assessment as a human-based value is available.

The ATSDR (2013, 1999) derived a chronic MRL of 2,100 µg/m³ (0.6 ppm) based on the incidence of neurological effects in exposed workers. A group of 14 exposed workers were compared with age-matched unexposed workers. The 8-hour time-weighted average exposure concentration of n-hexane was determined to be about 58 ppm (204,000 µg/m³). Workers also were co-exposed to acetone. Exposure durations were found to range from 1 to 12 years, with the average duration being about 6.2 years. A significant trend in decreased muscle strength was observed in exposed workers. Significantly decreased nerve conduction velocities and increased residual latency of motor nerve conduction were observed in exposed workers. The LOAEL was determined to be 58 ppm. No adjustment for continuous exposure was made, as the ATSDR states that steady-state concentrations of n-hexane in blood are reached after 100 minutes of exposure. An uncertainty factor of 100 was applied to the LOAEL to account for the use of a LOAEL instead of a NOAEL (10), and intraspecies variability (10). This value was not selected, as the influence that acetone co-exposure may have had on the exposed workers is not clear.

The OEHHA (2014, 2000) established a chronic REL of 7,000 µg/m³ based on peripheral neuropathy in mice. In the key study, male SM-A mice were exposed to 0, 100, 250, 500, 1,000 or 2,000 ppm commercial hexane (approximately 67.5% n-hexane) continuously, 6 days/week for a duration of 1 year. A significant, dose-related increase in muscle neurophysiology and dose-related abnormalities in posture and muscle atrophy were observed at concentrations 250 ppm and above. The study NOAEL was identified as 100 ppm for commercial hexane, and 68 ppm for n-hexane (based on the mixture containing about 67.5% n-hexane, and the exposure frequency of 6 days/week). An uncertainty factor of 30 was applied to this value to account for interspecies differences (3), and intraspecies variability (10). This value was not used in the chronic assessment, as human-based values are available.

The BC MOE (2014) has derived a vapour standard of 700 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, this value was not selected for use, as supporting documentation was not available.

C15.3 Oral Exposure Limits

n-Hexane was not incorporated into the multiple pathway exposure assessment because it did not exceed the physical-chemical criteria to be defined as a non-volatile chemical. Thus, a chronic oral exposure limit was not required for n-hexane.

C15.4 Summary of Exposure Limits

Table 15-3
Summary of Exposure Limits Selected for Hexane

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	–	–	–	–	–
Chronic	Annual	Inhalation	RsC	–	–	–	–
Chronic	Annual	Inhalation	ReV	670	µg/m³	TCEQ 2013, 2007	Neurological effects
		Multiple Exposure Pathway	n/a	n/a	n/a	n/a	n/a

– = not available
n/a = not applicable

C15.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for n-Hexane. July 1999. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available at: http://www.atsdr.cdc.gov/toxprofiles/tp113.html

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Chang, C.M., C.W. Yu, K.Y. Fong, S.Y. Leung, T.W. Tsin, Y.L. Yu, T.F. Cheung and S.Y. Chan. 1993. N-hexane neuropathy in offset printers. J Neurol Neurosurg Psychiatry 56(5): 538-542.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2010. 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. September 2010.

Huang, J; K. Kato, E. Shibata, K. Sugimura, N. Hisanaga, Y. Ono and Y. Takeuchi. 1989. Effects of chronic n-hexane exposure on nervous system-specific and muscle-specific proteins. Arch Toxicol 63:381-385.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Determination of Noncancer Chronic Reference Exposure Levels. Chronic Toxicity Summary n-Hexane. April 2000. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=292

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2005. Ontario Air Standards for n-Hexane. Standards Development Branch, Ontario Ministry of the Environment. June 2005.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

Sanagi, S., Y. Seki, K. Sugimoto and M. Hirata. 1980. Peripheral nervous system functions of workers exposed to n-hexane at a low level. Int Arch Occup Environ Health 47:69-79.

TCEQ (Texas Commission on Environmental Quality). 2007. n-Hexane (CAS Registry Number: 100-54-3). Developmental Support Document, Final, October 2007. Prepared by: Kadlubar, Bernard J. Toxicology Section, Chief Engineer's Office, Texas Commission on Environmental Quality: Austin Texas.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2005. Toxicological Review of n-Hexane (CAS No. 110-54-3) In Support of Summary Information on the IRIS. July 2005. US EPA Washington DC. Available at: http://www.epa.gov/ncea/iris/toxreviews/0486tr.pdf

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C16.0 HYDROGEN SULPHIDE

C16.1 Acute Inhalation Exposure Limits

Table 16-1
Acute Inhalation Exposure Limits for Hydrogen Sulphide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	1-hour MRL	98	ATSDR 2013, 2006
BC MOE	1-hour	7 (Level A) 28 (Level B)	BC MOE 2013
	24-hour	3 (Level A) 6 (Level B)	
ESRD	1-hour AAQO 24-hour AAQO	14 4	ESRD 2013
OEHHA	1-hour REL	42	OEHHA 2014, 2008
OMOE	24-hour standard	7	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	24-hour AQG	150	WHO 2000

– = Not available

The ATSDR (2013, 2006) derived an acute inhalation MRL for hydrogen sulphide of 0.07 ppm (98 µg/m³). This MRL is based on a LOAEL of 2 ppm for changes in airway resistance and specific airway conductance in excess of 30% in two of the 10 individuals examined. The test subjects all had bronchial asthma requiring medication for 1 to 13 years, but none of the subjects had severe asthma. The subjects were exposed for a half-hour and their respiratory function in response to a histamine challenge was assessed prior to and following exposure. Although the two subjects showed changes in airway resistance and specific airway conductance after exposure to 2 ppm hydrogen sulphide, no statistically significant alterations in lung function were observed at this concentration. The ATSDR (2006) applied a combined uncertainty factor of 30 to account for intraspecies variability (3), use of a minimal LOAEL (3) and the lack of studies in children (3). The acute MRL of 98 µg/m³ based on respiratory irritation was used as a 1-hour exposure limit in the acute assessment of hydrogen sulphide.

The BC MOE (2013) has derived Level A and B Pollution Control Objectives for the forest industry for total reduced sulphur compounds, measured as hydrogen sulphide. Due to the specific nature of the industry cited (forestry), and the lack of a supporting scientific document, these values were not used in the assessment.

ESRD (2013) provides 1-hour and 24-hour AAQOs for hydrogen sulphide of 14 µg/m³ and 4 µg/m³, respectively. These guidelines are odour-based rather than health-based and thus were not used in the acute assessment for hydrogen sulphide.

The OMOE (2012) provides a 24-hour standard of 7 µg/m³ for hydrogen sulphide based on the US EPA chronic RfC of 2 µg/m³. As the OMOE value is based on chronic data, it was not used in the assessment.

The OEHHA (2014, 2008) derived an acute REL of 42 µg/m³ based on physiological responses to odour, including headache and nausea. Sixteen individuals were exposed to increasing concentrations of hydrogen sulphide until their odour threshold was reached. The LOAEL was based on the range of odour thresholds of 0.012 to 0.069 ppm that was identified among the individuals. The geometric mean of the odour thresholds (0.03 ppm) was used to develop the acute REL (OEHHA 2008). An uncertainty factor of 1 was applied to the geometric mean, resulting in an acute REL of 0.03 ppm (42 µg/m³) (OEHHA 2008). It is possible that the symptoms were not the result of direct systemic toxicity, but rather physiological responses triggered by the foul smell of the gas. As a result, the OEHHA acute REL for hydrogen sulphide was not used in the acute assessment.

The WHO (2000) has developed a 24-hour guideline based on eye irritation. However, details regarding the study on which this value is based are not provided. As a result, this value was not used in the assessment.

C16.2 Chronic Inhalation Exposure Limits

Table 16-2
Chronic Inhalation Exposure Limits for Hydrogen Sulphide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	REL –	10 –	OEHHA 2014, 2000 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	RfC	2	US EPA 2014, 2003
WHO	–	–	WHO 2000

– = Not available

The US EPA (2014, 2003) developed an RfC of 2 µg/m³ based on the incidence of nasal lesions of the olfactory mucosa reported in a rat inhalation study by Brenneman *et al.* (2000). Male CD rats were exposed to 0, 10, 30, or 80 ppm (0, 13.9, 42, or 111 mg/m³) of hydrogen sulphide for 6 hours/day, 7 days/week for a duration of 10 weeks. A NOAEL of 10 ppm (13.9 mg/m³) was identified for olfactory loss in males. The US EPA (2003) adjusted the NOAEL for intermittent exposure (6/24 hours) to a concentration of 3.48 mg/m³. The $NOAEL_{ADJ}$ was converted to a HEC using the RGDR methodology.

$$RGDR_{ET} = \frac{(VE/SA_{ET})^A}{(VE/SA_{ET})^H}$$

$$RGDR_{ET} = \frac{(0.019\ L/min\ /\ 15\ cm^2)}{(13.8\ L/min\ /\ 200\ cm^2)}$$

Where:

$RGDR_{ET}$ = regional gas dosimetry ratio in the extrathoracic region
VE = minute volume in rats $(VE)_A$ or humans $(VE)_H$
SA_{ET} = extrathoracic surface area in rats $(SA_{ET})_A$ or humans $(SA_{ET})_H$

The $NOAEL_{ADJ}$ was then multiplied by the $RGDR_{ET}$ of 0.18 to yield a $NOAEL_{HEC}$ of 0.64 mg/m³, as follows:

$$NOAEL_{HEC} = NOAEL_{ADJ} \times RGDR_{ET}$$
$$NOAEL_{HEC} = 3.84\ mg/m^3 \times 0.18$$

The US EPA (2003) applied an uncertainty factor of 300 to the NOAELHEC to account for interspecies variability (3), intraspecies variability (10) and subchronic exposure duration (10). An uncertainty factor of 3 was used instead of the default value of 10 for extrapolation from rats to humans because the calculation of an HEC addresses one of the two areas of uncertainty encompassed in an interspecies uncertainty factor (US EPA 2003). The HEC adjustment addresses the pharmacokinetic component of the extrapolation factor, leaving the pharmacodynamic area of uncertainty. The US EPA RfC of 2 µg/m³ based on nasal irritation was selected as the chronic inhalation limit for hydrogen sulphide.

The OEHHA (2014, 2000) also derived a chronic exposure limit for hydrogen sulphide. The chronic REL of 10 µg/m³ is based on a NOAEL of 30.5 ppm (42.5 mg/m³). In the key study, mice were exposed to 0, 10, 30 or 80 ppm (0, 14, 43, or 112 mg/m³) of hydrogen sulphide via inhalation for 6 hours/day, 5 days/week for a duration of 90 days. Weight loss and inflammation of the nasal mucosa were observed in mice exposed to 80 ppm. The study NOAEL was identified as 30 ppm (43 mg/m³). This REL was not used in the chronic effects assessment as the US EPA value is more conservative, and based on more recent data.

C16.3 Oral Exposure Limits

Hydrogen sulphide was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, hydrogen sulphide was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C16.4 Summary of Exposure Limits

Table 16-3
Summary of Exposure Limits Selected for Hydrogen Sulphide

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	MRL	98	µg/m³	ATSDR 2013, 2006	Respiratory irritation
Chronic	Annual	Inhalation	RfC	2	µg/m³	US EPA 2014, 2003	Nasal irritation
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	n/a	n/a

n/a = not applicable.
– = not available

C16.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2006. Toxicological Profile for Hydrogen Sulfide. Atlanta, GA: US Department of Health and Human Services, Public Health Service. July 2006.

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

BC MOE (British Columbia Ministry of the Environment). 2013. B.C. Ambient Air Quality Objectives. http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Brenneman, K.A., James, R.A., Gross, E.A. and Dorman, D.C. 2000. Olfactory neuron loss in adult male CD rats following subchronic inhalation exposure to hydrogen sulphide. Toxicol Pathol 28(2):326-333. Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OEHHA (California Office of Environmental Health Hazard). 2000. Determination of Noncancer Chronic Reference Exposure Levels. Chronic Toxicity Summary. Hydrogen sulfide. April 2000. Available at: http://oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=321

OEHHA (California Office of Environmental Health Hazard). 2008. TSD for Non-cancer RELs Acute Toxicity Summary. Hydrogen sulfide.. June 2008. Available at: http://oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=144

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2003. Toxicological Review of Hydrogen Sulfide (CAS No. 7783-06-4) In Support of Summary Information on the Integrated Risk Information System (IRIS). June 2003. Available at: http://www.epa.gov/ncea/iris/toxreviews/0061tr.pdf

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

C17.0 ISOPROPYLBENZENE (CUMENE)

C17.1 Acute Inhalation Exposure Limits

Table 17-1
Acute Inhalation Exposure Limits for Isopropylbenzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO	500	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Standard	400	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OMOE (2012) has developed a 24-hour air standard of 400 µg/m³. As a supporting document was not available for this value, it was not selected for use in the assessment.

The ESRD (2013) has established an AAQO of 500 µg/m³ for a 1-hour averaging period, which was adopted from a previous Texas Natural Resource Conservation Commission limit. However, no supporting documentation is available.

In the absence of available limits from the agencies listed in the table above, the search was extended to include AEGL-1 values from the US EPA (2013) and short-term values from the ACGIH (2013).

The US EPA (2007) has derived a 1-hour AEGL-1 of 250 mg/m³. This value is based on what appears to be an anecdotal report from an occupational setting (Dow 1948) that was published but has been since withdrawn, according to the US EPA (2007) reference list. As a result, this value was not considered in the acute effects assessment as the supporting information could not be verified.

No acute ceiling or STEL values were available from the ACGIH (2013).

As no defensible acute exposure limits were identified for isopropylbenzene, it was included in the acute inhalation assessment as part of the aromatic C_9-C_{16} group.

C17.2 Chronic Inhalation Exposure Limits

Table 17-2
Chronic Inhalation Exposure Limits for Isopropylbenzene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE(1)	–	400	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TRV	400	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2014 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	RfC	400	US EPA 2014
WHO	–	–	WHO 2000

– = Not available
(1) Based on the agricultural, urban park and residential use vapour standard

The US EPA (2014, 1997) has derived an inhalation RfC of 400 µg/m³ for increased kidney weights in female rats and increased adrenal weights in male and female rats (US EPA 1997). The key study was conducted in two parts. In the first part, male and female F344 rats (21 per sex per group) were exposed to 0, 100, 496 or 1,202 ppm (0, 492, 2,438 or 5,909 mg/m³) of isopropylbenzene vapour for 6 hours/day, 5 days/week for a duration of 13 weeks. In the second part, male and female F344 rats (15 per sex per group) were exposed to 0, 50, 100, 496, or 1,202 ppm (0, 246, 492, 2,438 or 5,909 mg/m³) for 6 hours/day, 5 days/week for a duration of 13 weeks, followed by a 4-week recovery period. Full histopathological analysis was completed on animals from both parts of the study, and some quantitative and morphologic evaluations of spermatogenesis were conducted to evaluate potential reproductive effects. Significant increases in relative and absolute kidney and adrenal gland weights were observed at the highest dose (1,202 ppm or 5,909 mg/m³) in both males and females in the first study. In the second study, increased relative and absolute adrenal weights were reported for females in the highest dose group. Although abnormal renal lesions were observed in male rats in the two highest dose groups, the US EPA determined that the reported observations were consistent with a male rat-specific condition known as alpha-2u-globulin nephropathy, and should not be used in the determination of a point of departure. No significant adverse effects on other organ systems were observed. The US EPA (1997) selected the NOAEL of 2,438 mg/m³ for kidney effects in female rats and adrenal gland effects in both sexes following inhalation exposure to isopropylbenzene for 6 hours/day, 5 days/week for 13 weeks. The NOAEL was adjusted for intermittent exposure (6/24 hours × 5/7 days) to a concentration of 435 mg/m³. A $NOAEL_{HEC}$ was calculated assuming a default value of 1.0 since *b:a* lambda values are unknown for the experimental animal species (a) and humans (h) (US EPA 1997). An uncertainty factor of 1,000 was applied to the $NOAEL_{HEC}$ to account for subchronic-to-chronic extrapolation (10), intraspecies variability (10), interspecies extrapolation (3), and for database deficiencies (3). A 3-fold uncertainty factor for interspecies variability was considered appropriate by the US EPA (1997) as the HEC adjustment addresses the pharmacokinetic component of the extrapolation

factor, leaving the pharmacodynamic area of uncertainty. The RfC of 400 µg/m³ was used in the chronic inhalation effects assessment of isopropylbenzene, as well as in the aromatic C_9-C_{16} group as the limit for the group is more conservative than the RfC for isopropylbenzene.

The Health Canada (2010) inhalation TRV of 400 µg/m³ was based on the US EPA RfC (described above). The same NOAEL was selected by Health Canada, and the same adjustments for continuous exposure and uncertainty were applied as the US EPA.

The BC MOE (2014) has also derived a vapour standard of 400 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, this value was not selected for use, as supporting documentation was not available.

C17.3 Oral Exposure Limits

Isopropylbenzene was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, Isopropylbenzene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C17.4 Summary of Exposure Limits

Table 17-3
Summary of Exposure Limits Selected for Isopropylbenzene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	RfC	n/a	µg/m³	–	–
Chronic	Annual	Inhalation	RfC	400	µg/m³	US EPA 2007	Kidney effects, adrenal gland effects
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	–	–

– = Not available
n/a = Not applicable

C17.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. B.C. Ambient Air Quality Objectives. http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Dow (Dow Chemical Company). 1948. Toxicology and hygiene – isopropylbenzene. July 6, 1948. Study authors were E.M. Adams and V.K. Rowe. Previously released to NTIS/OTS #0206685, but since withdrawn. Provided 9/2004 courtesy of S. Ripple, Dow Corporation. Cited in: US EPA 2007.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Isopropylbenzene). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1997. Cumene (CASRN 98-82-8) Reference Concentration for Chronic Inhalation Exposure (RfC). http://www.epa.gov/ncea/iris/subst/0306.htm

US EPA (United States Environmental Protection Agency). 2007. Interim. Acute Exposure Guideline Levels (AEGLs) for Cumene CAS Reg. No 98-82-8. http://www.epa.gov/opptintr/aegl/pubs/cumene_tsd_interimversion1_8_28_07.pdf

US EPA (United States Environmental Protection Agency). 2013. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

C18.0 NAPHTHALENE

C18.1 Acute Inhalation Exposure Limits

Table 18-1
Acute Inhalation Exposure Limits for Naphthalene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour Guideline	22.5	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OMOE (2012) has developed a guideline for naphthalene of 22.5 µg/m³ based on a 24-hour averaging period. Although the 24-hour value is based on health considerations, the specific basis of its derivation remains unknown as no supporting documentation is available. Therefore this value was not considered for the acute inhalation assessment.

As acute exposure limits were not available from the other agencies listed in the table above, the search was expanded to include AEGL-1 values from the US EPA (2013a) and STELs or ceiling values from ACGIH (2013). However, an acute inhalation exposure limit was only identified from ACGIH.

The ACGIH (2013) recommends a STEL of 15 ppm (79 mg/m³) based on eye irritation as a result of occupational exposure to naphthalene. The STEL equates to a 15-minute air concentration that should not be exceeded at any time during a workday. The 15-minute STEL can be adjusted to an equivalent 1-hour concentration using a modified Haber's Law.

$$C_{ADJ}^{n} \times T_{ADJ} = C^{n} \times T$$

$$C^{1} \times 60 \text{ minutes} = (79 \text{ mg/m}^3)^{1} \times 15 \text{ minutes}$$

Where:

C_{ADJ} = duration-adjusted concentration
T_{ADJ} = desired time of exposure (60 minutes)
C = concentration of exposure (79 mg/m³)
T = time of exposure (15 minutes)
n = chemical-specific modification factor designed to account for the toxicity of a chemical being concentration and/or duration dependent. The OEHHA recommends using a default n value of 1 in the adjustment for less than 1-hour exposure.

Based on the above conversion factor, the STEL was adjusted to a concentration of 20 mg/m³. A cumulative uncertainty factor of 10 was applied to the duration adjusted STEL to account for intraspecies variability (10). The result is a 1-hour exposure limit of 2,000 µg/m³ based on eye irritation.

As this limit was also selected to assess the aromatic C_9-C_{16} group, of which naphthalene is a component, naphthalene was not assessed individually using this limit. Naphthalene was evaluated within the aromatic C_9-C_{16} group in the acute inhalation assessment.

C18.2 Chronic Inhalation Exposure Limits

Table 18-2
Chronic Inhalation Exposure Limits for Naphthalene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	3.7	ATSDR 2013, 2005
BC MOE	–	–	BC MOE 2013
BC MOE[1]	–	3	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	3	Health Canada 2010
OEHHA	REL RsC	9 0.3	OEHHA 2014, 2000 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	RfC	3	US EPA 2014, 1998
WHO	–	–	WHO 2000

– = Not available
[1] Based on the agricultural, urban park and residential use vapour standard

The US EPA (2014, 1998) has derived a chronic inhalation RfC of 3 µg/m³ for naphthalene. This RfC was estimated from a chronic inhalation mouse study that reported a duration-adjusted $LOAEL_{HEC}$ of 9.3 mg/m³ based on hyperplasia and metaplasia in respiratory and olfactory epithelium in the nasal cavity of treated mice (NTP 1992). Male and female $B6C3F_1$ mice were exposed to 0, 10, or 30 ppm for 6 hours/day, 5 days/week for a duration of 104 weeks. No significant increase in tumour incidence was observed in males, but the incidence of pulmonary alveolar/bronchiolar adenomas was increased in females exposed to 30 ppm relative to controls. Non-neoplastic lesions were observed in the nasal passages and lungs of both male and female mice, namely lesions indicative of an inflammatory response. Both males and females in the 10 and 30 ppm groups had exposure-related increases in alveolar histiocyte and lymphocyte infiltration, alveolar hyperplasia, interstitial fibrosis, and in more advanced lesions – granulomatous inflammation. Bronchial submucousal glands were also observed to be distended when the above lesions were present. Mild lesions in the nasal passages of exposed mice were also observed. The US EPA (1998) applied an uncertainty factor of 3,000 to the $LOAEL_{HEC}$ of 9.3 mg/m³ to account for interspecies variability (10), sensitive human individuals in the population (10), extrapolation from a NOAEL to a LOAEL (10), and for database uncertainties (3). Database uncertainties included the lack of a two generation reproductive toxicity study and chronic inhalation data for other animal species.

Naphthalene was assessed individually in the chronic inhalation assessment using the US EPA RfC of 3 µg/m³ based on the appearance of nasal lesions. In addition, naphthalene was evaluated as a component of the aromatic C_9-C_{16} group. Please refer to the toxicity profile for the aromatic C_9-C_{16} group for details.

Health Canada (2010) also provides a TC of 3 µg/m³, as it adopted this value from the US EPA (described above).

The BC MOE (2014) has also derived a vapour standard of 3 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not available.

The OEHHA (2014, 2000) has derived a chronic REL of 9 µg/m³ (0.002 ppm) that also is based on the same NTP (1992) bioassay used by the US EPA. The OEHHA (2000) determined that the study LOAEL was 10 ppm, but note that almost all animals (>96%) exposed to this concentration exhibited some type of an adverse effect, which limits the reliance of this study with respect to being the basis of a health-protective value. The LOAEL of 10 ppm was adjusted for continuous exposure to 1.8 ppm (6/24 hours, 5/7 days). To account for uncertainties, a cumulative UF of 1,000 was applied to the adjusted LOAEL. This factor took into account: the use of a LOAEL (10), interspecies differences (10), and intraspecies variability (10). The US EPA value was selected over the OEHHA value, primarily as it is the more conservative value. This is of importance in light of the high incidence of adverse effects at the lowest dose level in the NTP (1992).

The ATSDR (2013, 2005) chronic MRL of 3.7 µg/m³ (0.0007 ppm) is also based on the NTP study used in the derivation of the US EPA and OEHHA values. As well the ATSDR considers a more recent study in rats (NTP 2000) in which male and female F344 rats were exposed to 0, 10, 30 or 60 ppm for 6 hours/day, 5 days/week for a duration of 105 weeks. A LOAEL of 10 ppm was identified for the incidence of non-cancerous lesions in olfactory epithelium in rats (NTP 2000) and mice (NTP 1992). This LOAEL was adjusted for continuous exposure (6/24 hours × 5/7 days) to 1.8 ppm (or 9,400 µg/m³). This value was further adjusted to a $LOAEL_{HEC}$ of 0.2 ppm (1,000 µg/m³) by multiplying the $LOAEL_{ADJ}$ by an RGDR of 0.132 (calculated by the ATSDR). The $LOAEL_{HEC}$ was divided by an uncertainty factor of 300 to account for the use of a LOAEL (10), interspecies differences (3, due to the calculation of a HEC), and intraspecies variability (10). The resulting MRL of 3.7 was not selected for use as the US EPA value is more conservative.

In addition, the OEHHA (2009) presents a cancer unit risk value of 3.4E-05 $(\mu g/m^3)^{-1}$ (equivalent to an RsC of 0.3 µg/m³). This value is based on the two bioassays by the NTP (1992, 2000) described above. In the NTP (2000) study, increased incidences of respiratory epithelial adenoma and olfactory epithelial blastoma were observed in both male and female rats. A positive dose-response relationship was observed in male rats only for the respiratory epithelial adenomas, and the incidences of these tumours were statistically significant at all exposure concentrations. The incidences of these tumours were not statistically significant or were of marginal significance in females. The olfactory epithelial neuroblastomas were significantly increased in all exposure levels in females, and in the 30 and 60 ppm groups for males. The exposure concentrations were adjusted for continuous exposure (6/24 hours × 5/7 days) and converted to mg/m³. Dose scaling based on body weight and breathing rates was conducted. In addition, pharmacokinetic modelling was conducted for both rats and mice and all modelling runs confirmed that the dose-response relationship was linear. A linearized multistage model

and a benchmark dose model were both applied to the data set, and similar ranges of unit risk values were calculated. The OEHHA (2009) notes that no naphthalene related tumours have been observed in humans. Given that the US EPA and other agencies have not derived cancer-based values, it suggests that the weight of evidence at the current time in support of human carcinogenicity in association with naphthalene exposure is limited. As such, this value was not selected for use in the assessment.

C18.3 Oral Exposure Limits

Table 18-3
Chronic Oral Exposure Limits for Naphthalene

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	TDI	20	Health Canada 2010
	–	–	Health Canada 2013
OEHHA	RsD	0.08	OEHHA 2009
	–	–	OEHHA 2014
RIVM	TDI	40	RIVM 2009, 2001
US EPA	RfD	20	US EPA 2014, 1998
WHO	–	–	WHO 2014

– = Not available

The US EPA (2014, 1998) presents an RfD of 20 µg/kg bw/day for naphthalene. This RfD was derived from a subchronic oral rat study that reported a NOAEL of 100 mg/kg bw/day for decreased mean terminal body weight. Groups of male and female Fisher rats were administered 0, 25, 50, 100, 200, or 400 mg/kg naphthalene via corn oil gavage 5 days/week for a duration of 13 weeks. Food consumption and body weight were examined weekly. Clinical signs of toxicity were examined twice a day. At termination, a suite of hematological parameters were examined and necropsy of all rats was completed. Twenty-seven organs and tissues (including the eyes, lungs, stomach, liver, kidney, reproductive organs, thymus and kidney) from the control group rats and 400 mg/kg group rats were examined histopathologically, as well as the thymuses of the 200 mg/kg group rats.

In the 400 mg/kg group, a greater than 10% increase in numbers of mature neutrophils in male and female rats, and a greater than 10% decrease in numbers of lymphocytes in male rats were revealed during the hematological examination. Low incidences in kidney lesions (focal cortical lymphocytic infiltration or focal tubular regeneration) were reported in the male rats of the 200 mg/kg and 400 mg/kg groups, respectively. However, these lesions appeared to occur inconsistently in relation to dose, and were not observed in females. In female rats, low incidences of lesions in the thymus were observed at the 400 mg/kg dose level. No lesions of the respective control kidneys or thymuses were observed. Although food consumption was not reportedly affected, the mean terminal body weights of the male rats exposed to 200 mg/kg and 400 mg/kg were 12% and 29% depressed relative to the control group, respectively, and 3% depressed in female rats exposed to 400 mg/kg. On this basis, a LOAEL of 200 mg/kg bw/day and a NOAEL of 100 mg/kg bw/d were identified for a greater than 10% decrease in mean terminal body weight in male rats. The US EPA also applied a benchmark dose modelling approach of the study data, however, the US EPA decided not to use the benchmark dose information as it did not reduce uncertainty or provide an advantage in this instance compared

to the NOAEL-LOAEL approach. The US EPA adjusted the NOAEL for continuous exposure (100 mg/kg bw/day × 5/7 days) to a dose of 71 mg/kg bw/day, and applied a cumulative uncertainty factor of 3,000 to the duration-adjusted NOAEL to account for interspecies differences (10), intrahuman variability (10), subchronic to chronic extrapolation (10), and database deficiencies (3), including lack of chronic oral exposure studies and limited reproductive toxicity studies. The result is the RfD of 0.02 mg/kg bw/day (or 20 µg/kg bw/day), which was used as the chronic oral limit for naphthalene in the assessment.

Health Canada (2010) provides a TDI of 20 µg/kg bw/day based on the US EPA analysis discussed above.

RIVM (2001) provides a TDI of 40 µg/kg bw/day based on the TPHCWG RfD of 40 µg/kg bw/day for the aromatic C_9-C_{16} group. This RfD was not used in the assessment as the chronic oral exposure limit provided by the US EPA and Health Canada is more conservative and based on the oral toxicity of naphthalene alone.

The OEHHA (2009) presents an oral slope factor of 1.2E-01 $(mg/kg\ bw/day)^{-1}$ (equivalent to an RsD of 0.08 µg/kg bw/day). However, this value is based on the two chronic inhalation bioassays by the NTP (1992, 2000) described above. Given the limited evidence at this time in support of human carcinogenicity following naphthalene exposure and the uncertainty associated with route-to-route extrapolation for a non-systemic effect such as nasal tumours, this value was not selected for use in the assessment.

C18.4 Summary of Exposure Limits

Table 18-4
Summary of Exposure Limits Selected for Naphthalene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	STEL	2,000	µg/m³	ACGIH 2013	Eye irritation
Chronic	Annual	Inhalation	RfC	3	µg/m³	US EPA 1998	Nasal irritation
		Multiple Exposure Pathway	RfD	20	µg/kg bw/d	Health Canada 2010; US EPA 1998	–

– = Not available

C18.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

ATSDR (Agency for Toxic Substances and Disease Registry). 2005. Toxicological Profile for Naphthalene, 1-Methylnaphthalene, and 2-Methylnaphthalene. August 2005. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available at: http://www.atsdr.cdc.gov/toxprofiles/tp67.html#bookmark08

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

NTP (National Toxicology Program). 1992. Toxicology and carcinogenesis studies of naphthalene in B6C3F$_1$ mice (inhalation studies). Technical Report Series No. 410. NIH Publication No. 92-3141. US Department of Health and Human Services, Public Health Services, National Institute of Health. NTP, Research Triangle Park, NC. Cited In: US EPA 1998.

NTP (National Toxicology Program). 2000. Toxicology and Carcinogenesis Studies of Naphthalene in F344/N Rats (Inhalation Studies), NTP TR500. NIH Publication No. 01-4434.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Determination of Noncancer Chronic Reference Exposure Levels. Chronic Toxicity Summary Naphthalene. April 2000. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=413

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1998. Toxicological Review of Naphthalene (CAS No. 91-20-3) In Support of Summary Information on the IRIS. August 1998. US EPA Washington DC. Available at: http://www.epa.gov/ncea/iris/toxreviews/0436tr.pdf

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C19.0 NITROGEN DIOXIDE (NO_2)

C19.1 Acute Inhalation Exposure Limits

Table 19-1
Acute Inhalation Exposure Limits for NO_2

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	1-hour MAL 1-hour MTL 24-hour MAL 24-hour MTL	400 1,000 2,000 3,000	BC MOE 2013
ESRD	1-hour AAQO	300	ESRD 2013
OEHHA	1-hour REL	470	OEHHA 2014
OMOE	1-hour 24-hour	400 200	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	1-hour Standard	188	US EPA 2014, 2010
WHO	1-hour Standard	200	WHO 2006

– = Not available, MAL: Maximum Acceptable Level, MTL: Maximum Tolerable Level

Although no RfC was available from US EPA (2014), a 1-hour National Air Standard has been derived by the US EPA (2010). This value is based on a 3-year average 98th percentile of the annual distribution of daily maximum 1-hour concentrations. Although it is derived from NO_2 exposure data, it is intended to apply to all NO_x compounds. Experimental evidence from human and animal studies indicates that respiratory effects attributable to NO_2 can occur after brief exposures (*e.g.*, less than 1 hour, up to 3 hours). The US EPA's 2008 Integrated Science Assessment concluded that 1-hour exposures of 100 ppb may result in small, significant increases in airway responsiveness. This is based in part on the observations from human clinical studies where airway inflammation and increased airway responsiveness were observed in asthmatics at concentrations less than 2 ppm. In contrast, airway inflammation has been observed at much higher concentrations (100 to 200 ppm/minute, or 1 ppm for 2 to 3 hours) in healthy individuals. The 1-hour standard of 100 ppb (188 µg/m³) is intended to be protective of sensitive individuals in the population, including asthmatics and individuals with pre-existing respiratory conditions. As this value represents the most recent regulatory review of the health effects of NO_2 and provides the most detailed supporting documentation for its basis, it was selected for use in the assessment.

The WHO (2006) has derived a 1-hour guideline of 200 µg/m³ for NO_2. This value is based upon the increased incidence of adverse respiratory effects in animal and epidemiological studies at concentrations above 200 µg/m³. This value was used in addition to the US EPA 1-hour value.

The BC MOE (2013) has derived MAL and MTL levels for NO_2 on both a 1-hour and 24-hour basis of 400 and 1000 µg/m³, respectively. However, these values are currently under review (Personal Communication, 2014). As a result, the BC MOE values were not selected for use in the assessment.

ESRD (2013) has a 1-hour AAQO for NO_2 of 159 ppb (300 µg/m³) based on respiratory effects. The previous 24-hour AAQO of 200 µg/m³ has been withdrawn by ESRD. However, limited information is provided regarding the rationale of deriving 300 µg/m³ as the 1-hour objective. The Alberta Environment 2007 Assessment Report for NO_2 provides a general overview of the potential health effects associated with NO_2, however, it does not provide information regarding the derivation of the 1-hour value. Although it is noted that healthy individuals may experience adverse effects at NO_2 concentrations greater than 2 ppm, it is also noted that sensitive individuals may respond at lower concentrations. It is not clear what effect threshold or uncertainty factors were selected by ESRD in the derivation of the new 1-hour AAQO of 300 µg/m³. This value was not selected for use in the assessment, due to a lack of available information.

The OEHHA (2014) has derived a 1-hour REL of 470 µg/m³ based upon respiratory effects. The key study upon which this is based is not well described within OEHHA (2014) and the supporting document cited (CARB 1992) is not readily available. As a result, the basis and derivation of this value could not be independently evaluated, and this value was not used in the assessment as a result.

The OMOE (2012) provides 1-hour and 24-hour standards of 400 µg/m³ and 200 µg/m³, respectively, based on health. However, as no supporting documentation for the 1-hour and 24-hour standards are available, these values were not considered for the acute inhalation assessment.

C19.2 Chronic Inhalation Exposure Limits

Table 19-2
Chronic Inhalation Exposure Limits for NO_2

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	Annual MDL	60	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	Annual Standard	45	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	– –	– –	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	Annual Standard	100	US EPA 2014, 2010
WHO	Annual Standard	40	WHO 2006

– = Not available,
MDL: Maximum Desirable Level

The BC MOE (2013) has derived an annual MDL of 60 µg/m³ and an annual MAL of 100 µg/m³. However, these values are currently under review (Personal Communication, 2014). As a result, they were not selected for use in the assessment.

The US EPA (2014) does not have an RfC value available due to the existence of a National Ambient Air Quality Standard (NAAQS). The US EPA (2010) has maintained the AAQS of 53 ppb (100 µg/m³) derived by the US EPA in 1971 (US EPA 2010), which was subsequently upheld in scientific and regulatory reviews between 1971 and 2010. Although the 1971 document is not readily available, the scientific reviews conducted in 1993 and 2010 by the US EPA suggest that the annual standard is associated with the potential for human health effects. A scientific review of the annual air standard conducted in 1993 suggests that the standard of 100 µg/m³ was upheld, based upon the results of a meta-analysis of epidemiological studies conducted in children ages 5 to 12. Within this review, an increase in 0.015 ppm or 28 µg/m³ of NO_2 over an averaging period of 2 weeks was associated with a 20% increase in respiratory symptoms. The NO_2 sources included both indoor and outdoor sources, and average concentrations in the studies were noted to range from 0.008 to 0.065 ppm (US EPA 1993). In 1996, the annual standard was maintained by the US EPA on the basis that, in combination with the short-term standard, the annual standard was protective of both the potential short-term and long-term human health effects of NO_2 exposure (US EPA 1996). The most recent edition of the Final Rule (US EPA 2010) indicates that the annual standard was upheld due to the uncertainty associated with the potential long-term effects of NO_2.

The ESRD 2013 has an annual AAQO of 24 ppb (45 µg/m³) which is based on vegetation effects. The Alberta Environment 2007 Assessment Report provides a general overview of the potential chronic human health and vegetation health effects, but does not provide detailed information regarding exposure concentrations above which adverse effects would be anticipated in humans. As this annual objective was not based on human health effects, this value was not considered for use in the assessment.

The WHO (2006) guideline value of 40 µg/m³ (0.023 ppm) represents an annual value recommended by the WHO International Program on Chemical Safety (IPCS). WHO IPCS (1997) indicates that the 40 µg/m³ is based on consideration of background concentrations and the observation that adverse health impacts may occur when concentrations in addition to background are above 28 µg/m³. As this value is not well substantiated in the available supporting documentation, the US EPA value was used in the chronic assessment.

C19.3 Oral Exposure Limits

Nitrogen dioxide is a gaseous criteria air contaminant which acts on the point of contact once it is inhaled, (*i.e.*, the respiratory system). As such, it was not evaluated in the multiple pathway assessment.

C19.4 Summary of Exposure Limits

Table 19-3
Summary of Exposure Limits Selected for NO_2

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	AAQS	188	µg/m³	US EPA 2010	Respiratory irritant
Chronic	Annual	Inhalation	RfC	100	µg/m³	US EPA 2010	Respiratory irritant
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	n/a	n/a

n/a = Not applicable.

C19.5 References

Alberta Environment. 2007. Assessment Report for Nitrogen Dioxide for Developing Ambient Air Quality Objectives. Prepared by Toxico-Logic Consulting Inc. for Alberta Environment. ISBN No. 978-0-7785-9959-3. Available at: http://environment.gov.ab.ca/info/library/8302.pdf

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

CARB (California Air Resources Board). 1992. Review of the one-hour ambient air quality standard for nitrogen dioxide technical support document. Sacramento: State of California Air Resources Board Technical Support Division; December 1992. Cited in: OEHHA 2008

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Personal Communication. 2014. Glenn Okrainetz, British Columbia Ministry of the Environment (BC MOE) to Bart Koppe, Intrinsik. May 15, 2014.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1993. Air Quality Criteria for Oxides of Nitrogen. EPA 600/8-91/049bF. August 1993.

US EPA (United States Environmental Protection Agency). 1996. 40 CFR Part 50. Vol 61. No. 196. 52852. October 8, 1996. National Ambient Air Quality Standards for Nitrogen Dioxide: Final Decision.

US EPA (United States Environmental Protection Agency). 2008. Integrated Science Assessment for Oxides of Nitrogen - Health Criteria. National Center for Environmental Assessment. EPA/600/R-08/071.

US EPA (United States Environmental Protection Agency). 2010. 40 CFR Parts 50 and 58. Primary National Ambient Air Quality Standards for Nitrogen Dioxide. Final Rule.

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 1997. International Programme on Chemical Safety (IPCS). Environmental Health Criteria 188: Nitrogen Oxides - 2nd ed. ISBN 92 4 157188 8, ISSN 0250-863X. Available at: http://www.inchem.org/documents/ehc/ehc/ehc188.htm

WHO (World Health Organization). 2006. Air Quality Guidelines: Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. ISBN 92 890 2192 6.

C20.0 PARTICULATE MATTER ($PM_{2.5}$)

C20.1 Acute Inhalation Exposure Limits

Table 20-1
Acute Inhalation Exposure Limits for $PM_{2.5}$

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	24-hour AQO (98th percentile)	25	BC MOE 2013
CCME	24-hour	28 (2015) 27 (2020)	CCME 2012
ESRD	1-hour AAQG 24-hour AAQO	80 30	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	–	–	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	24-hour Standard	35	US EPA 2006
WHO	24-hour (99th percentile) Guideline	25	WHO 2006

– = Not available

The BC MOE (2013) has derived a 24-hour Provincial Air Quality Objective of 25 µg/m³ for $PM_{2.5,}$ while the Canadian Ambient Air Quality Standard is 28 µg/m³ (CCME 2012). The BC MOE (2013) presents a guideline of 25 µg/m³ for comparison with the 98th percentile of daily averages. In contrast, The WHO (2006) has also derived a 24-hour value of 25 µg/m³ based on the 99th percentile of daily averages. In the supporting documentation for this value, WHO (2006) notes that the use of a 24-hour value is useful in protecting against excess mortality or morbidity on an episodic basis. The value of 25 µg/m³ was selected for use in the acute inhalation assessment.

Two 24-hour Canadian Ambient Air Quality Standards have been derived by the CCME: 28 µg/m³ (for compliance by 2015) and 27 µg/m³ (for compliance by 2020). These two values are intended to be used with the 3-year average of the annual 98th percentile of daily 24-hour average concentrations. A supporting document is available (CCME 2012). The lower value of these two (27 µg/m³) was not selected for use in the assessment, due to the existence of the more conservative values from the BC MOE and WHO.

ESRD (2013) cites the previous Canada-Wide Standard for PM (CCME 2000) for its 1-hour AAQG and 24-hour AAQO for fine particulate matter, based on the 2nd highest 24-hour value. The 1-hour value is intended for use in monitoring and reporting of the Ambient Air Quality Index, but was not selected for use in the assessment.

The US EPA (2006) presents a 24-hour ambient air quality standard of 35 µg/m³ for $PM_{2.5}$ for primary and secondary particulate, which is intended to be protective of human health effects as well as several environmental and socioeconomic endpoints. As of 2012, this 24-hour standard is maintained (Federal Register 2012), following a comprehensive Integrated Science

Assessment (ISA) on fine particulate matter and adverse human health outcomes (US EPA 2009). This value is based on the 98th percentile of 24-hour concentrations over a 3-year period. As this value is less conservative than some of the other values available, it was not selected for use in the assessment.

C20.2 Chronic Inhalation Exposure Limits

Table 20-2
Chronic Inhalation Exposure Limits for $PM_{2.5}$

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	Annual Objective Annual Planning Goal	8 6	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	Annual Standard	– – 12	OEHHA 2009 OEHHA 2014 CARB 2005, 2002
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	Annual Standard	12	US EPA 2012
WHO	Annual Guideline	10	WHO 2006

– = Not available

The BC MOE (2013) has derived an annual AAQO of 8 µg/m³ based on the protection of human health. The BC MOE (2008) notes that several scientific studies have suggested that mortality rates are more closely associated with long-term exposures than with short-term, episodic peak exposures. The goal of the annual AAQO is to reduce long-term exposure potential for people living in BC. This AAQO of 8 µg/m³ was selected for use in the assessment, as it represents the most conservative value.

In 1997, the US EPA first set National Ambient Air Quality Standards (NAAQS) for fine particles. Two primary $PM_{2.5}$ standards were set: an annual standard of 15 µg/m³ to protect against health effects caused by exposures ranging from days to years and a 24-hour standard to provide additional protection on days with high peak $PM_{2.5}$ concentrations. In September 2006, the US EPA (2006) issued a new suite of standards to better protect public health from particle pollution, but retained the annual standard of 15 µg/m³ based on the 3-year average of annual $PM_{2.5}$ concentrations (US EPA 2006)). Based upon a comprehensive Integrated Science Assessment (US EPA 2009), the US EPA has reduced the annual NAAQS to 12 µg/m³ (US EPA 2014).

The WHO (2006) recommends an annual average of 10 µg/m³, and suggests the annual average should take precedence over the daily guideline because at low levels there is less concern for episodic excursions. The annual average guideline is based on long-term exposure studies using the American Cancer Society data (Pope *et al.* 2002) and Harvard Six-Cities data (Dockery *et al.* 1993). The studies reported a robust association between PM exposure and

mortality. Historical mean $PM_{2.5}$ concentrations across cities in these two studies were 18 and 20 µg/m³, respectively, but average concentrations in individual cities were as low as 11 µg/m³ over the period of study. An annual mean guideline concentration of 10 µg/m³ was therefore noted to be below the mean for most likely effects (WHO 2006). The WHO guideline was not selected, as it is less conservative than the and BC MOE values.

The California Air Resources Board (CARB) identified an air quality annual average standard for $PM_{2.5}$ of 12 µg/m³ (CARB 2005, 2002). This recommended arithmetic mean value was "based on a growing body of epidemiological and toxicological studies showing significant toxicity (resulting in mortality and morbidity) related to exposure to fine particles". Similar to the CEPA/FPAC (1999) reference level, the value was derived based on the average 24-hour concentrations in cities where statistically significant increases in health responses were detected. The CARB Staff report recommendation was adopted by the State of California as an ambient air quality standard in June of 2002. Due to the existence of local, more stringent criteria, the CARB standard was not selected for use in the assessment.

C20.3 Summary of Exposure Limits

Table 20-3
Summary of Exposure Limits Selected for $PM_{2.5}$

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	24-hour	Inhalation	AAQO	25	µg/m³	BC MOE 2013	–
Chronic	Annual	Inhalation	RfC	8	µg/m³	BC MOE 2013	–
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	n/a	n/a

– = Not available
n/a = Not applicable

C20.4 References

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

BC MOE (British Columbia Ministry of the Environment). 2008. Provincial Ambient Air Quality Criteria for PM2.5. Ministry Intentions Paper. http://www.bcairquality.ca/reports/pdfs/aq_criteria_intentions.pdf

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

CARB (California Air Resources Board). 2002. Staff Report: Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates. California Environmental Protection Agency, Air Resources Board. May, 2002.

CARB (California Air Resources Board). 2005. Particulate Matter - Overview. Available at: http://www.arb.ca.gov/research/aaqs/caaqs/pm/pm.htm. Webpage updated April, 2005.

CCME (Canadian Council of Ministers of the Environment). 2000. Canada-Wide Standards for Particulate Matter (PM) and Ozone. Accepted November 29, 1999, for endorsement in May 2000.

CCME (Canadian Council of Ministers of the Environment). 2012. Guidance Document on Achievement Determination Canadian Ambient Air Quality Standards for Fine Particulate Matter and Ozone.

CEPA/FPAC (Canadian Environmental Protection Act/Federal-Provincial Advisory Committee). 1999. The National Ambient Air Quality Objectives for Particulate Matter: Part 1: Scientific Assessment Document. Minister of Public Works and Government Services. Ottawa, ON. ISBN 0-662-26715-X. Cat. No. H46-2/98-220-1E.

Dockery, D.W., C.A. Pope III, X. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris Jr. and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N Engl J Med. 329(24):1753-1759.Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Federal Register. 2012. Environmental Protection Agency. 40 CFR Parts 50, 51, 52, 53 and 58. RIN 2060-AO47 National Ambient Air Quality Standards for Particulate Matter. Proposed Rules. Vol. 77, No. 126. Friday June 29 2012.

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Pope III, C.A., R.T. Burnett, M.J. Thun, E.E. Calle, D. Krewski, K. Ito and G.D. Thurston. 2002. Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate air pollution. JAMA. 287(9):1132-1141.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2005. Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. EPA-452/D-05-001. OAQPS Staff Paper – Second Draft. Research Triangle Park, NC: Office of Air Quality Planning and Standards, US Environmental Protection Agency.

US EPA (United States Environmental Protection Agency). 2006. Fact Sheet – Final Revisions to the National Ambient Air Quality Standards for Particle Pollution (Particulate Matter). Available at: http://epa.gov/pm/pdfs/20060921_factsheet.pdf

US EPA (United States Environmental Protection Agency). 2009. Integrated Science Assessment for Particulate Matter. National Center for Environmental Assessment – RTP Division. Office of Research and Development. December 2009. EPA/600/R-08/139F.

US EPA (United States Environmental Protection Agency). 2012. US EPA (United States Environmental Protection Agency). 2013. National Ambient Air Quality Standards. Accessed at: http://www.epa.gov/air/criteria.html

WHO (World Health Organization). 2006. Air Quality Guidelines: Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. ISBN 92 890 2192 6.

C21.0 PENTANE

C21.1 Acute Inhalation Exposure Limits

Table 21-1
Acute Inhalation Exposure Limits for Pentane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	–	–	OMOE 2012, 2005
TCEQ	1-hour ReV	200,000 (n-pentane)	TCEQ 2013, 2011
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The TCEQ (2013, 2011) has derived a 1-hour ReV of 200,000 µg/m³ for n-pentane. In the key study by Lammers *et al.* (2011), two acute experiments were conducted. In the first experiment, male WAG/RijCHBR rats (8 per group) were exposed to 0, 2,000, 6,500, or 20,000 mg/m³ of n-pentane for 8-hours per day for 3 consecutive days. An assessment of motor activity and neurobehavioural functions was conducted using a standardized functional observational battery of tests. No significant adverse neurological effects were observed in any of the exposure groups.

In the second experiment, male WAG/RijCHBR rats (8 per group) were exposed to 0, 2,000, 6,500, or 20,000 mg/m³ of n-pentane for 8-hours per day for 3 consecutive days, with tests for cognitive performance being conducted after exposure. Mild, reversible changes in learning performance speed were observed in the two lowest exposure groups, but not in the high-exposure group. Tests conducted 1 day post-exposure revealed no adverse effects due to n-pentane exposure. The TCEQ (2011) identified 20,000 mg/m³ (19,872 mg/m³ average measured concentration) as a free-standing NOAEL. The recommended default RGDR of 1 (TCEQ 2006) was applied to account for the ratio of the blood:gas coefficients of rats to humans being less than one, resulting in a POD of 19,872 mg/m³ (equivalent to the NOAEL). An uncertainty factor of 90 was applied to the POD to account for interspecies differences (3, due to the use of an RGDR), intraspecies differences (10), and database deficiencies (3), resulting in a 1-hour ReV of 200,000 µg/m³.

The ReV of 200,000 µg/m³ for pentane was selected to assess the aliphatic C_5-C_8 group in the acute inhalation assessment. Pentane was assessed separately in addition to its evaluation as part of the aliphatic C_5-C_8 group. Please refer to the toxicity profile for the aliphatic C_5-C_8 group for details.

C21.2 Chronic Inhalation Limits

Table 21-2
Chronic Inhalation Exposure Limits for Pentane

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

None of the agencies listed above provide chronic inhalation exposure limits for pentane. Expanding the search to include occupational TLV-TWA values from the ACGIH (2013), intermediate inhalation MRLs from ATSDR (2013), and PPRTVs from the US EPA (2013b) did not yield any chronic limits for pentane either. As a result, pentane was not evaluated individually in the chronic inhalation assessment, but due to its structural characteristics was assessed as part of the aliphatic C_5-C_8 group. Please refer to the toxicity profile for the aliphatic C_5-C_8 group for details.

C21.3 Oral Exposure Limits

Pentane was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, pentane was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C21.4 Summary of Exposure Limits

Table 21-3
Summary of Exposure Limits Selected for Pentane

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	ReV	200,000	µg/m³	TCEQ 2013, 2011	–
Chronic	Annual	Inhalation	–	–	–	–	–
		Multiple Exposure Pathway	n/a	n/a	n/a	n/a	–

– = Not available
n/a = Not applicable

C21.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

Lammers, J.H.C.M., H. Muijser, D.E. Owen, B.M. Kulig and R.H. McKee. 2011. Neurobehavioral effects of acute exposure to normal (n-) paraffins. International Journal of Toxicology 30(1):47-58.

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2005. Ontario Air Standards for n-Hexane. Standards Development Branch. Ontario Ministry of the Environment. June 2005.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2006. Guidelines to develop effects screening levels, reference values, and unit risk factors. Chief Engineer's Office. RG-442. Available from: http://www.tceq.state.tx.us/implementation/tox/esl/guidelines/about.html

TCEQ (Texas Commission on Environmental Quality). 2011. Pentane, All Isomers (CAS Registry Numbers: n-Pentane: 109-66-0, Isopentane: 78-78-4, Neopentane: 463-82-1. Developmental Support Document, Final, July 29, 2011. Prepared by: Jong-Song Lee, Ph.D. Toxicology Division, Chief Engineer's Office, Texas Commission on Environmental Quality.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

C22.0 PYRENE

C22.1 Acute Inhalation Exposure Limits

Table 22-1
Acute Inhalation Exposure Limits for Pyrene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	–	–	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

Acute inhalation exposure limits for pyrene are not available from the agencies listed above. The search for limits was expanded to include short-term occupational limit values (*i.e.*, STEL and Ceiling) developed by the ACGIH (2013), and AEGLs-1 developed by the US EPA (2013a). Acute exposure limits for pyrene were not available from these sources either, therefore pyrene was not assessed individually on an acute basis. Pyrene was included in the acute inhalation assessment as a component of the aromatic C_9-C_{16} group. Please refer to the toxicity profile for the aromatic C_9-C_{16} group for details.

C22.2 Chronic Inhalation Exposure Limits

Table 22-2
Chronic Inhalation Exposure Limits for Pyrene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2014
	–	–	OEHHA 2009
RIVM	–	–	RIVM 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

None of the agencies listed above provide chronic inhalation exposure limits for pyrene. Expanding the search to include occupational TLV-TWA values from the ACGIH (2013), intermediate inhalation MRLs from ATSDR (2013), and PPRTVs from the US EPA (2013b) did not yield any chronic limits for pyrene either. As a result, pyrene was not evaluated individually in the chronic inhalation assessment, but was assessed as part of the aromatic C_9-C_{16} group. Please refer to the toxicity profile for the aromatic C_9-C_{16} group for details.

C22.3 Oral Exposure Limits

Table 22-3
Chronic Oral Exposure Limits for Pyrene

Regulatory Agency	Type	Value (µg/kg bw/d)	Reference
ATSDR	–	–	ATSDR 2013
Health Canada	TDI	30	Health Canada 2010
	–	–	Health Canada 2013
OEHHA	–	–	OEHHA 2014
	–	–	OEHHA 2009
RIVM	CR_{oral}	50	RIVM 2001
US EPA	RfD	30	US EPA 2014, 1993
WHO	–	–	WHO 2014

– = Not available

The US EPA (2014,1993) presents a chronic RfD of 30 µg/kg/day for pyrene based on kidney effects in mice. Male and female CD-1 mice (20 per sex per dose) were exposed to 0, 75, 125, or 250 mg/kg/day pyrene in corn oil via oral gavage for 13 weeks. Mild kidney lesions were observed in all dose groups in both sexes, primarily renal tubular degeneration sometimes appearing with interstitial lymphocytic infiltrates or fibrosis. Relative and absolute kidney weights were reduced in the 125 and 250 mg/kg/day dose groups. The lowest dose group (75 mg/kg/day) was determined to be the NOAEL, while the 125 mg/kg/day was identified to be the LOAEL. An uncertainty factor of 3,000 was applied to the NOAEL to account for interspecies differences (10), intraspecies variability (10), the use of a subchronic study (10), and lack of data in another species and reproductive/developmental studies (3). The result is an oral RfD of 30 µg/kg bw/d based on kidney effects. This value was selected for use in the assessment.

The Health Canada (2010) TDI of 30 µg/kg/day was adopted from the US EPA (described above).

RIVM (2001) presents a CR_{oral} of 500 µg/kg/day for pyrene, which is associated with a lifetime excess cancer risk of one in 10,000. Converted to a risk level of one in 100,000, this value is 50 µg/kg/day. As limited information regarding this value was provided in the supporting documentation, it was not used in the assessment.

C22.4 Summary of Exposure Limits

Table 22-4
Summary of Exposure Limits Selected for Pyrene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	–	–	µg/m³	–	–
Chronic	Annual	Inhalation	–	–	µg/m³	–	–
		Multiple Exposure Pathway	RfD	30	µg/kg bw/d	US EPA 2014, 1993	Kidney effects

– = Not available

C22.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

Health Canada. 2013. Guidelines for Canadian Drinking Water Quality -Technical Documents. Available at: http://hc-sc.gc.ca/ewh-semt/pubs/water-eau/index-eng.php#tech_doc

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 1993. IRIS Summary of Pyrene (129-00-0). Reference Dose for Chronic Oral Exposure (RfD). Available at: http://www.epa.gov/ncea/iris/subst/0445.htm#reforal

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C23.0 SULPHUR DIOXIDE (SO_2)

C23.1 Acute Inhalation Exposure Limits

Table 23-1
Acute Inhalation Exposure Limits for Sulphur Dioxide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	1-hour MRL	26	ATSDR 2013, 1998
BC MOE	1-hour Level A	450	BC MOE 2011
	1-hour Level B	900	
	1-hour Level C	900-1,300	
	3-hour Level A	375	
	3-hour Level B	665	
	3-hour Level C	–	
	24-hour Level A	160	
	24-hour Level B	260	
	24-hour Level C	360	
ESRD	1-hour AAQO	450	ESRD 2013
	24-hour AAQO	125	
OEHHA	1-hour	660	OEHHA 2014, 2008
OMOE	1-hour	690	OMOE 2012
	24-hour	275	
TCEQ	–	–	TCEQ 2013
US EPA	1-hour NAAQS	196	US EPA 2010
WHO	24-hour AQG	20	WHO 2006
	10-minute AQG	500	WHO 2000

– = Not available

The 1-hour AAQO of 450 µg/m³ from the BC MOE (2013) was not selected for use in the assessment, as it is currently under review by the BC MOE (Personal Communication, 2014).

The US EPA (2010) has derived a 1-hour NAAQS of 75 ppb (196 µg/m³) for SO_2 that is intended to protect against short-term effects such as: decrements in lung function, respiratory symptoms, and respiratory morbidity as reflected by emergency department visits and hospital admissions. The value is based on a comparison with a 3-year average of the 99th percentile of the daily maximum 1-hour average concentrations of SO_2. This 1-hour NAAQS was used in the acute effects assessment for SO_2.

The ATSDR (2013, 1998,) has derived a 1-hour acute MRL of 26 µg/m³ based on respiratory irritation in two studies that involved asthmatics. In the first study, seven people were exposed to 0.1, 0.25 or 0.5 ppm of SO_2 (262, 655, or 1,310 µg/m³) via a mouthpiece, during exercise. The duration of exposure was not described by the ATSDR (1998). Significant effects on respiratory capacity were observed at 0.25 ppm and above, with very slight effects observed at 0.1 ppm. In the second study, two experiments were conducted. In the first experiment, six individuals were exposed to 1 ppm (2,620 µg/m³) of SO_2 via a mouthpiece for 5 minutes during exercise. In the second experiment, individuals were exposed to SO_2 concentrations of 1 ppm (2,620 µg/m³) and asked to voluntarily hyperventilate for an unknown duration of time. In both

experiments, all individuals experienced decreased respiratory function and wheezing. The ATSDR (1998) identified 0.1 ppm (262 µg/m$^{3)}$ as a minimal LOAEL for acute SO_2 exposure. An uncertainty factor of 9 was applied to this LOAEL to account for intraspecies differences (3) and the use of a LOAEL (3), resulting in a value of 0.01 ppm or 26 µg/m³. This value was not selected for use in the assessment for a number of reasons. Firstly, the studies upon which it is based involved the direct inhalation of SO_2 via a mouthpiece, as opposed to breathing within an exposure chamber (which would be more relevant to ambient air exposures). In addition, some elements of the study design were not clear, for example no control subjects were discussed, and the duration of exposure to each concentration was not well described. The results also seemed to be a mixture of exercising, resting and hyperventilating subjects, as well as individuals of varying health status. All of these factors influence the robustness of the ATSDR value, and as a result, this MRL was not selected for use in the assessment.

The OMOE (2012) present 1-hour and 24-hour air standards for sulphur dioxide of 690 and 275 µg/m³, both based on the protection of health and vegetation. However, no detailed supporting documentation is available for these standards. As a result, they were not used in the assessment.

The OEHHA (2014, 2008) presents a 1-hour value of 660 µg/m³ based on a NOAEL of 0.25 ppm (660 µg/m³), which is based on a review of multiple studies of clinical SO_2 exposure in humans. The studies reviewed by the OEHHA included normal, healthy individuals as well as asthmatics and atopic individuals, with exposure to SO_2 taking place during exercise as well as rest. Very limited information regarding study design or individual study results was provided, and uncertainty factors were not applied to the NOAEL in the derivation of the REL (OEHHA 2008). Due to the existence of a more conservative value from the US EPA that is supported by documentation, the OEHHA value was not used in the assessment.

WHO (2006) presents a 24-hour value of 20 µg/m³. However, the basis of this value with respect to human effect thresholds is not particularly clear. In addition, the supporting document (WHO 2006) notes that it is not certain whether or not the effects observed in large-scale epidemiological studies are attributable to SO_2 or another air contaminant such as ultrafine particulate. Given the lack of clarity regarding the basis of this guideline, and due to the existence of the more robust US EPA value, the WHO value was not selected for use in the assessment.

SO_2 also was assessed using a 10-minute AQG of 500 µg/m³ developed by the WHO (2000). This AQG is based on changes in lung function in asthmatics (WHO 2000). The 10-minute exposure period is relevant, given that the effects of sulphur dioxide exposure in humans primarily involved irritation at the point of contact (irritation) and 'peak' in severity within the first moments of exposure (WHO 2000).

Using the above objectives and guidelines, the acute assessment for sulphur dioxide was completed on a 10-minute and 1-hour basis.

C23.2 Chronic Inhalation Exposure Limits

Table 23-2
Chronic Inhalation Exposure Limits for Sulphur Dioxide

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	-	ATSDR 2013
BC MOE	Annual Level A Annual Level B Annual Level C	25 50 80	BC MOE 2013
BC MOE	–	–	BC MOE 2014
ESRD	Annual AAQO	20	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	– –	– –	OEHHA 2009 OEHHA 2014
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	-	WHO 2000

– = Not available

The BC MOE (2013) has derived Level A, B and C annual AAQOs of 25, 50, 80 µg/m³, respectively. These values have been obtained from pollution control objectives (PCO) for various sectors, and are currently under review. As a result, these values were not used in the assessment.

ESRD 2013 provides an annual AAQO of 20 µg/m³**.** This AAQO was adopted from the European Union, but was not used in the assessment as it is based on ecosystem effects rather than human health, and no supporting documentation is available.

C23.3 Oral Exposure Limits

Sulphur dioxide is a gaseous criteria air contaminant which acts on the point of contact once it is inhaled, (*i.e.*, the respiratory system). As such, it was not evaluated in the multiple pathway assessment.

C23.4 Summary of Exposure Limits

Table 23-3
Summary of Exposure Limits Selected for Sulphur Dioxide

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	10-minute	Inhalation	n/a	500	µg/m³	WHO 2000	Respiratory irritant
	1-hour	Inhalation	NAAQS	196	µg/m³	US EPA 2010	Respiratory irritant
Chronic	Annual	Inhalation	n/a	n/a	µg/m³	n/a	n/a
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	n/a	n/a

n/a = Not available

C23.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Sulphur Dioxide. Prepared by Sciences International, Inc. under subcontract to: Research Triangle Institute under contract No. 205-93-0606. Prepared for US Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry. Atlanta, GA. December 1998.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health-based Guidance Values for Substances on the Second Priority Substances List. ISBN 0-662-37275-1.

Health Canada. 2006. Regulations Related to Health and Air Quality. Available at: http://www.hc-sc.gc.ca/ewh-semt/air/out-ext/reg-eng.php

Health Canada. 2010. 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. September 2010.

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. TSD for Noncancer RELs. Appendix D. Individual acute, 8-hour, and chronic 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/AppendixD2_final.pdf#page=272

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

Personal Communication. 2014. Glenn Okrainetz, British Columbia Ministry of the Environment (BC MOE) to Bart Koppe, Intrinsik. May 15, 2014.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2010. 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 Rule.

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2006. Air Quality Guidelines: Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. ISBN 92 890 2192 6.

C24.0 TOLUENE

C24.1 Acute Inhalation Exposure Limits

Table 24-1
Acute Inhalation Exposure Limits for Toluene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	24-hour MRL	3,800	ATSDR 2013, 2000
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO 24-hour AAQO	1,880 400	ESRD 2013, 2004
OEHHA	1-hour REL	37,000	OEHHA 2014, 2008
OMOE	–	–	OMOE 2012
TCEQ	1-hour ReV	15,000	TCEQ 2013, 2008
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The same key study (Anderson *et al.* 1983) was chosen by the TCEQ, the ATSDR and OEHHA as the basis for their values.

As reported by Andersen *et al.* (1983), 16 healthy subjects with no previous exposure to organic solvents were exposed to toluene for 6 hours/day over 4 consecutive days. The concentration of toluene was 0, 10, 40, or 100 ppm with each group exposed to a different toluene concentration each day. After 1 hour of exposure to the desired toluene concentration, physiological measurements and performance assessments test were carried out on all subjects. The tests were repeated in the 5th and 6th hours of exposure. No adverse effects were reported at the 10 and 40 ppm levels, but statistically significant increased irritation was experienced in the eyes and nose at the 100 ppm concentration. There was also a statistically significant increase in the occurrence of headaches, dizziness, and feeling of intoxication. A NOAEL of 40 ppm (150 mg/m³) was identified.

The TCEQ, ATSDR and OEHHA share the opinion that the NOAEL of 40 ppm (150 mg/m³) is appropriate for short-term inhalation of toluene and that an uncertainty factor of 10 is sufficiently protective of the general population. The discrepancies between the limits derived arise from the duration adjustments applied by the individual regulatory agencies.

The TCEQ (2008) elected not to adjust the exposure duration based on a weight of evidence that suggests that concentration rather than duration is the primary determinant of the effects of toluene. The TCEQ (2008) only applied the uncertainty factor of 10 for intraspecies variability to the NOAEL of 40 ppm (150 mg/m³). The result is an acute ReV of 15,000 µg/m³, which was selected as the 1-hour exposure limit in the acute effects assessment of toluene, as it represents the most conservative value that takes into account the short-term, concentration-related effects of toluene. The toxicological basis of this value includes neurological effects and both eye and nasal irritation.

The ATSDR (2000) adjusted the NOAEL of 40 ppm to account for intermittent exposure (8/24 hours × 5/7 days). An uncertainty factor of 10 was applied to the adjusted NOAEL to account for intraspecies variability, resulting in an MRL 0f 0.95 ppm, which was rounded to 1 ppm (3,800 µg/m³). This value was not selected due to the adjustment to a 24 hour MRL.

The OEHHA (2014, 2008) converted the 6-hour exposure duration to a 1-hour REL of 98 ppm (370 mg/m³) based on a modified Haber's Law, and applied an uncertainty factor for intraspecies variability (10), resulting in an acute 1-hour REL of 37,000 µg/m³. This value was not used as the TCEQ value is more conservative.

ESRD 2013 has established a 1-hour AAQO of 1,880 µg/m³, which was adopted from the TCEQ ESL. However, TCEQ has since updated their health based acute ReVs and ESLs and therefore the ESRD limit is not up to date. The ESRD also provides a 24-hour AAQO of 400 µg/m³ adopted from the Michigan Department of Environmental Quality and the WA DOE (ESRD 2013, Alberta Environment 2004). These regulatory agencies based their 24-hour guidelines on the US EPA chronic inhalation RfC of 400 µg/m³ which has since been revised to be 5,000 µg/m³. As the ESRD values are based on out-dated chronic information, they were not considered further for use in the assessment.

C24.2 Chronic Inhalation Exposure Limits

Table 24-2
Chronic Inhalation Exposure Limits for Toluene

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	300	ATSDR 2013, 2000
BC MOE	–	–	BC MOE 2013
BC MOE[(1)]	–	5000	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	3,800	Health Canada 2010
OEHHA	REL	300	OEHHA 2014, 2000
RIVM	TCA	400	RIVM 2001
TCEQ	ReV	4,100	TCEQ 2013, 2008
US EPA	RfC	5,000	US EPA 2014, 2005
WHO	–	–	WHO 2000

– = Not available
(1) Based on the agricultural, urban park and residential use vapour standard

The US EPA (2014, 2005) has derived an inhalation RfC based on the findings of 10 human studies, each of which examined the neurological effects in occupationally exposed workers. These studies are more recent than the studies used by Health Canada and the ATSDR. An average NOAEL of 34 ppm (128 mg/m³) was identified from the meta-analysis. This NOAEL was adjusted for the differences in breathing rates between workers and members of the public and the reduced weekly exposure time (US EPA 2005):

$$NOAEL_{ADJ} = NOAEL \times \frac{MV_{ho}}{MV_{h}} \times \frac{Exp_{ho}}{Exp_{h}}$$

Where:

$NOAEL_{ADJ}$	=	no-observable-adverse-effects level in the human population from continuous exposure to toluene (mg/m³)
NOAEL	=	no-observable-adverse-effects level for discontinuous exposure in an occupational setting (128 mg/m³)
MV_{ho}	=	amount of air used by a worker during an 8-hour work period (10 m³/d)
MV_h	=	amount of air used by an individual in the general population during a day (20 m³/d)
Exp_{ho}	=	days per week a worker is exposed (5 days)
Exp_h	=	days per week an individual in the general population is exposed (7 days)

The US EPA (2005) also applied an uncertainty factor of 10 to the $NOAEL_{ADJ}$ to account for human variability. The US EPA RfC of 5,000 µg/m³ based on neurological effects represents the most recent analysis of the available scientific literature and therefore was used in the current assessment.

The BC MOE (2014) has also derived a vapour standard of 5000 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not provided.

The ATSDR (2013, 2000) has derived a chronic inhalation MRL of 0.08 ppm (300 µg/m³) based on colour vision impairment in workers exposed to toluene. Three groups of Croatian workers were examined through interviews, medical examinations and colour vision testing (Zavalic *et al.* 1998). A LOAEL of 35 ppm (130 mg/m³) was determined for alcohol- and age-adjusted colour vision impairment. The LOAEL was adjusted for intermittent exposure (8/24 hours × 5/7 days) to a concentration of 8 ppm (30 mg/m³). The ATSDR (2000) applied an uncertainty factor of 100 to the duration-adjusted LOAEL to account for intraspecies variability (10), and the use of a LOAEL (10). This MRL was not used as the chronic exposure limit for toluene as it was developed from a LOAEL, as opposed to the NOAEL used in the US EPA derivation.

Health Canada (2010) established its chronic tolerable concentration of 3,800 µg/m³ on the same lowest reported NOAEL of 150 mg/m³ (40 ppm) for neurological effects and respiratory irritation in human volunteers as used by the ATSDR to derive the acute MRL (Andersen *et al.* 1983; Government of Canada 1992). The study NOAEL was adjusted from 6-hour daily dosing to continuous exposure and an uncertainty factor of 10 was applied to account for intraspecies variability. This value was not selected for use as it was not based on a study of chronic duration.

The OEHHA (2014, 2000) has derived a chronic REL of 300 µg/m³ based on a rat study and supported by human data. In the key animal study, male rats were exposed to 0, 40, 80, 160 or 320 ppm for 6 hours/day, 5 days/week for 4 weeks (Hillefors-Berglund *et al.* 1995). Significantly decreased brain weights (specifically the caudate-putamen and subcortical limbic areas), and altered dopaminergic nerve receptor activity were observed at concentrations of 80 ppm and above. A human occupational study of female workers in an electronics assembly plant exposed on average to toluene vapours for about 5.7 years also suggested a LOAEL of about 88 ppm (Foo *et al.* 1990). The OEHHA selected 40 ppm as a NOAEL based on the animal data from Hillefors-Berglund *et al.* 1995. This value was adjusted for continuous exposure (6/24 hours, 5/7 days) to a $NOAEL_{ADJ}$ of 7 ppm. The OEHHA (2000) applied a cumulative uncertainty factor of 100 to account for the use of a subchronic study (10), a study of subchronic duration (3), and human variability (10). This value was not used in the chronic effects

assessment, as although it is verified by some human data, its basis is primarily derived from a subchronic animal study.

The TCEQ (2013, 2008) derived the chronic ReV of 4,100 µg/m³ based on the same study as the ATSDR (2013, 2000) value described above. A significant increase in colour confusion was observed at 132 ppm and a NOAEL of 32 ppm for the incidence of neurological effects was identified. This NOAEL was determined by the TCEQ to be supported by the results of three other studies, where average LOAELs ranging from 50 to 140 ppm were reported. The NOAEL of 32 ppm was adjusted to account for differences in the air volume inhaled by workers versus the general public, and to adjust for continuous exposure (10/20 m³/day × 5/7 days). The $NOAEL_{ADJ}$ was determined to be about 11.4 ppm. The TCEQ applied an uncertainty factor of 10 to this value to account for human variability. Preference was given to the US EPA RfC because its NOAEL was derived from the analysis of 10 different studies and is based on a greater scientific weight of evidence.

The RIVM (2001) has developed a TCA of 400 µg/m³ for toluene. This TCA was adopted from a previous US EPA RfC, which has since been revised. As a result, the RIVM value was not used in the chronic inhalation effects assessment for toluene.

C24.3 Oral Exposure Limits

Toluene was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, toluene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C24.4 Summary of Exposure Limits

Table 24-3
Summary of Exposure Limits Selected for Toluene

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	ReV	15,000	µg/m³	TCEQ 2013, 2008	Eye and nasal irritation, neurological effects
Chronic	Annual	Inhalation	RfC	5,000	µg/m³	US EPA 2014, 2005	Neurological effects
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	–	–

– = Not available
n/a = Not applicable

C24.5 References

Alberta Environment. 2004. Assessment Report on Toluene for Developing Ambient Air Quality Objectives. Edmonton, AB: Science and Standards Branch, Alberta Environment. ISBN No. 0-7785-3981-4.

Andersen, I., G.R. Lundqvist, L. Molhave, O.F. Pedersen, D.F. Proctor, M. Vaeth and D.P. Wyon. 1983. Human response to controlled levels of toluene in six-hour exposures. Scand J Work Environ Health 9:405-418.

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Toluene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. September 2000.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Foo, S.C., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7):480-484. Cited in: OEHHA 2000.

Government of Canada. 1992. Canadian Environmental Protection Act. Priority Substances List Assessment Report. Toluene. Ottawa, ON: Government of Canada.

Health Canada. 2010. 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. September 2010.

Hillefors-Berglund, M., Y. Liu and G. von Euler. 1995. Persistent, specific and dose-dependent effects of toluene exposure on dopamine D2 agonist binding in the rat caudate-putamen. Toxicology 100:185-94. Cited in: OEHHA 2000.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Determination of Chronic Noncancer Reference Exposure Levels. Chronic Toxicity Summary. Toluene.. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=560

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. TSD for Noncancer RELs. Acute Toxicity Summary Toluene. Appendix D2. June 2008. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=285

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

TCEQ (Texas Commission on Environmental Quality). 2008. Toluene (CAS Registry Number 108-88-3). Development Support Document, Final. April 15, 2008. Prepared by Manual Reyna Toxicology Section. Chief Engineer's Office.

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2005. Toxicological Review of Toluene (CAS No. 108-88-3). Available at: http://www.epa.gov/ncea/iris/toxreviews/0118tr.pdf

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

Zavalic, M., Z. Mandic, R. Turk, *et al.* 1998. Quantitative assessment of colour vision impairment in workers exposed to toluene. Am J Med 32: 297-304. Cited in: ATSDR 2000.

C25.0 TRIMETHYLBENZENES

C25.1 Acute Inhalation Exposure Limits

Table 25-1
Acute Inhalation Exposure Limits for Trimethylbenzenes

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
ESRD	–	–	ESRD 2013
OEHHA	–	–	OEHHA 2014
OMOE	24-hour	220	OMOE 2012
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available

The OMOE (2012) presents a 24-hour value of 220 µg/m³ for trimethylbenzenes; however, as no supporting documentation is available, this value was not considered in the risk assessment. Due to a lack of available limits from other agencies listed in the table above, the search was expanded to include values from the US EPA AEGL-1 (US EPA 2013a) list, and the ACGIH (2013) STEL or ceiling values.

The US EPA (2013a, 2007) has derived an AEGL-1 of 140 ppm (690,000 µg/m³) for all isomers of trimethylbenzene. Due to a lack of available human data for acute trimethylbenzene exposure, the AEGL-1 was derived from an analysis of several animal studies. Korsak and Rydzynski (1996) conducted a study involving acute (4-hour) exposure to 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene and 1,2,3-trimethylbenzene at concentrations ranging from 250 to 2,000 ppm (individual doses not specified) within a controlled chamber. Concentration-related changes were observed in rotarod performance in the exposed rats (male only). EC_{50} values for each isomer based upon disturbances in rotarod function were determined to be: 4,693 mg/m³ (95% CI 3,891 to 5,493 mg/m³) for 1,2,4-trimethylbenzene; 4,738 mg/m³ (95% CI 3,675 to 5,453 mg/m³) for 1,3,5-trimethylbenzene; and 3,779 mg/m³ (95% CI 2,832 to 4,615 mg/m³) for 1,2,3-trimethylbenzene. Changes in pain sensitivity also were observed for the three isomers in the acute study. EC_{50} values for pain sensitivity (demonstrated by the paw lick response) were determined to be the following: 5,682 mg/m³ (95% CI 2,715 to 7,596 mg/m³) for 1,2,4-trimethylbenzene; 5,938 mg/m³ (95% CI of 5,194 to 6,512 mg/m³) for 1,3,5-trimethylbenzene; and 4,155 mg/m³ (3,400 to 4,811 mg/m³ for 1,2,3-trimethylbenzene. Of the two endpoints, rotarod disturbance seems to be the more sensitive effect. Korzack and Rydzynski (1996) note that the 1,2,3-trimethylbenzene isomer appeared to demonstrate more neurotoxic potential than the other two isomers.

Also cited as a key study by US EPA (2007), Korsak *et al.* (1995) conducted a similar study with only 1,2,4-trimethylbenzene in male rats. Rats were exposed for a duration of 4 hours to 250 to 2,000 ppm (individual dose levels not specified) within a controlled chamber. Altered rotorod activity indicative of neurotoxicity, altered pain response and decreased respiratory rate were observed in association with concentration-dependent responses. EC_{50} values for rotorod

performance, pain sensitivity and respiratory depression were determined to be 4,693 mg/m³ (95% CI 3,891 to 5,493 mg/m³), 5,682 mg/m³ (95% CI 2,715 to 7,596 mg/m³) and 2,840 mg/m³ (95%,CI 1,500 to 3,900 mg/m³), respectively.

Although it is not clear how the US EPA calculated the value, the average EC_{50} for neurological effects was determined to be 900 ppm from the animal data, and served as the point of departure for the derivation of the AEGL. The Haber's Law approach was used by the US EPA (2007) to convert the 4-hour concentration to a 1-hour concentration:

$$(C_1)^n \times T_1 = (C_2)^n \times T_2$$

Where:

C_1 = 4-hour concentration (mg/m³)
T_1 = 4 hours
C_2 = converted 1-hour concentration (mg/m³)
T_2 = 1 hour
n = 3 (US EPA 2007)

A 1-hour concentration of 1,429 mg/m³ was calculated, and then adjusted by a total uncertainty factor of 10 to account for interspecies differences (3), and intraspecies differences (3), to result in the 1-hour AEGL of 690 mg/m³ (690,000 µg/m³).

The ACGIH (2013) has not derived a STEL for acute exposures.

As a result, the 1-hour AEGL from the US EPA (2007) of 690,000 µg/m³ based on neurotoxic effects was selected for use as a 1-hour exposure limit in the acute inhalation assessment.

C25.2 Chronic Inhalation Exposure Limits

Table 25-2
Chronic Inhalation Exposure Limits for Trimethylbenzenes

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	–	–	ATSDR 2013
BC MOE	–	–	BC MOE 2013
BC MOE[(1)]	–	6	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	–	–	Health Canada 2010, 2004
OEHHA	–	–	OEHHA 2014 OEHHA 2009
RIVM	–	–	RIVM 2009, 2001
TCEQ	–	–	TCEQ 2013
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

– = Not available
(1) Based on the agricultural, urban park and residential use vapour standard

The BC MOE (2014) derived a vapour standard of 6 µg/m³ for both 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not available. Therefore, this value was not selected for use in the chronic inhalation assessment.

As no other values were available from the sources listed in the table above, the search was expanded to include values from the ACGIH (2013) and the US EPA PPRTVs (US EPA 2013b).

The US EPA (2013b, 2010) has derived a chronic inhalation RfC as part of the PPRTVs. In the subchronic experiment within Korsak and Rydzinski (1996), male and female rats were exposed to 0, 25, 100 or 250 ppm of 1,2,4-trimethylbenzene or 1,2,3-trimethylbenzene for 6 hours/day, 5 days/week for a duration of 3 months. Tests for pain sensitivity and neurological performance (rotarod performance) were conducted over the 3-month period. No significant effects on body weight or clinical parameters were observed. Exposure to 1,2,3-trimethylbenzene was associated with abnormal rotarod performance at 100 ppm and 250 ppm. For 1,2,4-trimethylbenzene, decrements in rotarod performance were observed only at 250 ppm. No signs of improvement were observed in the 250 ppm group for either isomer in relation to rotarod performance after 2 weeks following the cessation of exposure. Changes in sensitivity were observed to be concentration dependent, and were statistically significant at all exposure concentrations of 1,2,3-trimethylbenzene, and at 100 ppm and 250 ppm of 1,2,4-trimethylbenzene. Recovery in pain sensitivity was observed following a 2-week recovery period after the last exposure. The US EPA (2010) conducted benchmark dose analysis based upon the pain sensitivity data for 1,2,3-trimethylbenzene, and selected a response level of 1 standard deviation from the mean. To conduct the benchmark dose modelling, the data from the highest exposure group was dropped, and the analysis focused on the lower dose levels. The 95% $BMCL_{1sd}$ was determined to be 97 mg/m³, which was further adjusted to a $BMCL_{ADJ}$ of 17 mg/m³. As the study was conducted in rats, the US EPA (2010) converted this $BMCL_{ADJ}$ to a $BMCL_{HEC}$ through the application of adjustments for continuous exposure and blood-to-air transfer coefficients for a category 3 gas using the following equation and variables:

$$BMCL_{HEC} = BMCL_{ADJ} \times \frac{(Hb/g)A}{(Hb/g)H}$$

$$= 17 \text{ mg/m}^3 \times \frac{62.6}{66.5}$$

$$= 16 \text{ mg/m}^3$$

The $BMCL_{HEC}$ was adjusted with an uncertainty factor of 3,000 to account for interspecies differences (3, due to the use of a HEC), database uncertainties (10), the use of a subchronic study (10), and intraspecies variability (10). The resulting RfC of 5 µg/m³ based on neurotoxicity was selected for use in the chronic inhalation assessment.

The US EPA (2007) also has derived a PPRTV for 1,2,4-trimethylbenzene of 7 µg/m³. The basis of this value is a study by Korsak *et al.* (2000), where male and female Imp: WIST rats were exposed to 0. 123, 492, or 1,230 mg/m³ of 1,2,4-trimethylbenzene for 6 hours/day, 5 days/week for a duration of 3 months. Blood samples were taken before the study began, as well as 1 week before study exposure termination. Pathological and histopathological assessments were conducted post-mortem. No clinical abnormalities were observed, and no significant differences in organ weights were noted. However, significant decreases in red blood

cells and increases in white blood cells were observed in males in the 1,230 mg/m³ group. In females, a concentration related decrease in reticulocyte counts was observed in the 1,230 mg/m³ group. Decreased clotting time was significant in females in the 492 and 1,230 mg/m³ groups. All exposed male rats demonstrated significantly increased sorbitol dehydrogenase that did not seem to be concentration-dependent. Male rats exposed to 492 mg/m³ demonstrated pulmonary lesions including increased proliferation of peribronchial lymphatic tissues and increases in interstitial lymphocytic infiltrations of tissue. Similar effects were not observed in the highest dose group of male rats (1,230 mg/m³), although increased alveolar macrophages in high dose males were observed in pulmonary tissues. High dose females exhibited pulmonary lesions with interstitial lymphocytic infiltrations. The US EPA (2007) determined that the study NOAEL was 123 mg/m³ based upon the incidence of haematological and respiratory lesions at and above 492 mg/m³. The US EPA (2007) adjusted for continuous exposure (6/24 hours, 5/7 days) to a $NOAEL_{ADJ}$ of 22 mg/m³. As the study was conducted in rats, the US EPA also adjusted the value to a $NOAEL_{HEC}$ using a default animal and human blood-to-air coefficient of 1 (due to a lack of chemical-specific information in either rats or humans for 1,2,4-trimethylbenzene). An uncertainty factor of 3,000 was applied to account for interspecies differences (3), database deficiencies (10), the use of subchronic data (10) and intraspecies differences (10). This value was not selected, as the PPRTV for 1,2,3-trimethylbenzene is slightly more conservative and is based upon benchmark dose modelling.

No chronic PPRTV for 1,3,5-trimethylbenzene was available from the US EPA.

An 8-hour TLV-TWA of 25 ppm (123,000 µg/m³) was identified. This value was derived from an occupational study where 27 workers were exposed to a solvent mixture containing 30% 1,3,5-trimethylbenzene and 50% 1,2,4-trimethylbenzene. Exposure of 10 to 60 ppm was associated with CNS effects (anxiety, nervousness), asthmatic bronchitis, and blood changes (hypochromic anemia, abnormal coagulation). The ACGIH notes that potential contamination of the solvent with benzene may have contributed to the blood changes. The original study cited by the ACGIH is available only in German, and this information could not be independently verified. From this the ACGIH (1992) derived a TLV-TWA of 25 ppm. As the ACGIH TLV-TWA is intended to apply to an 8-hour workday (rather than a continuous exposure over a 24-hour period), the TLV-TWA was adjusted for continuous exposure based upon the differences in inhalation volume over a 24-hour period vs. 8 hours and also over a 7-day week rather than a typical 5-day workweek (10/20 m³/day × 5/7 days) to a value of 9 ppm (44,000 µg/m³). As it is not clear whether or not an uncertainty factor was applied for intraspecies differences, an uncertainty factor of 10 was applied to the ACGIH TLV-TWA, resulting in an adjusted value of 4,400 µg/m³. This value was not selected, as the original study information could not be verified.

C25.3 Oral Exposure Limits

Trimethylbenzene was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, trimethylbenzene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C25.4 Summary of Exposure Limits

Table 25-3
Summary of Exposure Limits Selected for Trimethylbenzenes

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	1-hr AEGL	690,000	µg/m³	US EPA 2007	Neurological effects
Chronic	Annual	Inhalation	RfC	5	µg/m³	US EPA 2013b, 2010	Neurological effects
		Multiple Exposure Pathway	n/a	n/a	µg/kg bw/d	–	–

– = Not available
n/a = Not applicable

C25.5 References

ACGIH (American Conference of Governmental Industrial Hygienists). 1992. Trimethyl Benzene Isomers. CAS: 25552-13-7 (mixed isomers). 1,2,3-Trimethyl benzene CAS: 526-72-8, Hemimellitene. 1,2,4-Trimethyl benzene CAS: 95-63-6, Pseudocumene. 1,3,5-Trimethyl benzene CAS: 108-67-8, Mesitylene. Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. ACGIH®, Cincinnati. OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 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.

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Health Canada. 2004. Health based Guidance Values for Substances on the Second Priority Substances List. ISBN 0 662 37275 1.

Health Canada. 2010. 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. September 2010.

Korsak, Z. and K. Rydzynski. 1996. Neurotoxic effects of acute and subchronic inhalation exposure to trimethylbenzene isomers (pseudocumene, mesitylene, hemimellitene) in rats. Int J Occup Med Environ Health 9(4): 341-349.

Korsak, Z., J. Stetkiewicz, W. Majcherek, I. Stetkiewicz, J. Jajte and K. Rydzyński. 2000. Sub-chronic inhalation toxicity of 1,2,4-trimethylbenzene (pseudocumene) in rats. Int. J. Occup. Med. Environ. Health. 13(2):155-164. Cited in: US EPA 2007

Korsak, Z., S. Radoslaw and K. Rydzynski. 1995. Toxic effects of acute inhalation exposure to 1,2,4-trimethylbenzene (pseudocumene) in experimental animals. Int J Occup Med Environ Health 8(4):331-337

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Trimethylbenzenes). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

RIVM (National Institute of Public Health and the Environment). 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf

TCEQ (Texas Commission on Environmental Quality). 2013. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

US EPA (United States Environmental Protection Agency). 2007. Provisional Peer-Reviewed Toxicity Values for 1,2,4-trimethylbenzene. Superfund Health Risk Assessment Technical Center, National Center for Environmental Assessment. CASRN 95-63-6.

US EPA (United States Environmental Protection Agency). 2010. Provisional Peer Review Toxicity Values for 1,2,3-trimethylbenzene (CASRN 526-73-8). Available at: http://hhpprtv.ornl.gov/issue_papers/Trimethylbenzene123.pdf

US EPA (United States Environmental Protection Agency). 2013a. Acute Exposure Guideline Levels (AEGL) Chemicals. Available at: http://www.epa.gov/opptintr/aegl/pubs/chemlist.htm

US EPA (United States Environmental Protection Agency). 2013b. Provisional Peer Review Toxicity Values for Superfund (PPRTV) Derivation Support Documents. Available at: http://hhpprtv.ornl.gov/quickview/pprtv_papers.php

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91. ISBN 92 890 1358 3 ISSN 0378-2255

WHO (World Health Organization). 2014. Guidelines for Drinking-water Quality – Review Documents. Available at: http://www.who.int/water_sanitation_health/dwq/chemicals/en/index.html

C26.0 XYLENES

C26.1 Acute Inhalation Exposure Limits

Table 26-1
Acute Inhalation Exposure Limits for Xylenes

Regulatory Agency	Type	Value (µg/m³)[(a)]	Reference
ATSDR	1-hour MRL	8,700	ATSDR 2013, 2007
BC MOE	–	–	BC MOE 2013
ESRD	1-hour AAQO 24-hour AAQO	2,300 700	ESRD 2013
OEHHA	1-hour REL	22,000	OEHHA 2014, 2008
OMOE	24-hour standard	730	OMOE 2012, 2005
TCEQ	1-hour ReV	7,400	TCEQ 2014a,b
US EPA	–	–	US EPA 2014
WHO	–	–	WHO 2000

[(a)] Exposure limit provided for m-xylene, o-xylene, p-xylene or mixed isomers.
– = Not available

The TCEQ (2014a,b) has derived an acute ReV of 1.7 ppm (7,400 µg/m³) for xylenes based on mild respiratory effects and subjective symptoms of neurotoxicity. Ernstgard *et al.* (2002) was selected as the key study for the derivation of the acute ReV. In this study, 56 human volunteers were exposed to 50 ppm *m*-xylene, clean air, or 150 ppm 2-propanol for 2 hours in an inhalation chamber (TCEQ 2014b). The TCEQ (2014b) identified a LOAEL of 50 ppm based on breathing difficulty in both sexes and discomfort in the throat and airways of females. In addition, symptoms of neurotoxicity were reported, including fatigue, headache, dizziness, and a feeling of intoxication. All of these effects were considered minimal (TCEQ 2009). The LOAEL was not adjusted to a 1-hour exposure duration because the exposure concentration, as opposed to the duration of exposure, was identified as the primary determinant of the adverse effects of xylene (TCEQ 2009). An uncertainty factor of 10 was applied to the LOAEL to account for intraspecies variability and an uncertainty factor of 3 was applied to the LOAEL to account for use of a minimal LOAEL.

The ATSDR (2013, 2007) also selected the study by Ernstgard *et al.* (2002) as the basis of their MRL. A concentration of 50 ppm (200 mg/m³) was designated as a LOAEL for slight respiratory effects (*e.g.*, reduced forced vital capacity, increased discomfort in throat and airways in women and breathing difficulties in both sexes) and subjective symptoms of neurotoxicity (*e.g.*, headache, dizziness, feelings of intoxication). The LOAEL was considered minimal due to the minor nature of the effects observed (ATSDR 2007). The ATSDR (2007) applied an uncertainty factor of 30 for intraspecies variability (10) and use of a (minimal) LOAEL (3), resulting in an acute MRL of 2 ppm (8,700 µg/m³).

Although the TCEQ and ATSDR selected the same study and LOAEL based on respiratory irritation and neurological effects, the exposure limits are slightly different due to rounding differences. Given that the TCEQ provides a lower limit, this acute ReV of 7,400 µg/m³ was used as a 1-hour exposure limit in the acute assessment.

The OEHHA (2014, 2008) has derived a REL for 1-hour exposure of 22,000 µg/m³ based on irritation of the eyes, nose, and throat. In the study by Hastings *et al.* (1984), 50 healthy human volunteers were exposed for 30 minutes to concentrations of 430, 860, or 1,720 mg/m³ of technical grade (mixed) xylene. A NOAEL of 100 ppm (430 mg/m³) was identified by Hastings *et al.* (1984) as it was observed that the incidence of eye irritation was comparable to what was reported in the control group. The NOAEL was adjusted to a 1-hour exposure of 50 ppm (C × 60 minutes = 100 ppm × 30 minutes). A cumulative uncertainty factor of 10 was applied to the NOAEL to account for intraspecies variation. The result is an acute REL of 5 ppm (22,000 µg/m³). As the OEHHA limit is less conservative than the limit provided by TCEQ (2013, 2009) this exposure limit was not selected for use in the acute assessment.

ESRD 2013 adopted the OMOE's half-hour point-of-impingement of 2,300 µg/m³ as its 1-hour AAQO. However, this POI was based on odour perception and has since been updated (OMOE 2012). ESRD (2013) also provides a 24-hour AAQO of 700 µg/m³ which was adopted from the OEHHA. However, as the OEHHA value is based on chronic studies, it was not considered appropriate for use in the acute assessment.

The OMOE (2012, 2005) has derived a 24-hour criteria of 730 µg/m³ based on adverse neurological effects. A LOAEL of 62 mg/m³ was established for headaches, eye and nasal irritation and light headedness (floating sensation) in approximately 300 workers, 175 of whom were occupationally exposed for an average of 7 years. The LOAEL was adjusted by the OMOE (2005) to account for discontinuous exposure to a concentration of 22.1 mg/m³. As this 24-hour value is based on chronic exposure, it was not used in the assessment.

C26.2 Chronic Inhalation Exposure Limits

Table 26-2
Chronic Inhalation Exposure Limits for Xylenes

Regulatory Agency	Type	Value (µg/m³)	Reference
ATSDR	MRL	220	ATSDR 2013, 2007
BC MOE	–	–	BC MOE 2013
BC MOE[(1)]	–	100	BC MOE 2014
ESRD	–	–	ESRD 2013
Health Canada	TC	180	Health Canada 2010
OEHHA	REL –	700 –	OEHHA 2014, 2000 OEHHA 2009
RIVM	TCA	870	RIVM 2001
TCEQ	ReV	610	TCEQ 2014a,b
US EPA	RfC	100	US EPA 2014, 2003
WHO	–	–	WHO 2000

– = Not available
(1) Based on the agricultural, urban park and residential use vapour standard

The TCEQ (2014a,b) has derived a chronic ReV of 610 µg/m³ based on a study by Uchida *et al.* (1993), in which a LOAEL of 14 ppm was identified from a population of 175 workers who were exposed to xylenes for an average of 7 years. Eye irritation, sore throat and mild neurological effects were reported as the critical effects. Two supporting rat studies that present NOAELs

and LOAELs that are higher than the LOAEL of 14 ppm from the occupational study also are discussed by the TCEQ. No adjustments for continuous exposure were made by the TCEQ as xylene is quickly absorbed and excreted. A cumulative uncertainty factor of 100 was applied to the LOAEL of 14 ppm to account for the use of a minimal LOAEL (3), database uncertainties (3), and intraspecies variability (10). This resulting chronic ReV of 0.14 ppm (610 µg/m³) based on eye, upper respiratory and neurological effects in humans was selected for use in the chronic effects assessment.

The ATSDR (2013, 2007) derived a chronic MRL of 0.05 ppm (220 µg/m³) based on the same study as the TCEQ (2013, 2009) value described above (Uchida *et al.* 1993). The ATSDR also determined the study LOAEL to be 14 ppm, based on neurotoxicity, and nasal, throat and eye irritation. The ATSDR MRL of 220 µg/m³ is lower than the TCEQ, due to the use of a total uncertainty factor of 300 (compared to the uncertainty factor of 100 used by the TCEQ). The difference between the two uncertainty factors is related specifically to the difference in the uncertainty factors used to account for the use of a LOAEL. A factor of 3 was employed by the TCEQ on the basis that the effects associated with the LOAEL were minor in severity and mild in magnitude. The ATSDR used a standard factor of 10, and does not appear to comment on the severity or magnitude of effects in relation to the LOAEL. Review of the key study suggests that the effects observed were mild in nature. The TCEQ value was selected for use in the assessment.

The OEHHA (2014, 2000) has developed a chronic REL of 700 µg/m³ based on the incidence of eye irritation, sore throat and mild neurological effects using the same study selected by the TCEQ (described above). The OEHHA also identified a LOAEL of 14 ppm, however the OEHHA adjusted the LOAEL to 5.1 ppm to account for continuous exposure (taking into account the differences in breathing air volumes/day between workers and the general public (10/20 m³/day) and the number of days in a work-week (5 days/week) whereas the TCEQ did not. The OEHHA applied an uncertainty factor of 30 to the adjusted LOAEL to account for the use of a LOAEL (3, due to the minor nature of the adverse effects) and for human variability (10). This value was not chosen as the TCEQ value is more conservative.

The US EPA (2014, 2003) RfC of 100 µg/m³ was derived from a NOAEL of 217 mg/m³ for impaired motor coordination from a subchronic inhalation study in male rats (Korsak *et al.* 1994). In this study, male rats were exposed to 0, 50, or 100 ppm of *m*-xylene, n-butyl alcohol, or a 1:1 mixture of toluene and xylenes for 6 hours/day, 5 days/week for a 3-month duration. A LOAEL of 100 ppm and a NOAEL of 50 ppm were identified based on neurological effects (decreased rotarod performance and response to heat). The NOAEL of 50 ppm (217 mg/m³) was adjusted for continuous exposure (6/24 hours, 5/7 days). A safety factor of 300 was applied to the adjusted NOAEL to account for laboratory animal-to-human differences (3), intraspecies uncertainty to account for human variability and sensitive populations (10), extrapolation from subchronic to chronic duration (3), and uncertainties in the database (3). Although this value is the most conservative of those presented in the table above, it is based on animal data, and thus is associated with a greater degree of uncertainty. This value was not used, due to the existence of a defensible human-based value.

The BC MOE (2014) has also derived a vapour standard of 100 µg/m³ for agricultural, urban park, and residential land uses under the contaminated sites regulation of the Environmental Management Act (BC Regulation 375/96). However, supporting documentation was not available.

Health Canada (2010) provides a provisional TC of 180 µg/m³. This value is based on a LOAEL of 250,000 reported in a rat study in which pregnant rats were exposed to xylenes 24 hours/day for gestational days 7 to 15. The dose of 250,000 µg/m³ was the LOAEL for maternal effects as well as fetal retardation, increased fetal mortality and re-absorption. This value was adjusted to a human–child equivalent dose of 180,000 µg/m³. An uncertainty factor of 1,000 (intraspecies variability (10), interspecies variability (10), use of a LOAEL and study limitations (10)) was applied, resulting in a tolerable concentration of 180 µg/m³. This value was not used, due to the existence of a defensible human-based value.

The RIVM (2001) has developed a TCA of 870 µg/m³ for developmental neurotoxicity. In the key study, decreased rotarod performance was observed in the offspring of rats exposed to 200 ppm (870 mg/m³) technical grade xylene for 6 hours/day on gestational days 6 through 20 (Hass and Jakobsen 1993). The inhaled xylene concentration of 870 mg/m³ was reported by RIVM as the study LOAEL (no other exposure levels were reported and no NOAEL was identified). A cumulative uncertainty factor of 1,000 was applied to the LOAEL to account for interspecies variability (10), intraspecies variability (10), and use of a LOAL instead of a NOAEL (10). In a later study by the same group of investigators, Hass *et al.* (1995) questioned the rotarod performance test in the original study, as it was not conducted by experimenters who were blind to the exposure status of the rats. Further, decreased rotarod performance was not observed in the later Hass *et al.* (1995) study, which exposed rats to 500 ppm (2,200 mg/m³) mixed xylenes for 6 hours/day on gestation days 7 through 20. As well, offspring of rats exposed to 800 or 1,600 ppm (6,900 mg/m³) *p*-xylene for 6 hours/day on gestation days 7 through 16 performed similarly to offspring of non-exposed rats in tests of central nervous system development (Rosen *et al.* 1986). Due to the inconclusive significance of the toxicological endpoint (rotarod performance), and the existence of a human-based value, this TCA was not used in the chronic inhalation assessment for xylenes.

C26.3 Oral Exposure Limits

Xylene was not incorporated into the multiple exposure pathway assessment because it did not exceed the physical-chemical criteria used to determine whether a chemical is persistent or bioaccumulative in the environment. Thus, xylene was not evaluated in the multiple exposure pathway assessment and a chronic oral limit was not required.

C26.4 Summary of Exposure Limits

Table 26-3
Summary of Exposure Limits Selected for Xylenes

Duration	Averaging-Time	Exposure Pathway	Type	Value	Units	Reference	Endpoint Relevant to Mixtures
Acute	1-hour	Inhalation	ReV	7,400	µg/m³	TCEQ 2014a,b	Respiratory irritation, neurological effects
Chronic	Annual	Inhalation	ReV	610	µg/m³	TCEQ 2014a,b	Eye irritation, nasal irritation, and neurological effects
		Multiple Exposure Pathway	–	–	µg/kg bw/d	–	–

– = Not available

C26.5 References

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

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

BC MOE (British Columbia Ministry of the Environment). 2013. Ambient Air Quality Objectives – Updated August 12, 2013. Available at: http://www.bcairquality.ca/reports/pdfs/aqotable.pdf

Ernstgard, L., E. Gullstrand, A. Lof and G. Johanson. 2002. Are women more sensitive than men to 2-propanol and m-xylene vapours? Occup Environ Med. 59:759-767.

ESRD (Environment and Sustainable Resource Development). 2013. Alberta Ambient Air Quality Objectives and Guidelines. Air Policy Branch. ISBN: 978-1-4601-1253-3. Issued August 2013. Available at: http://environment.gov.ab.ca/info/library/5726.pdf

Hass, U. and B.M. Jakobsen. 1993. Prenatal toxicity of xylene inhalation in the rat: a teratogenicity and postnatal study. Pharmacol Toxicol 73:20-23

Hass, U., S.P. Lund, L. Simonsen and A.S. Fries. 1995. Effects of prenatal exposure to xylene on postnatal development and behavior in rats. Neurotoxicol Teratol 17(3):341-349.

Hastings, L., G.P. Cooper and W. Burg. 1984. Human sensory response to selected petroleum hydrocarbons. In: Advances in Modern Environmental Toxicology. Volume VI. Applied Toxicology of Petroleum Hydrocarbons. 1984. MacFarland, H.N., Holdsworth, C.E., MacGregor, J.A., Call, R.W. and Lane, M.L, eds. Princeton Scientific Publishers, Inc., Princeton, New Jersey. p. 255-270.

Health Canada. 2010. 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. September 2010.

Korsak, Z., J. Wisniewska-Knypl and R. Swiercz. 1994. Toxic effects of subchronic combined exposure to n-butyl alcohol and m-xylene in rats. International Journal of Occupational Medicine and Environmental Health 7:155-166. Cited In: US EPA 2014.

OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Determination of Noncancer Reference Exposure Levels. Chronic Toxicity Summary. Xylenes .April 2000. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=602

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. Determination of Noncancer Reference Exposure Levels. Acute Toxicity Summary. Xylenes. June 2008. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD2_final.pdf#page=312

OEHHA (California Office of Environmental Health Hazard Assessment). 2009. Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. May 2009. Available at: http://www.oehha.ca.gov/air/hot_spots

OEHHA (California Office of Environmental Health Hazard Assessment). 2014. Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment. Sacramento, CA. Available at: http://www.oehha.ca.gov/air/allrels.html

OMOE (Ontario Ministry of the Environment). 2005. Ontario Air Standards for Xylenes. Standards Development Branch, Ontario Ministry of the Environment.

OMOE (Ontario Ministry of the Environment). 2012. Summary of Standards and Guidelines to Support Ontario Regulation 419/05 - Air Pollution – Local Air Quality (including Schedule 6 of O. Reg. 419/05 on Upper Risk Thresholds) (sorted by Chemical Name). Standards Development Branch, Ontario Ministry of the Environment. PIBS # 6569e01. April 2012.

RIVM (National Institute of Public Health and the Environment). 2001. Re-evaluation of human toxicological maximum permissible risk levels. RIVM Report 711701 025. March 2001.

Rosen, M.B., K.M. Crofton and N. Chernoff. 1986. Postnatal evaluation of prenatal exposure to p-xylene in the rat. Toxicol Let 34:223-229.

TCEQ (Texas Commission on Environmental Quality). 2014a. Final Development Support Documents (DSDs). Available at: http://www.tceq.state.tx.us/implementation/tox/dsd/final.html

TCEQ (Texas Commission on Environmental Quality). 2014b. Xylenes Development Support Document.. March 14, 2014.

Uchida, Y., H. Nakatsuka, H. Ukai, T. Watanabe, Y-T. Liu, M-Y. Huang, Y-L. Want, F-Z. Zhu, H. Yin and M. Ikeda. 1993. Symptoms and signs in workers exposed predominately to xylenes. Int Arch Occup Environ Health 64:597-605.

US EPA (United States Environmental Protection Agency). 2003. IRIS Summary of Xylenes (1330-20-7). Reference Concentration for Chronic Inhalation Exposure (RfC). Available at: http://www.epa.gov/ncea/iris/subst/0270.htm#refinhal

US EPA (United States Environmental Protection Agency). 2014. 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

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, Second Edition. World Health Organization, Regional Office for Europe, Copenhagen. WHO Regional Publications, European Series, No. 91.

Appendix D - Worked Example

Table of Contents

List of Tables

List of Examples

Glossary

%	percent
µg/d	microgram per day
µg/m³	microgram per cubic metre
µg/mg	microgram per milligram
µm	micrometre
AE	assimilation efficiency
AIR	air inhalation rate
atm m³/mol	Henry's Law constant in atmospheres cubic meter per mole
BC MOE	British Columbia Ministry of the Environment
BC OGC NEWT	British Columbia Oil and Gas Commission North East Water Tool
BCF	Bioconcentration factor(s)
BD	bulk density
BTF	biotransfer factors
BW	body weight
C	chemical concentration
CF	conversion factor
cm	centimetre
cm³	cubic centimetre
CO_2	carbon dioxide
COPC	chemical(s) of potential concern
D	deposition
DL	dust level
DW	dry weight
e.g.	Latin "for example"
EDI	estimated daily intake
FMR	free-living (or field) metabolic rate
g/cm³	gram(s) per square metre(s)
g/d	gram(s) per day
g/yr	gram(s) per year
GE	gross energy
HHRA	human health risk assessment
ILCR	incremental lifetime cancer risk
IR	ingestion rate
K	coefficient
K	Kelvin
kcal/d	kilocalorie(s) per day
kg	kilogram(s)
kg/m²/yr	kilogram(s) per squared metre(s) per year
kg/m³	kilogram(s) per cubic metre(s)
kJ/kcal	kilojoule per kilocalorie
K_{ow}	octanol-water partition coefficient
L	load
L/d	litre(d) per day
L/kg	litre(s) per kilogram
log K_{ow}	logarithmic octanol-water partition coefficient
m	metre(s)
m²	squared metre(s)
m³	cubic metre(s)
m³/L	cubic metre(s) per litre(s)

m^3/yr	cubic metre(s) per year
ME	metabolizable energy
MF	metabolism factor(s)
mg/d	milligram(s) per day
mg/kg	milligram(s) per kilogram
mg/kg/yr	milligram(s) per kilogram(s) per year
mg/L	milligram(s) per litre
$mg/m^2/yr$	milligram(s) per squared metre(s) per year
mg/yr	milligram(s) per year
ml	millilitre
mm/kg ww	millimetre(s) per kilogram(s) wet weight
mmHg	millimetres of mercury (vapour pressure)
MPOI	maximum point of impingement
na	not applicable
PAH	polycyclic aromatic hydrocarbons
RAF	relative absorption factor
RAIS	Risk Assessment Information System
RfD	reference dose
RQ	risk quotient
RsD	risk-specific dose
RTI	Research Triangle Institute
S	solubility
SLRA	screening level risk assessment
t	time
US EPA OSW	United States Environmental Protection Agency Office of Solid Waste
US EPA	United States Environmental Protection Agency
V	velocity
V	volume
VOC	volatile organic compounds
VP	vapour pressure
WW	wet weight
yrs	years
Z	zone

D1.0 INTRODUCTION

The detailed human health risk assessment (HHRA) focused on both the direct and indirect health risks associated with air emissions from oil and gas activities alone and in combination with other emissions sources within the study area. Chemicals of potential concern (COPC) are emitted directly into air from various sources within the study area. Thus, people residing in the study area, as well as people visiting the area could be exposed to the COPC via inhalation.

The primary pathway of exposure that was assessed in the detailed HHRA was inhalation; however, people that live in the area might also be exposed to COPC via secondary exposure pathways. Some COPC emitted to the atmosphere via air emissions may be considered bio-accumulative and persistent, and have the potential to be deposited onto the soils and plants in the study area. Chemical deposition and uptake could affect soil, water and food (*i.e.*, plants, game and fish) quality depending on the physical and chemical properties of the COPC and the potential of the COPC to bioaccumulate within environmental media or tissues. Screening of the COPC based on physical and chemical characteristics was completed in Section 3.3.3.1 of the HHRA. The COPC assessed in the multiple pathway exposure model were as follows:

- Benzo(a)pyrene;
- Benz(a)anthracene;
- Benzo(b)fluoranthene;
- Benzo(k)fluoranthene;
- Chrysene;
- Fluoranthene;
- Indeno(1,2,3-cd)pyrene;
- Phenanthrene;
- Acetaldehyde;
- Acrolein;
- Formaldehyde.

Health risks associated with indirect exposure pathways such as consumption of game and other country foods were characterized through a detailed multimedia or multiple pathway exposure model used to predict long term exposures from non-volatile, persistent or bioaccumulative COPC. Chronic multiple pathway risks were estimated with oral COPC exposure limits considered protective of human health and sensitive individuals (*e.g.,* children, elderly, people with compromised health).

This Appendix presents the methods and calculations used to estimate media concentrations and human exposures to the COPC from long-term (chronic) multiple pathway exposures from the detailed HHRA. Many of the methods, equations and assumptions used to predict concentrations in various environmental media were obtained from the United States Environmental Protection Agency Office of Solid Waste (US EPA OSW 2005) and Health Canada (2012). Potential multiple pathway exposures to the COPC were predicted for using the highest annual average concentrations for the entire study area (*e.g.* MPOI) and the highest predicted annual average concentrations for each community included in the detailed HHRA.

D2.0 ENVIRONMENTAL MEDIA CONCENTRATIONS

In order to quantify potential human exposures (and associated potential health impacts) through multiple exposure pathways, predicted chemical concentrations in various environmental media were required to estimate exposures and characterize risks. Chemical concentrations in the following media were estimated for the multiple pathway exposure model:

- Soil;
- Surface water;
- Foods consumed by wildlife and agricultural animals (forage, aquatic plants, invertebrates);
- Traditional foods (berries, wild above ground plants such as Labrador tea);
- Garden vegetables (above and below ground plants);
- Dusts;
- Agricultural foods (beef, dairy, chicken and chicken eggs);
- Wild game meat (large and small game mammals, wild birds);
- Fish

Media concentrations and multiple pathway health risks were predicted for two different emission scenarios (Oil and Gas Scenario, Cumulative Scenario) and lifestyles (*i.e.,* Aboriginal, Agricultural, and Community resident). Community-specific risk estimates were calculated and presented in the detailed HHRA. This worked example is presented for an Agricultural toddler exposed to benzo(a)pyrene in the Oil and Gas Scenario for the community of Arras. The results are presented for the toddler lifestage in this example, as toddlers typically represent the most sensitive life stage due to their exposure rates relative to body weight.

D2.1 Chemical Concentrations in Air

The detailed HHRA relied on predicted agricultural food, wild game meat, surface water and dugout concentrations based on the predicted annual average concentrations at the maximum point of impingement (MPOI). In addition, the HHRA used predicted soil, traditional plants, garden vegetables, and dust concentrations based on the predicted annual average concentrations at residential and / or community locations.

D2.1.1 Chemical Deposition

Atmospheric deposition is based on two forms of deposition (*i.e.*, dry and wet) and two chemical phases (*i.e.*, vapour and particulate). The US EPA OSW (2005) recommends calculating chemical deposition based on the following four parameters:

- D_{ydv} = Unitized yearly average dry deposition from vapor phase (mass/m^2-yr)
- D_{ywv} = Unitized yearly average wet deposition from vapor phase (mass/m^2-yr)
- D_{ydp} = Unitized yearly average dry deposition from particle phase (mass/m^2-yr)
- D_{ywp} = Unitized yearly average wet deposition from particle phase (mass/m^2-yr)

Predicted deposition in the human and ecological models was simplified by combining the dry vapour and dry particulate phases and combining the wet vapour and wet particle phases since the same deposition rate was applied to both forms and the calculation can be reduced.

A description of the predicted dry and wet chemical deposition is provided below.

D2.1.2 Dry Deposition

Dry deposition rates were estimated with the following equation:

$$D_{dry} = C_a \times V_d \times CF1 \times CF2$$

Where:

D_{dry}	=	deposition rate of COPC (mg/m²/yr)
C_a	=	COPC concentration in air (µg/m³)
V_d	=	dry deposition velocity for COPC (7.6E-03 m/s)
CF1	=	conversion factor from seconds per day (31,536,000 sec/year)
CF2	=	conversion factor from µg to mg (0.001 mg)

Example 1 Dry deposition rate of benzo(a)pyrene for prediction of ecological exposures

$$D_{dry} = 1.71E-05 \times 0.0076 \times 31{,}536{,}000 \times 0.001$$

$$D_{dry} = 4.11E-03\ mg/m^2/yr$$

Example 2 Dry deposition rate of benzo(a)pyrene for prediction of human exposure

$$D_{dry} = 6.23E-08 \times 0.0076 \times 31{,}536{,}000 \times 0.001$$

$$D_{dry} = 1.49E-05\ mg/m^2/yr$$

D2.1.3 Wet Deposition

Wet deposition rates were estimated with the following equation:

$$D_{wet} = C_a \times V_w \times CF1 \times CF2$$

Where:

D_{wet}	=	deposition rate of COPC (mg/m²/yr)
C_a	=	COPC concentration in air (µg/m³)
V_w	=	wet deposition velocity for COPC (0.00325 m/s)
CF1	=	conversion factor from seconds per day (31,536,000 sec/year)
CF2	=	conversion factor from µg to mg (0.001 mg)

Example 3 Wet deposition rate of benzo(a)pyrene for prediction of ecological exposure

$$D_{wet} = 1.71E-05 \times 3.25E-03 \times 31{,}536{,}000 \times 0.001$$

$$D_{wet} = 1.75E-03\ mg/m^2/yr$$

Example 4 Wet deposition rate of benzo(a)pyrene for prediction of human exposure

$$D_{wet} = 6.23E-08 \times 3.25E-03 \times 31{,}536{,}000 \times 0.001$$

$$D_{wet} = 6.38E-06\ mg/m^2/yr$$

D2.1.4 Total Deposition

Total deposition rates were estimated with the following equation:

$$D_{tot} = D_{dry} + D_{wet}$$

Where:

D_{tot}	=	deposition rate of COPC (mg/m²/yr)
D_{dry}	=	dry deposition (mg/m²/yr)
D_{wet}	=	wet deposition (mg/m²/yr)

Example 5 Total deposition rate of benzo(a)pyrene for prediction of ecological exposures

$$D_{tot} = 4.11E-03 + 1.75E-03$$

$$D_{tot} = 5.86E-03\ mg/m^2/yr$$

Example 6 Total deposition rate of benzo(a)pyrene for prediction of human exposure

$$D_{tot} = 1.49E-05 + 6.38E-06$$

$$D_{tot} = 2.13E-05\ mg/m^2/yr$$

D2.2 Chemical Concentrations in Soil

D2.2.1 Predicted Chemical Concentrations in Soil

Soil concentrations were estimated based on the calculated chemical-specific deposition rates. Deposition to soil on a mass basis was calculated using the following equation:

$$D_s = \frac{D_{tot}}{Z_s \times BD}$$

Where:

D_s	=	chemical-specific deposition (mg/kg/yr)
D_{tot}	=	chemical-specific deposition rate (mg/m^2/yr)
Z_s	=	soil mixing zone depth (m)
BD	=	soil bulk density (kg/m^3)

For the current assessment, the bulk density was assumed to be 1,500 kg/m^3, and soil concentrations were predicted for two mixing depths (*i.e.*, 2 cm and 20 cm) to calculate surface soil and soil concentrations, respectively.

Example 7 Deposition of benzo(a)pyrene to surface soil for prediction of ecological exposure

$$D_s = \frac{5.86E-03}{0.02 \times 1{,}500}$$

$$D_s = 1.95E-04\ mg/kg/yr$$

Example 8 Deposition of benzo(a)pyrene to surface soil for prediction of human exposure

$$D_s = \frac{2.13E-05}{0.02 \times 1{,}500}$$

$$D_s = 7.10E-07\ mg/kg/yr$$

Example 9 Deposition of benzo(a)pyrene to soil for prediction of ecological exposure

$$D_s = \frac{5.86E-03}{0.2 \times 1{,}500}$$

$$D_s = 1.95E-05\ mg/kg/yr$$

Example 10 Deposition of benzo(a)pyrene to soil for prediction of human exposure

$$D_s = \frac{2.13E-05}{0.2 \times 1{,}500}$$

$$D_s = 7.10E-08\ mg/kg/yr$$

D2.2.2 Calculating Chemical Loss Constants

Chemicals may be lost from soil by leaching, runoff, erosion, biotic and abiotic degradation, and volatilization. Only abiotic and biotic degradation and volatilization processes were considered for this assessment. The total rate at which a chemical is lost from soil was designated as *kt*.

D2.2.3 Chemical Loss via Biotic and Abiotic Degradation

The soil half-life values for abiotic and biotic degradation (*i.e.*, ks) were obtained from the US EPA OSW (2005). The US EPA OSW (2005) recommends a soil loss constant (ks) of 0.48 yrs^{-1} for benzo(a)pyrene.

D2.2.4 Chemical Loss via Volatilization

Chemical loss from volatilization was predicted as follows (Swan *et al.* 1979):

$$t_{1/2} = 1.58E-08 \times \left(\frac{K_{oc} \times S}{VP} \right)$$

Where:

$t_{1/2}$	=	soil half-life (days)
K_{oc}	=	organic carbon partition coefficient (L/kg)
S	=	water solubility (mg/L)
VP	=	vapour pressure (mmHg)

The half-life is then converted to a rate constant (yrs^{-1}) using the following equation:

$$kv = \frac{0.693}{\left(t_{1/2} / 365 \right)}$$

Example 11 Chemical loss or degradation from soil as a result of volatilization of benzo(a)pyrene

Soil half-life:

$$t_{1/2} = 1.58E-08 \times \left(\frac{6.31E+05 \times 1.62E-03}{5.49E-09} \right)$$

$$t_{1/2} = 2.94E+03 \; days$$

Loss as a result of volatilization:

$$kv = \frac{0.693}{\left(2.94E+03 / 365 \right)}$$

$$kv = 8.60E-02 \; yrs^{-1}$$

D2.2.5 Total Soil Loss Constant

$$kt = ks + kv$$

Where:

kt	=	chemical-specific soil loss constant as a result of all processes (yrs^{-1})
ks	=	chemical-specific soil loss constant as a result of abiotic and biotic degradation (yrs^{-1})
kv	=	chemical-specific soil loss constant as a result of volatilization (yrs^{-1})

Example 12 Total soil loss constant as a result of all processes for benzo(a)pyrene

$$kt = 0.48 + 8.60E-02$$

$$kt = 5.66E-01\ yrs^{-1}$$

D2.2.6 Calculation of Soil Concentrations

Soil concentrations were calculated on a mass per mass basis (mg/kg) based on the following equation:

$$C_s = \frac{D_s \times [1 - \exp(-kt \times tD)]}{kt}$$

Where:

C_s	=	average soil concentration over exposure duration (mg/kg soil)
D_s	=	deposition to surface soil or soil (mg of chemical/kg of soil/yr)
kt	=	chemical soil loss constant due to all processes (degradation or loss due to volatilization) (yrs^{-1})
tD	=	time period over which deposition occurs (yrs)

Chemical deposition for the detailed HHRA was assumed to occur for 80 years or a life time.

Example 13 Concentration of benzo(a)pyrene in surface soil for the prediction ecological exposure

$$C_s = \frac{1.95E-04 \times [1 - \exp(-5.66E-01 \times 80)]}{5.66E-01}$$

$$C_s = 3.45E-04\ mg/kg$$

Example 14 Concentration of benzo(a)pyrene in surface soil for the prediction of human exposure

$$C_s = \frac{7.10E-07 \times [1 - \exp(-5.66E-01 \times 80)]}{5.66E-01}$$

$$C_s = 1.25E-06\ mg/kg$$

Example 15 Concentration of benzo(a)pyrene in soil the prediction of ecological exposure

$$C_s = \frac{1.95E-05 \times [1 - \exp(-5.66E-01 \times 80)]}{5.66E-01}$$

$$C_s = 3.45E-05\ mg/kg$$

Example 16 Concentration of benzo(a)pyrene in soil for the prediction of human exposure

$$C_s = \frac{7.10E-08 \times [1 - \exp(-5.66E-01 \times 80)]}{5.66E-01}$$

$$C_s = 1.25E-07\ mg/kg$$

D3.0 CHEMICAL CONCENTRATIONS IN SURFACE WATER

COPC concentrations in surface water due to deposition were predicted based on guidance provided by US EPA OSW (2005). A single water body area (Charlie Lake) was selected to estimate human (with the exception of the Agricultural group) and ecological exposures (with the exception of agricultural animals) to surface water based on the predicted highest annual average air concentration. Charlie Lake is one of the larger surface water bodies in the HHRA study area, and is located in an area identified in the Screening Level Risk Assessment (SLRA) as having a high density of air pollutant emissions and a high density of oil and gas development. Parameters for this lake and its drainage area were obtained from BC OGC NEWT database (BC OGC 2014), the BC MOE (1985) and the Charlie Lake Conservation Society (2005).

This lake was used to predict human exposures (for all the lifestyle categories) for the following pathways of exposure:

- Surface water ingestion through swimming;
- Dermal uptake during swimming;
- Fish consumption.

The water body was used to predict ecological exposures for the following pathways:

- Surface water ingestion;
- Consumption of aquatic plants by large game animals.

For the Agricultural animals and residents, it was assumed that water from a dugout was consumed. The dimensions of this dugout were generic, and obtained from a document entitled “Water Development Standards on Crown Land within the Peace Forest District” as per guidance from the Peace River Forest District (Personal Communication, 2014) regarding

minimum dugout requirements for the Peace region. Information regarding surface runoff parameters were assumed to be the same as for the lake as the landscapes were assumed to be generally similar.

Based on US EPA OSW (2005), COPC emissions to the atmosphere can impact surface water bodies based on the following mechanisms of mass transfer or loading:

- Direct deposition from the atmosphere;
- Runoff from impervious surfaces (*e.g.*, concrete or asphalt);
- Runoff from pervious surfaces (*e.g.*, grasslands or forests);
- Soil erosion;
- Vapour diffusion; and
- Transformation of chemicals via chemical or biological mechanisms.

As recommended by the US EPA OSW (2005), the loading of COPC via transformation was assumed to be zero. In addition, surface water loading from runoff from impervious surfaces was assumed to be zero since the local study area is assumed to consist of pervious surfaces primarily and that impervious surfaces would have minimal influence. Finally, vapour diffusion was not predicted explicitly since vapour deposition was already included within the direct deposition calculations.

D3.1 Mass Loading onto Surface Water

Mass loading of a chemical onto the surface water body is assumed equal to the total deposition of the chemical emitted into air in the study area and deposited onto surface water. For mass loading onto surface water, the predicted annual average air concentration for the MPOI was used. Using the benzo(a)pyrene air concentration (Oil and Gas Scenario) of 2.96E-07 µg/m³, the predicted deposition of benzo(a)pyrene to surface water in the Oil and Gas Scenario was 1.01E-04 mg/m²/year. The calculation example is presented for both the dugout and the lake, although only the dugout water source was considered for the Agricultural toddler. The following equation was used to predict the mass loading of COPC to a water body:

$$L_d = ML_{sw} \times LA$$

Where:

L_d	=	total annual mass loaded to surface water (mg/yr)
ML_{sw}	=	mass of chemical loaded to water body on an annual basis (mg/yr) = D_{tot} (1.01-04 mg/m^2/yr)
LA	=	lake area of water body (m^2)

Example 17 Mass loading of benzo(a)pyrene to surface water (dugout)

$$L_d = 5.86E-03 \times 797$$

L_d = 4.67E+00

Example 18 Mass loading of benzo(a)pyrene to surface water (lake)

$$L_d = 1.01E-04 \times 19{,}000{,}000$$

$$L_d = 1.92E+03\ mg/yr$$

D3.2 Runoff from Pervious Surfaces

The following equation is recommended by US EPA OSW (2005) to predict the runoff load of dissolved COPC to a water body from pervious soil surfaces within the watershed. The calculation example is presented for both the dugout and the lake, although only the dugout water source was considered for the Agricultural toddler.

$$L_r = RO \times A_p \times \frac{C_s \times BD}{\theta_{sw} + k_d \times BD} \times CF$$

Where:

L_r	=	runoff load from pervious surfaces (mg/yr)
RO	=	average annual surface water runoff from pervious areas (cm/yr)
A_p	=	pervious watershed area (m^2)
C_s	=	soil concentration (mg/kg)
BD	=	bulk density of soil (g/cm^3)
θ_{sw}	=	soil volumetric water content (ml-water / cm^3-soil)
K_d	=	soil-water partition coefficient (cm^3-water / g-soil or L/kg)
CF	=	conversion factor 10 (kg-cm^2 / g-m^2)

Example 19 Runoff load of benzo(a)pyrene to surface water (dugout)

$$L_r = 6.9 \times 7.97E+05 \times \frac{3.45E-05 \times 1.5}{0.2 + 3154.8 \times 1.5} \times 10$$

$$L_r = 6.0E-01\ mg/yr$$

Example 20 Runoff load of benzo(a)pyrene to surface water (lake)

$$L_r = 6.9 \times 2.90E+08 \times \frac{3.45E-05 \times 1.5}{0.2 + 3154.8 \times 1.5} \times 10$$

$$L_r = 2.19E+02\ mg/yr$$

D3.3 Soil Erosion

The following equation is recommended by US EPA OSW (2005) to predict the soil erosion load of COPC to a water body from pervious soil surfaces within the watershed:

$$L_e = X_e \times A_p \times SD \times ER \times \frac{C_s \times K_d \times BD}{\theta_{sw} + K_d \times BD}$$

Where:

L_e	=	soil erosion load (mg/yr)
X_e	=	unit soil loss (kg/m^2/yr)
A_p	=	pervious watershed area (m^2)
SD	=	sediment delivery ratio (Unitless)
ER	=	soil enrichment ratio (Unitless)
C_s	=	soil concentration (mg/kg)
BD	=	bulk density of soil (g/cm^3)
O_{sw}	=	soil volumetric water content (ml-water / cm^3-soil)
K_d	=	soil-water partition coefficient (cm^3-water / g-soil or L/kg)

Example 21 Soil erosion load of benzo(a)pyrene to surface water (dugout)

$$L_e = 8.92E-01 \times 7.97E+05 \times 0.384 \times 3 \times \frac{3.45E-05 \times 3154.8 \times 1.5}{0.2 + 3154.8 \times 1.5}$$

$$L_e = 28.28 mg/yr$$

Example 22 Soil erosion load of benzo(a)pyrene to surface water (lake)

$$L_e = 8.92E-01 \times 2.9E+08 \times 0.105 \times 3 \times \frac{3.45E-05 \times 3154.8 \times 1.5}{0.2 + 3154.8 \times 1.5}$$

$$L_e = 2.81E+03\ mg/yr$$

D3.4 Total COPC Loading to Surface Water

Total water body load was based on the following equation:

$$L_t = L_d + L_r + L_e$$

Where:

L_t	=	total COPC water load (mg/yr)
L_d	=	deposition loading onto surface water (mg/yr)
L_r	=	runoff load (mg/yr)
L_e	=	erosion load (mg/yr)

Under certain circumstances, the predicted total COPC load to the water body exceeds the mass of COPC that was deposited from the atmosphere, which is not possible. Therefore, in these circumstances the model defaulted to the total mass deposited from the atmosphere as the COPC input load to the aquatic system.

Example 23 Total loading of benzo(a)pyrene to surface water (dugout)

$$L_t = 4.67E+00 + 6.02E-02 + 2.83E+01$$

$$L_t = 3.36E+02\ mg/yr$$

Example 24 Total loading of benzo(a)pyrene to surface water (lake)

$$L_t = 1.92E+03 + 2.19E+02 + 2.81E+03$$

$$L_t = 4.96E+03\ mg/yr$$

D3.5 Predicted Surface Water Concentration

The predicted surface water concentration was based on the following equation (US EPA OSW 2005):

$$C_{sw} = \frac{L_t}{V_f \times F_{wc} + k_{sw} \times V_{sw}} \times CF$$

Where:

C_{sw}	=	water body COPC concentration (mg/L)
L_t	=	total COPC load to the water body (g/yr)
V_f	=	average volumetric flow rate through the water body (m^3/yr)
F_{wc}	=	fraction of water body COPC concentration in the water column (Unitless)
k_{sw}	=	COPC dissipation rate constant (yr^{-1})
V_{sw}	=	volume of water body (m^3)
CF	=	conversion factor 0.001 (m^3/L)

Example 25 Concentration of benzo(a)pyrene in surface water for the prediction ecological exposure (dugout)

$$C_{sw} = \frac{3.36E+01}{3.16E+03 \times 1 + 3.57 \times 3.16E+03} \times 0.001$$

$$C_{sw} = 2.33E-06 mg/L$$

Example 26 Concentration of benzo(a)pyrene in surface water for the prediction ecological exposure (lake)

$$C_{sw} = \frac{4.96E+03}{9.78E+05 \times 1 + 3.57 \times 1.33E+08} \times 0.001$$

$$C_{sw} = 1.04E-8 mg/L$$

Example 27 Concentration of benzo(a)pyrene in surface water for the prediction of human exposure (dugout and lake)

Same calculation as above

D4.0 CHEMICAL CONCENTRATIONS IN DUST

The chemical concentrations in dust were calculated using the measured and/or predicted soil concentration, as follows (Health Canada 2012):

$$C_{dust} = DL \times C_s \times CF$$

Where:

C_{dust}	=	chemical concentration in dust (µg/m³)
DL	=	dust level (kg/m³)
C_s	=	surface soil concentration from deposition over time (mg/kg)
CF	=	conversion factor from mg to µg (1,000 µg/mg)

A dust level of 250 µg/m³ (2.5E-07 kg/m³) was recommended by Health Canada (2012) for areas with significant vehicle traffic on unpaved roads based on the average airborne concentration of respirable particulate matter (<10 µm aerodynamic diameter).

Example 17 Concentration of benzo(a)pyrene in dust for the prediction of ecological exposure

$$C_{dust} = 2.50E-07 \times 3.45E-04 \times 1{,}000$$

$$C_{dust} = 8.63E-08 \mu g/m^3$$

Example 18 Concentration of benzo(a)pyrene in dust for prediction of human exposure

$$C_{dust} = 2.50E-07 \times 1.25E-06 \times 1{,}000$$

$$C_{dust} = 3.14E-10 \mu g/m^3$$

D5.0 CHEMICAL CONCENTRATIONS IN PLANTS

The methodology used to estimate the contribution from each route of the chemical uptake in plants are described in the following sections. The following mechanisms were included when estimating the uptake of the chemicals into the tissue of plants.

- air to above-ground plants (particle deposition to leaves or foliage)

- air to above-ground plants (vapour transfer to leaves or foliage)
- soil to above-ground plants (root uptake)
- soil to below-ground plants (root uptake)

The worked example is provided for forage; however, Table 1 presents the input parameters that were used for the remaining plant groups included in the ecological and HHRA models. The current assessment did not adjust concentrations in plants for human consumption with a washing and peeling factor to account for potential reduction in exposures where washing or peeling occurs.

Table 1
Input Parameters for Predicting Plant Concentrations [(a)]

Parameter	Abbreviation	Forage	Plants	Berries	Lab Tea	Root	Wild roots
Fraction volatile for benzo(a)pyrene [%]	Fv	30	30	30	30	na	na
Intercept fraction [unitless]	R_p	0.5	0.39	0.39	0.39	na	na
Plant surface loss coefficient [yr^{-1}]	k_p	18	18	18	18	na	na
Length of plant exposure [year]	T_p	0.12	0.16	0.16	0.16	na	na
Yield or productivity [kg DW/m²]	Y_p	0.24	2.24	2.24	2.24	na	na
Moisture content [%]	MC	85	85	85	85	85	85
Reduction factor	RF	100	100	100	100	na	na
Empirical correction factor [unitless]	VG_{ag}	na	0.01	0.01	0.01	na	na

(a) Parameter values derived from US EPA OSW (2005) unless noted otherwise.
(b) na: not applicable

D5.1 Plant Concentrations as a Result of Direct Deposition

The following equation was used to predict concentrations of forage for consumption by ecological receptors as a result of deposition processes on a dry weight (DW) basis (US EPA OSW 2005):

$$Pd = \frac{\left[D_d + (D_w \times 0.6)\right] \times Rp \times \left[1.0 - \exp(-kp \times Tp)\right]}{Yp \times kp}$$

Where:

Pd	=	forage concentration as a result of direct deposition (mg/kg DW)
D_d	=	dry deposition, particle fraction = (mg/m²/yr)
D_w	=	wet deposition, particle fraction = (mg/m²/yr)
Fv	=	fraction that is volatile (%)
Rp	=	intercept fraction of edible portions of plant (unitless)
kp	=	plant surface loss coefficient (yr^{-1})
Tp	=	length of plant exposure to deposition per harvest of the edible portion of the ith plant group (yr)
Yp	=	yield or productivity (kg DW/m²)

The US EPA OSW (2005) recommends the use of the default intercept fraction of edible portions of plant (*Rp*) value (unitless), because it represents the most current information available with respect to productivity and relative ingestion rates. A default *Rp* value of 0.5 was recommended for forage.

The *kp* value is a measure of the amount of chemical lost as a result of removal by wind and water and growth dilution. The US EPA OSW (2005) recommends a default *kp* value of 18 yr^{-1} for forage, which corresponds to a 14-day half-life.

The US EPA OSW (2005) recommends using a *Yp* value of 0.24 kg DW/m² for forage.

Example 19 Concentration of benzo(a)pyrene in forage as a result of direct deposition for prediction of ecological exposures

$$Pd = \frac{[2.88E-03 + (1.23E-03 \times 0.6)] \times 0.5 \times [1.0 - \exp(-18 \times 0.12)]}{0.24 \times 18}$$

$$Pd = 3.70E-04\ mg/kg\ DW$$

D5.2 Plant Concentrations as A Result of Vapour Uptake

The concentration of chemicals in forage from direct vapour uptake was calculated using a mass-based air-to-plant biotransfer factor, which was derived from the volumetric air-to-plant biotransfer factor (US EPA OSW 2005).

Volumetric air-to-plant biotransfer factor

$$\log B_{vol} = 1.065 \times \log K_{ow} - \log\left(\frac{H}{R \times T}\right) - 1.654$$

Where:

B_{vol}	=	volumetric air-to-plant biotransfer factor (unitless; WW basis)
$log\ K_{ow}$	=	log of the octanol-water partition coefficient (unitless)
H	=	Henry's Law constant of the compound (atm m³/mol)
R	=	gas constant (0.000082 atm m³/mol)
T	=	room temperature (K)

Example 20 Volumetric air-to-plant biotransfer factor of benzo(a)pyrene

$$\log B_{vol} = 1.065 \times 6.13 - \log\left(\frac{4.57E-07}{8.2E-05 \times 288}\right) - 1.654$$

$$B_{vol} = 3.87E+09$$

Mass-based air to plant biotransfer factor:

$$B_v = \frac{P_{air} \times B_{vol}}{(1 - WC) \times P_{forage}}$$

Where:

B_v	=	mass-based air-to-plant biotransfer factor ([µg/g DW plant] / [µg/g air])
P_{air}	=	density of air (1.19 g/L; Weast 1981)
B_{vol}	=	volumetric air-to-plant biotransfer factor (unitless; WW basis)
WC	=	water or moisture content of plant (%)
P_{forage}	=	density of forage (770 g/L; McCrady and Maggard 1993)

Example 21 Mass-based air-to-plant biotransfer factor for benzo(a)pyrene in forage for the prediction of ecological exposures

$$B_v = \frac{1.19 \times 3.87E+09}{(1 - 0.85) \times 770}$$

$$B_v = 3.99E+07 \left[\mu g / g\ DW\ \ plant\right] / \left[\mu g / g\ air\right]$$

D5.3 Concentrations in Above-ground Forage Consumed by Ecological Receptors

The following equation was used to calculate above-ground plant concentrations as a result of vapour uptake (US EPA OSW 2005):

$$Pv = \frac{C_{air} \times (B_v / RF) \times F_v \times VG_{ag}}{\rho_{air}}$$

Where:

Pv	=	COPC concentration in forage as a result of vapour uptake (mg/kg DW)
C_{air}	=	COPC concentration in air (µg/m³)
B_v	=	mass-based air-to-plant biotransfer factor ([µg/g DW plant] / [µg/g air])
RF	=	reduction factor (unitless)
VG_{ag}	=	empirical correction factor (unitless)
F_v	=	fraction of chemical in vapour phase
P_{air}	=	density of air (1,200 g/m³; Weast 1981)

As recommended by the US EPA OSW (2005), the biotransfer factor for organics (except dioxins and furans) should be reduced by a factor of 100. In addition the US EPA OSW (2005) also recommends an empirical correction factor (*i.e.*, VG_{ag}) of 0.01 for COPC with a log K_{ow} greater than 4 and an empirical correction factor of 1 for COPC with a log K_{ow} less than 4. As recommended by US EPA OSW (2005) this additional empirical correction factor was not applied to the exposure pathways for ingestion of forage by ecological receptors, but was applied to the exposure pathway for ingestion of above ground plants for the human exposure assessment. A conversion from dry weight to wet weight (1 - WC) was also made to calculated concentrations in garden produce.

Example 22 Concentration of benzo(a)pyrene in forage as a result of vapour uptake for the prediction of ecological exposure

$$Pv = \frac{1.71E-05 \times (3.99E+07/100) \times 0.3 \times 1}{1{,}200}$$

$$Pv = 1.71E-03\ mg/kg\ DW$$

D5.4 Plant Concentrations as a Result of Root Uptake

COPC present in soil can be taken up into edible portions of plants. The US EPA OSW (2005) provides an equation to predict above-ground plant concentrations as a result of root uptake using soil concentrations and plant-to-soil bioconcentration factors (BCFs).

D5.4.1 Soil to Plant Bioconcentration Factor

The soil-to-plant BCFs were calculated based on the following equation recommended by the US EPA OSW (2005), adopted from Travis and Arms (1988) for organic compounds:

$$\log BCF = 1.588 - 0.578 \times \log K_{ow}$$

Where:

BCF	=	plant-soil bioconcentration factor (kg soil/kg plant DW)
log K_{ow}	=	log of the octanol-water partition coefficient (unitless)

The above equation was derived from experiments conducted on compounds with log K_{ow} values ranging from 1.15 to 9.35. Thus, BCF values for compounds with a log K_{ow} value less than 1.15 should be calculated using a log K_{ow} value of 1.15 and BCF values for compounds with a log K_{ow} greater than 9.35 should be calculated using a log K_{ow} value of 9.35 (US EPA OSW 2005).

A K_{ow} value of 1348962.9 or log K_{ow} value of 6.13 was used for benzo(a)pyrene, as the log K_{ow} value of 6.13 is within the range of log K_{ow} 1.15 to 9.35.

Example 23 Plant-to-soil bioconcentration factor for benzo(a)pyrene

$$\log BCF = 1.588 - 0.578 \times 6.13$$

$$BCF = 10\text{^}(1.588 - 0.578 \times 6.13)$$

$$BCF = 1.11E-02\ kg\ soil/kg\ plant\ DW$$

D5.5 Concentrations in Forage Consumed by Ecological Receptors

The following equation was used to predict the chemical concentration in above-ground forage as a result of root uptake (US EPA OSW 2005).

$$\mathrm{Pr} = C_s \times BCF$$

Where:

Pr	=	chemical concentration in above-ground plant as a result of root uptake (mg/kg DW)
C_s	=	chemical concentration in soil (mg/kg)
BCF	=	plant-soil bioconcentration factor for above-ground produce (kg soil/kg plant DW)

Example 24 Concentration of benzo(a)pyrene in forage as a result of root uptake for the prediction of ecological exposure

$$\mathrm{Pr} = 3.45E-05 \times 1.11E-02$$

$$\mathrm{Pr} = 3.83E-07\ mg/kg\ DW$$

D5.6 Total Chemical Concentration in Plants

The following equation was used to estimate the chemical concentration in above ground forage as a result of direct deposition, vapour uptake, and root uptake.

$$C_{plant} = \left(Pd + Pv + \mathrm{Pr}\right)$$

Where:

C_{plant}	=	total chemical concentration in plant (mg/kg).
Pd	=	plant concentration as a result of direct deposition (mg/kg)
Pv	=	COPC concentration in plant as a result of vapour uptake (mg/kg)
Pr	=	chemical concentration in above-ground plants as a result of root uptake (mg/kg)

Example 25 Concentration of benzo(a)pyrene in above-ground forage as a result of direct deposition, vapour uptake and root uptake for the prediction of ecological exposure

$$C_{plant} = \left(3.70E-04 + 1.71E-03 + 3.83E-07\right)$$

$$C_{plant} = 2.08E-03\ mg/kg$$

The same methods were used to predict chemical concentrations in above ground plants consumed by humans. However, the input parameters vary from that used in the prediction of chemical concentrations in forage (see Table 1) and the air concentrations used to predict the plant concentrations were community-specific, as opposed to the use of the MPOI air concentrations (as was completed for the prediction of forage concentrations). The following equation was used to estimate the chemical concentration in above ground plants in the Oil and Gas Scenario at the community of Arras as a result of direct deposition, vapour uptake, and root uptake.

Example 32 Concentration of benzo(a)pyrene in above-ground plants as a result of direct deposition, vapour uptake and root uptake for the prediction of human exposure

$$C_{plant} = (1.80E-08 + 9.31E-09 + 2.09E-10)$$

$$C_{plant} = 2.75E-08\ mg/kg$$

D5.7 Below-Ground Plant Concentrations as a Result of Root Uptake

Chemicals present in soil also can be taken up into edible portions of below-ground produce (*i.e.*, root vegetables). The US EPA OSW (2005) provides an equation to predict below-ground plant concentrations as a result of root uptake using soil concentrations and plant-to-soil BCFs in root vegetables. The below-ground produce concentration for root vegetables was calculated as follows (US EPA OSW 2005):

$$\mathrm{Pr}_{root} = C_s \times BCF \times WPF \times (1 - WC)$$

Where:

Pr_{root}	=	chemical concentration in below-ground produce as a result of root uptake (mg/kg WW)
C_s	=	chemical concentration in soil (mg/kg)
BCF	=	plant-to-soil bioconcentration factor for below-ground plants (kg soil/kg plant DW)
WPF	=	washing and peeling factor (unitless)
WC	=	water or moisture content of root vegetables (85%, US EPA OSW 2005)

Example 33 Concentration of benzo(a)pyrene in root vegetables as a result of root uptake for the prediction of human exposure

$$\mathrm{Pr}_{root} = 1.25E-07 \times 3.87E+00 \times 1.0 \times (1 - 0.85)$$

$$\mathrm{Pr}_{root} = 7.27E-08\ mg/kg\ WW$$

D6.0 CHEMICAL CONCENTRATIONS IN AQUATIC PLANTS

The chemical concentration in aquatic plants was derived using surface water concentrations and water-to-aquatic plant BCFs. Aquatic plant concentrations were predicted only for the calculation of ecological exposures. The water-to-aquatic plant BCFs were provided by the US EPA OSW (1999) or US EPA (2011).The following equation was used to predict the chemical concentration in aquatic plants:

$$C_{aqplant} = C_{sw} \times BCF$$

Where:

$C_{aqplant}$	=	chemical concentration in aquatic plants (mg/kg DW)
C_{sw}	=	chemical concentration in surface water (mg/L)
BCF	=	water-to-aquatic plant bioconcentration factor (L water/kg plant DW)

Example 26 Concentration of benzo(a)pyrene in aquatic plants for the prediction of ecological exposure

$$C_{aqplant} = 1.04E-08 \times 1.54E+04$$

$$C_{aqplant} = 1.60E-04\ mg/kg\ DW$$

D7.0 CHEMICAL CONCENTRATIONS IN TERRESTRIAL INVERTEBRATES

The chemical concentration in terrestrial invertebrates was derived using soil concentrations and soil-to-soil invertebrate BCFs. Terrestrial invertebrate concentrations were predicted only for the calculation of ecological exposures. The soil-to-soil invertebrate BCFs were provided by the US EPA OSW (1999).

The following equation was used to predict the chemical concentration in terrestrial invertebrates:

$$C_{invert} = C_s \times BCF$$

Where:

C_{invert}	=	chemical concentration in invertebrates (mg/kg DW)
C_s	=	chemical concentration in soil (mg/kg)
BCF	=	soil-to-soil invertebrate bioconcentration factor (kg soil/kg invertebrate DW)

Example 27 Concentration of benzo(a)pyrene in terrestrial invertebrates for the prediction of ecological exposure

$$C_{invert} = 3.45E-05 \times 0.4193$$

$$C_{invert} = 1.45E-05 mg/kg\ DW$$

D8.0 CHEMICAL CONCENTRATIONS IN FISH

Fish concentrations were predicted only for the calculation of human exposure. The BCF value for benzo(a)pyrene was provided by US EPA OSW (2005).

The following equation was used to predict the chemical concentration in fish:

$$C_{fish} = C_{sw} \times BCF$$

Where:

C_{fish}	=	chemical concentration in fish (mg/kg WW)
C_{sw}	=	chemical concentration in surface water (lake) (mg/L)
BCF	=	surface water-to-fish bioconcentration factor (L water/kg fish WW)

Example 28 Concentration of benzo(a)pyrene in fish for the prediction of human exposure

$$C_{fish} = 1.04E-08 \times 55$$

$$C_{fish} = 5.74E-07\ mg/kg\ WW$$

D9.0 ECOLOGICAL EXPOSURE CALCULATIONS

Tissue concentrations were calculated following the US EPA OSW (2005) methodology. To estimate tissue concentrations, ecological species were assumed to be exposed to chemicals through consumption or exposure to dust, soil, water and food. The following sections provide the equations used to calculate the total daily dose of a chemical via the individual exposure pathways for wildlife (moose, grouse and snowshoe hare) or agricultural animals (cattle and chickens) and the corresponding tissue concentrations (meat, milk, eggs). The following example calculation is for beef cattle.

D9.1 Food Ingestion Rates

The food ingestion rate is influenced by a number of factors, such as the metabolic rate and composition of the diet. The rate of food consumption that an animal must achieve to meet its metabolic needs can be calculated by dividing its free-living (or field) metabolic rate (FMR) by the metabolizable energy in its food (US EPA 1993; Nagy 1987).

D9.2 Metabolizable Energy

Metabolizable energy (ME) is the gross energy (GE) in a unit of food consumed minus the energy lost in feces and urine (US EPA 1993). Assimilation efficiency (AE) equals the ratio of metabolizable energy to gross energy, or the fraction of gross energy that is metabolizable (US EPA 1993). Thus, the metabolizable energy for dietary items can be calculated as follows:

$$ME = GE \times AE$$

Where:

ME	=	metabolizable energy of dietary item (kcal/kg)
GE	=	gross energy of dietary item (kcal/kg DW)
AE	=	assimilation efficiency of dietary item (%)

The assimilation efficiency and gross energy values for the different dietary items were provided by the US EPA (1993).

Example 29 Metabolizable energy of forage for beef cattle

$$ME = 4{,}200 \times 0.41$$

$$ME = 1.72E + 03\ kcal/kg$$

D9.2.1 Free-Living Metabolic Rate (Normalized)

Nagy (1987) provides allometric equations to estimate FMRs based on doubly-labelled water measurements of CO_2 production in free-living animals (US EPA 1993). The equations provided by Nagy (1987) are based on the following formula:

$$FMR = \frac{a \times BW^{b}}{4.184\ kJ/kcal}$$

Where:

FMR	=	free-living metabolic rate (kcal/d)
a	=	slope of the allometric equation for the FMR (unitless)
BW	=	body weight (g)
b	=	y-intercept of the allometric equation for the FMR (unitless)

Nagy *et al.* (1999) provide a number of slope and y-intercept values for FMRs specific to orders and trophic levels (*e.g.*, rodentia, galliformes, and herbivores). These values were used to estimate the FMR values for each species. Note: The equation used to calculate the FMR for moose does not require the conversion to kcal units; thus the conversion factor of 4.184 kJ/kcal is not needed. However, the conversion factor of 4.184 kJ/kcal is needed in the calculation of the FMR for grouse and snowshoe hare.

Example 30 Free-living metabolic rate for beef cattle

$$FMR = 7.94 \times 6.4E + 05^{0.646}$$

$$FMR = 4.47E + 04\ kcal/d$$

To normalize the FMR to body weight, the FMR was divided by the body weight of the species:

$$NFMR = \frac{FMR}{BW}$$

Where:

NFMR	=	normalized free-living metabolic rate (kcal/kg bw/d)
FMR	=	free-living metabolic rate (kcal/d)
BW	=	body weight (kg)

Example 31 Normalized free-living metabolic rate for beef cattle

$$NFMR = \frac{4.47E+04}{640}$$

$$NFMR = 6.99E+01\ kcal/kg\ bw/d$$

D9.3 Soil Ingestion Rates

The soil ingestion rates were calculated as a percentage of the total estimated food ingestion rate for all dietary items. The percentage of soil in the diet for each of the ecological species was obtained from the US EPA OSW (2005) and/or Suter *et al.* (2000).

The soil ingestion rates were calculated as follows:

$$SIR = P_{soil} \times FIR_{total}$$

Where:

SIR	=	soil ingestion rate (mg/d)
P_{soil}	=	percent of soil in diet (%)
FIR_{total}	=	total food ingestion rate of chemical for all dietary items (mg /d)

Example 32 Soil ingestion rate for beef cattle

$$SIR = 0.045 \times 2.6E+01$$

$$SIR = 1200\ mg/d$$

D10.0 ESTIMATED DAILY INTAKE OF CHEMICALS IN ECOLOGICAL RECEPTORS VIA ALL MEDIA

D10.1 Soil Ingestion

The estimated daily intake of a chemical through incidental ingestion of soil by ecological receptors was calculated by applying the soil ingestion rate to the chemical concentration in the soil.

$$EDI_{soil} = C_s \times SIR$$

Where:

EDI_{soil}	=	estimated daily intake of chemical in soil (mg/d)
C_s	=	chemical concentration in surface soil (mg/kg)
SIR	=	soil ingestion rate (mg/d)

Example 33 Estimated daily intake of benzo(a)pyrene from ingestion of soil by beef cattle

$$EDI_{soil} = 3.45E-04 \times 1200$$

$$EDI_{soil} = 4.04E-04\ mg/d$$

D10.2 Food Ingestion

The estimated daily intake of a chemical through ingestion of food (*i.e.*, invertebrates, forage and aquatic plants) by ecological receptors for each dietary item was calculated as follows:

$$EDI_i = \frac{FMR \times P_i \times C_i}{ME_i}$$

Where:

EDI_i	=	estimated daily intake of a chemical in the 'i' dietary item (mg/d)
FMR	=	free-living metabolic rate (kcal/d)
P_i	=	portion of diet consisting of 'i' dietary item (%)
C_i	=	concentration of 'i' chemical in 'i' dietary item (mg/kg)
ME_i	=	metabolizable energy of 'i' dietary item (kcal/kg)

Beef were assumed to consume a diet consisting of 100% forage.

Example 34 Estimated forage ingestion for beef

$$EDI_{browse} = \frac{4.47E+04 \times 1.0 \times 2.08E-03}{1{,}722}$$

$$EDI_{browse} = 5.4E-02 mg/d$$

The total estimated daily intake of a chemical from all dietary items was estimated by summing the individual EDIs for each dietary item:

$$EDI_{diet} = EDI_{invert} + EDI_{browse} + EDI_{aqplant}$$

Where:

EDI_{diet}	=	estimated daily intake of chemical for all dietary items (mg /d)
EDI_{invert}	=	estimated daily intake of chemical from ingestion of terrestrial invertebrates (mg/d)
EDI_{browse}	=	estimated daily intake of a chemical from ingestion of browse (*i.e.* forage) (mg/d)
$EDI_{aqplant}$	=	estimated daily intake of a chemical from ingestion of aquatic plants (mg/d)

Example 35 Total estimated daily intake of chemical from diet for moose

$$EDI_{diet} = 0 + 5.4E - 02 + 0$$

$$EDI_{diet} = 5.4E - 02\ mg/d$$

D10.3 Ingestion of Water

The estimated daily intake of a chemical through ingestion of surface water by ecological receptors was calculated by applying the water ingestion rate to either the lake (wildlife) or dugout (agricultural animals).

$$EDI_{water} = C_{sw} \times WIR$$

Where:

EDI_{water}	=	estimated daily intake of chemical in surface water (mg/d)
C_{sw}	=	chemical concentration in surface water (mg/L)
WIR	=	water ingestion rate (L/d)

Example 36 Estimated daily intake of benzo(a)pyrene from consumption of surface water (dugout) by beef

$$EDI_{water} = 2.33E - 06 \times 53$$

$$EDI_{water} = 1.23E - 04\ mg/d$$

D10.4 Ingestion of Dust

The air inhalation rate for ecological receptors was predicted using allometric equations for birds and mammals, as provided by the US EPA (1993).

D10.4.1 Inhalation rate for birds:

$$AIR = 0.4089 \times BW^{0.77}$$

D10.4.2 Inhalation rate for mammals:

$$AIR = 0.5458 \times BW^{0.80}$$

Where:

AIR	=	predicted air inhalation rate (m^3/d)
BW	=	body weight (kg)

Example 37 Predicted inhalation rate for beef cattle

$$AIR = 0.5458 \times 640^{0.80}$$

$$AIR = 9.6E+01\ m^3 / d$$

The estimated daily intake of a chemical through inhalation of predicted ground-level dust concentrations by beef cattle was calculated by applying the air inhalation rate to the predicted dust concentration.

$$EDI_{inh} = C_{dust} \times AIR \times CF$$

Where:

EDI_{inh}	=	estimated daily intake of chemical via inhalation (mg/d)
C_{dust}	=	chemical concentration in dust (µg/m³)
AIR	=	air inhalation rate (m³/d)
CF	=	conversion factor from µg to mg (0.001 mg/µg)

Example 38 Estimated daily intake of benzo(a)pyrene by beef cattle via inhalation

$$EDI_{inh} = 8.63E-08 \times 9.6E+01 \times 0.001$$

$$EDI_{inh} = 8.28E-09 mg / d$$

D10.5 Estimated Total Daily Intake

The estimated daily intake for wildlife or agricultural animals from all potential pathways of exposure was calculated as follows:

$$EDI_{total} = EDI_{soil} + EDI_{browse} + EDI_{aqplant} + EDI_{invert} + EDI_{water} + EDI_{inh}$$

Where:

EDI_{total}	=	total estimated daily intake of chemical via all routes of exposure (mg/d)
EDI_{soil}	=	estimated daily intake of chemical from ingestion of soil (mg/d)
EDI_{browse}	=	estimated daily intake of chemical from consumption of browse (mg/d)
$EDI_{aqplant}$	=	estimated daily intake of chemical from consumption of aquatic plants (mg/d)
EDI_{invert}	=	estimated daily intake of chemical from consumption of invertebrates (mg/d)
EDI_{water}	=	estimated daily intake of chemical from ingestion of water (mg/d)
EDI_{inh}	=	estimated daily intake of chemical from inhalation of air (mg/d)

Example 39 Total estimated daily intake of benzo(a)pyrene from all routes of exposure for beef cattle

$$EDI_{total} = 4.04E-04 + 5.40E-02 + 0 + 0 + 1.23E-04 + 8.28E-09$$

$$EDI_{total}\ 5.45E-02 mg/d$$

D11.0 ANIMAL TISSUE CONCENTRATIONS

D11.1 Biotransfer Factors

Biotransfer factors (BTFs) are used to translate an estimated dose of a chemical to a tissue concentration. Biotransfer models have been developed by the Research Triangle Institute (RTI 2005) and were incorporated within the current assessment, as recommended by the US EPA OSW (2005) for organic chemicals. The following equation was used to predict the transfer rate of the chemical intake into fat tissue (US EPA OSW 2005).

$$\log BTF = -0.099 \times \log K_{ow}{}^{2} + 1.07 \times \log K_{ow} - 3.56$$

Where:

BTF	=	biotransfer factor ([mg/kg fat] / [mg/d])
log K_{ow}	=	log of the octanol-water partition coefficient (unitless)

The BTF equation is appropriate for organic chemicals lacking empirical biotransfer data and having a log K_{ow} between -0.67 and 8.2.

Example 40 Biotransfer factor for benzo(a)pyrene

$$\log BTF = -0.099 \times 6.13^{2} + 1.07 \times 6.13 - 3.56$$

$$BTF = 1.90E-01\ [mg/kg\ fat]/[mg/d]$$

D11.2 Adjusted Biotransfer Factors

The fat tissue concentration can be converted to a tissue concentration by adjusting the BTF with the fat content of desired tissue (*e.g.*, moose, grouse, snowshoe hare, beef cattle, dairy milk, eggs). The fat content for wild game and agricultural food was assumed to be:

- 19% for beef, moose and snowshoe hare, (US EPA OSW 2005);
- 14% for chicken and ruffed grouse (US EPA OSW 2005);
- 8% for chicken egg (US EPA OSW 2005); and
- 4% for dairy (US EPA OSW 2005).

The BTF was adjusted to account for the amount of fat in the tissue based on the following equation (US EPA OSW 2005):

$$BTF_a = BTF \times FC$$

Where:

BTF_a	=	adjusted biotransfer factor for fat content of tissue ([mg/kg tissue] / [mg/d])
BTF	=	biotransfer factor ([mg/kg fat] / [mg/d])
FC	=	fat content of tissue (%)

Example 41 Adjusted biotransfer factor for benzo(a)pyrene for fat content of meat in beef

$$BTF_a = 1.90E-01 \times 0.19$$

$$BTF_a = 3.61E-02$$

D11.3 Metabolism Factors

As provided in the methodology for predicting cattle BTFs (RTI 2005, US EPA OSW 2005), the equation that is used to estimate BTF values might overestimate biotransfer of highly metabolized chemicals. The dataset used to estimate the polynomial relationship between BTFs and the K_{ow} is based on anthropogenic chemicals that are persistent (*e.g.*, pesticides) and can potentially biomagnify (*e.g.*, pesticides, polychlorinated biphenyls (PCBs), dioxins, and furans). Polycyclic aromatic hydrocarbons (PAHs) were not included in the dataset used to develop the empirical relationship and were identified as potentially highly metabolized chemicals by mammals. Depending on the compound, lipophilicity or K_{ow} measures are not always a good predictor of tissue concentrations (Hofelt *et al.* 2001).

Evidence strongly suggests that PAHs are extensively metabolized and eliminated. Ramesh *et al.* (2004), Laurent *et al.* (2001; 2002), and Grova *et al.* (2002) investigated the transfer of PAHs in the food chain to goats and pigs. Their studies demonstrate that PAHs are poorly absorbed from diet or readily metabolized and excreted. Hofelt *et al.* (2001) overcame these limitations for human health assessment by deriving PAH metabolism factors (MF) for use in multipathway hazard assessments. MF values reported for some PAHs are provided in Table 2. The MF values are derived for use with diverse matrices such as milk, beef, chicken, eggs, and pork (Ramesh *et al.* 2004).

Table 2
Metabolism Factors for PAHs

Chemical	Animal Model	Metabolism Factor (MF)
Benz(a)anthracene	Rat	0.001
Benzo(a)pyrene	Mouse	0.004
Pyrene	Rat	0.003

Hofelt *et al.* (2001) recommends a MF of 0.01 for PAHs. The MF is applied to the adjusted BTF for fat content of tissue to derive an adjusted BTF for metabolism, as follows:

$$BTF_{adj} = BTF_a \times MF$$

Where:

BTF_{adj}	=	adjusted biotransfer factor for metabolism ([mg/kg tissue] / [mg/d])
BTF_a	=	adjusted biotransfer factor for fat content of tissue ([mg/kg tissue] / [mg/d])
MF	=	metabolism factor (PAHs=0.01, VOCs=1.0, unitless)

Example 42 Adjusted biotransfer factor for benzo(a)pyrene metabolism in beef cattle

$$BTF_{adj} = 3.61E-02 \times 0.01$$

$$BTF_{adj} = 3.61E-04\ [mg/kg\ tissue]/[mg/d]$$

D11.4 Tissue Concentrations

Chemical concentrations in animal meat were predicted based on the following equation (US EPA OSW 2005):

$$C_{animal} = BTF_{adj} \times EDI_{total}$$

Where:

C_{animal}	=	chemical concentration in game meat (mg/kg WW)
BTF_{adj}	=	adjusted biotransfer factor for metabolism ([mg/kg tissue] / [mg/d])
EDI_{total}	=	total estimated daily intake of chemical via all routes of exposure (mg/d)

Example 43 Predicted concentration of benzo(a)pyrene in beef

$$C_{moose} = 3.61E-04 \times 5.45E-02$$

$$C_{moose} = 1.97E-05 mg/kg\ WW$$

Similar methods were applied to the calculation of agricultural and game meat concentrations.

D12.0 HUMAN EXPOSURE ESTIMATES

D12.1 Ingestion of Soil (Incidental)

The following equation was used to estimate human exposure via incidental ingestion of soil. Soil ingestion rates and equations used to predict exposures were based on recommendations from Health Canada (2012).

$$EDI_{soil} = C_s \times SIR \times CF1 \times CF2$$

Where:

EDI_{soil}	=	estimated daily intake of chemical via ingestion of soil (µg/d)
C_s	=	chemical concentration in surface soil (mg/kg)
SIR	=	incidental soil ingestion rate (g/d)
CF1	=	conversion factor from mg to µg (1,000 µg/mg)
CF2	=	conversion factor from g to kg (0.001 kg/g)

Example 44 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from incidental ingestion of soil

$$EDI_{soil} = 1.25E-06 \times 0.08 \times 1000 \times 0.001$$

$$EDI_{soil} = 1.00E-07 \mu g/d$$

D12.2 Ingestion of Drinking Water

It was assumed that agricultural residents consumed water from dugouts, and community residents and Aboriginals consumed surface water from a lake (with Charlie Lake being a surrogate). Water ingestion rates and equations used to predict exposures were based on recommendations from Health Canada (2012) and exposures were based on the following equation:

$$EDI_{water} = C_{dw} \times WIR \times CF$$

Where:

EDI_{water}	=	estimated daily intake of chemical via ingestion of water (µg/d)
C_{dw}	=	chemical concentration in drinking water (mg/L)
WIR	=	water ingestion rate (L/d)
CF	=	conversion factor from mg to µg (1,000 µg/mg)

Example 45 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from ingestion of dugout water

$$EDI_{water} = 2.33E-06 \times 0.6 \times 1{,}000$$

$$EDI_{water} = 1.40E-03 \mu g/d$$

D12.3 Inhalation/Ingestion of Dust

The following equation was used to estimate human exposure via inhalation / ingestion of dust. Air inhalation rates and equations used to predict exposures were based on recommendations from Health Canada (2012).

$$EDI_{dust} = C_{dust} \times AIR$$

Where:

EDI_{dust}	=	estimated daily intake of chemical via inhalation of dust (µg/d)
C_{dust}	=	chemical concentration in dust (µg/m³)
AIR	=	air inhalation rate (m³/d)

Example 46 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from inhalation of dust

$$EDI_{dust} = 3.14E-10 \times 8.3$$

$$EDI_{dust} = 2.60E-09\ \mu g/d$$

D12.4 Ingestion of Plants

D12.4.1 Leafy Vegetables

The following equation was used to estimate human exposure via consumption of leafy vegetables or wild leafy plants. Consumption rates and equations used to predict exposures were obtained from Health Canada (2012).

$$EDI_{plant} = C_{plant} \times IR_{plant}$$

Where:

EDI_{plant}	=	estimated daily intake of chemical via consumption of above-ground leafy plants (µg/d)
C_{plant}	=	total chemical concentration in leafy plant (mm/kg ww)
IR_{plant}	=	leafy plant ingestion rate (g/d)

Example 47 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of above-ground leafy plants

$$EDI_{plant} = 2.75E-08 \times 67$$

$$EDI_{plant} = 1.84E-06 \mu g/d$$

D12.4.2 Root Vegetables

The following equations were used to estimate human exposure via consumption of root vegetables. Consumption rates and equations used to predict exposures in the detailed HHRA were obtained from Health Canada (2012) and Chan *et al.* (2011).

The estimated exposure from consumption of root vegetables is (Health Canada 2012):

$$EDI_{root} = \mathrm{Pr}_{root} \times IR_{root}$$

Where:

EDI_{root}	=	estimated daily intake of chemical via consumption of root vegetables (µg/d)
Pr_{root}	=	chemical concentration in root vegetables from root uptake (mg/kg WW)
IR_{root}	=	root vegetable ingestion rate (g/d)

Example 48 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of root vegetables

$$EDI_{root} = 7.28E-08 \times 105$$

$$EDI_{root} = 7.64E-06\ \mu g/d$$

D12.4.3 Fruit and Wild Berries

Consumption rates and equations used to predict fruit exposures were obtained from Health Canada (2012) and Chan *et al.* (2011). The following equation was used to estimate human exposure via consumption of fruit and wild berries (Health Canada 2012).

$$EDI_{berry} = Pb \times IR_{berry}$$

Where:

EDI_{berry}	=	estimated daily intake of chemical via consumption of fruit and berries (µg/d)
Pb	=	chemical concentration in fruit and berries from root uptake (mg/kg WW)
IR_{berry}	=	fruit and berry ingestion rate (g/d)

Example 49 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of berries

$$EDI_{berry} = 2.75E-08 \times 40$$

$$EDI_{berry} = 1.10E-06\ \mu g/d$$

D12.4.4 Ingestion of Beef, Dairy, Chicken, Chicken Egg and Fish

Consumption rates and equations used to predict exposures were obtained from Health Canada (1994) and Health Canada (2007). The following equation was used to estimate human exposure via consumption of fish or wild game meat (Health Canada 2012).

$$EDI_{animal} = C_{animal} \times IR_{animal}$$

Where:

EDI_{animal}	=	estimated daily intake of chemical via consumption of fish or wild game (µg/d)
C_{animal}	=	chemical concentration in animal tissue (mg/kg WW)
IR_{animal}	=	fish or wild game ingestion rate (g/d)

Example 50 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of beef

$$EDI_{beef} = 1.97E-05 \times 39$$

$$EDI_{beef} = 7.68E-04 \mu g/d$$

Example 51 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of dairy

$$EDI_{dairy} = 3.96E-06 \times 677$$

$$EDI_{dairy} = 2.68E-03\ \mu g/d$$

Example 52 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of chicken

$$EDI_{chicken} = 4.28E-07 \times 13$$

$$EDI_{chicken} = 5.56E-06 \mu g/d$$

Example 53 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of chicken eggs

$$EDI_{chicken\_egg} = 2.45E-07 \times 24$$

$$EDI_{chicken\_egg} = 5.89E-06 \mu g/d$$

Example 54 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from consumption of fish

$$EDI_{fish} = 5.74E-07 \times 20$$

$$EDI_{fish} = 1.15E-05 \mu g/d$$

D12.5 Swimming Exposure Through Dermal and Ingestion Pathways

D12.5.1 Dermal Exposure to Surface Water

The following equation was used to estimate dermal exposure from swimming based on recommendations from US EPA (2004) and Health Canada (2012). The concentrations in the surface water were selected for estimating dermal exposure from swimming.

$$EDI_{derm+swim} = C_{sw} \times Kp \times SEF \times SAT \times CF1 \times CF2$$

Where:

$EDI_{derm+swim}$	=	estimated daily intake of chemical from dermal contact with surface water (µg/d)
C_{sw}	=	chemical concentration in surface water (mg/L)
Kp	=	dermal permeability coefficient in water (cm/hr)
SEF	=	swim exposure factor (hr/d)
SAT	=	surface area total (cm²)
CF1	=	conversion factor from mg to µg (1,000 µg/mg)
CF2	=	conversion factor from L to cm³ (0.001 L/cm³)

Example 55 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from dermal uptake during swimming

$$EDI_{derm+swim} = 1.04E-08 \times 6.83E-01 \times 0.255 \times 6130 \times 1{,}000 \times 0.001$$

$$EDI_{derm+swim} = 1.11E-05\ \mu g/d$$

D12.5.2 Incidental Ingestion of Surface Water During Swimming

The following equation was used to estimate ingestion exposure from swimming based on recommendations from US EPA (2004) and Health Canada (2012). The greater of the chemical concentrations in the lake and river media was selected for estimating ingestion exposure from swimming.

$$EDI_{ing+swim} = C_{sw} \times SEF \times SWIR \times CF1$$

Where:

$EDI_{ing+swim}$	=	estimated daily intake of chemical from ingestion of surface water during swimming (µg/d)
C_{sw}	=	chemical concentration in surface water (mg/L)
SEF	=	swim exposure factor (hr/d: 1hr/day x 90 days / 365 days)
SWIR	=	swimming ingestion rate (L/hr)
CF1	=	conversion factor from mg to µg (1,000 µg/mg)

Example 56 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from ingestion of surface water during swimming

$$EDI_{ing+swim} = 1.04E-08 \times 0.255 \times 0.05 \times 1{,}000$$

$$EDI_{ing+swim} = 1.33E-07\ \mu g/d$$

D12.5.3 Total Exposure to Surface Water During Swimming

The following equation was used to estimate total ingestion and dermal exposure from swimming.

$$EDI_{tot\_swim} = EDI_{derm+swim} + EDI_{ing+swim}$$

Where:

EDI_{tot_swim}	=	estimated daily intake of chemical from ingestion of and dermal contact with surface water during swimming (µg/d)
$EDI_{derm+swim}$	=	estimated daily intake of chemical from dermal contact with surface water during swimming (µg/d)
$EDI_{ing+swim}$	=	estimated daily intake of chemical from ingestion of surface water during swimming (µg/d)

Example 57 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from ingestion of and dermal contact with surface water during swimming

$$EDI_{tot\_swim} = 1.11E-05 + 1.33E-07$$

$$EDI_{tot\_swim} = 1.13E-05\ \mu g/d$$

D12.6 Dermal Exposures

D12.6.1 Dermal Exposures from Soil

Potential dermal exposure was estimated by applying soil loading rates to exposed skin, skin surface areas, and dermal absorption factors to measured or predicted soil concentrations. Dermal exposures were estimated separately for hands only and for surfaces other than hands (*e.g.*, arms and legs).

D12.6.2 Dermal Exposure to Hands

The following equation was used to estimate dermal exposure for hands only. Dermal exposures were based on recommendations from Health Canada (2010) or RAIS (2009) and Health Canada (2012).

$$EDI_{dermal\_h} = C_s \times SAH \times SLH \times RAF_{dermal}$$

Where:

EDI_{dermal_h}	=	estimated daily intake of chemical from dermal contact of hands with soil (µg/d)
C_s	=	chemical concentration in surface soil (mg/kg)
SAH	=	skin surface area of hands (cm²)
SLH	=	soil loading rate to exposed skin on hands (g/cm²/event)
RAF_{dermal}	=	relative dermal absorption factor (%)

Example 58 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from dermal exposure to soil with hands only

$$EDI_{dermal\_h} = 1.25E-06 \times 430 \times 0.0001 \times 0.15$$

$$EDI_{dermal\_h} = 8.09E-09\ \mu g/d$$

D12.6.3 Dermal Exposure to Surfaces Other than Hands

The following equation was used to estimate dermal exposure for surfaces other than hands. Dermal exposures were based on recommendations from Health Canada (2010) or RAIS (2009).

$$EDI_{dermal\_o} = C_s \times SAO \times SLO \times RAF_{dermal}$$

Where:

EDI_{dermal_o}	=	estimated daily intake of chemical from dermal contact of surfaces other than hands with soil (µg/d)
C_s	=	chemical concentration in surface soil (mg/kg)
SAO	=	skin surface area of upper and lower arms and legs (cm²)
SLO	=	soil loading rate to exposed skin on surfaces other than hands (g/cm²/event)
RAF_{dermal}	=	relative dermal absorption factor (%)

Example 59 Estimated daily intake of benzo(a)pyrene by an agricultural toddler resident from dermal exposure to soil with surfaces other than hands

$$EDI_{dermal\_o} = 1.25E-06 \times 2{,}580 \times 1.0E-05 \times 0.15$$

$$EDI_{dermal\_o} = 4.86E-09 \mu g/d$$

D12.7 Ingestion of Breast Milk by Infants

The potential health effects associated with the ingestion of the chemical-affected breast milk by nursing infants was considered in the current assessment. The estimated exposure from consumption of breast milk was calculated as the product of the breast milk consumption rate and predicted chemical concentration in breast milk. The equations used to predict the chemical concentration in breast milk are described in the following sections. The multiple pathway exposure model assumed that infants (*i.e.*, 0 to 6 months of age) obtained their

nutrients entirely from breast milk, and not from solid foods derived from the study area (*e.g.*, traditional plants and game meat).

D12.7.1 Breast Milk Biotransfer Factor

With the exception of dioxin and furans, the BTF for breast milk was used to convert the adult mother's total predicted exposure to a chemical concentration in her breast milk. Breast milk concentrations and exposures to the infant were based on recommended methods by the US EPA OSW (2005). For organic chemicals, the maximum fraction of the chemical expected to bioaccumulate was calculated using the following approach (McKone 1992):

$$BTF_{BM} = 2.0E-07 \times K_{ow}$$

Where:

BTF_{BM}	=	breast milk biotransfer factor ([µg/kg milk] / [µg/d intake])
K_{ow}	=	octanol-water partition coefficient (unitless)

As only infants were assumed to consume breast milk, the sample calculations below is based on a resident infant.

Example 60 Breast milk biotransfer factor for benzo(a)pyrene for an infant resident

$$BTF_{BM} = 2.0E-07 \times 1.35E+06$$

$$BTF_{BM} = 2.70E-01 \left[\mu g / kg\ milk\right] / \left[\mu g / d\ \text{intake}\right]$$

D12.7.2 Chemical Concentrations in Breast Milk

The predicted breast milk concentration was calculated as follows (McKone 1992):

$$C_{BM} = \frac{EDI_{mother} \times BTF_{BM}}{CF}$$

Where:

C_{BM}	=	predicted concentration of chemical in breast milk (µg/g milk)
EDI_{mother}	=	mother's total daily exposure to chemical via all routes (µg/d)
BTF_{BM}	=	breast milk biotransfer factor ([µg/kg milk] / [µg/d intake])
CF	=	conversion factor from kg to g (1,000 g/kg)

Example 61 Concentration of benzo(a)pyrene in breast milk for an infant resident

$$C_{BM} = \frac{6.71E-03 \times 2.70E-01}{1{,}000}$$

$$C_{BM} = 1.81E-06 \mu g / g\ milk$$

D12.7.3 Breast Milk Consumption

The estimated exposure from consumption of breast milk for infants was calculated as follows (Health Canada 2012):

$$EDI_{BM} = C_{BM} \times IR_{BM}$$

Where:

EDI_{BM}	=	estimated daily intake of chemical from consumption of breast milk (µg/d)
C_{BM}	=	concentration of chemical in breast milk (µg/g milk)
IR_{BM}	=	breast milk ingestion rate

Breast milk consumption was assumed to be 664 g/d (O'Connor and Richardson 1997).

Example 62 Estimated daily intake of benzo(a)pyrene for an agricultural infant from breast milk consumption

$$EDI_{BM} = 1.81E-06 \times 664$$

$$EDI_{BM} = 1.20E-03\ \mu g/d$$

D12.8 Total Human Exposure

Total exposure was calculated by summing the individual exposures from each medium (*i.e.*, soil, water, dust, and food intake) for all relevant exposure pathways on a per chemical and per life stage basis (Health Canada 2012):

$$EDI_{total} = EDI_{soil} + EDI_{water} + EDI_{dust} + EDI_{food} + EDI_{swim} + EDI_{dermal} + EDI_{BM}$$

Where:

EDI_{total}	=	total estimated daily intake of chemical via all routes (µg/d)
EDI_{soil}	=	estimated daily intake of chemical from soil ingestion (µg/d)
EDI_{water}	=	estimated daily intake of chemical from ingestion of water (µg/d)
EDI_{dust}	=	estimated daily intake of chemical from dust and air inhalation (µg/d)
EDI_{food}	=	estimated daily intake of chemical from consumption of all food types (µg/d [sum of leafy plants, root vegetables, berries, Labrador tea, wild rootsl, fish, moose, grouse, snowshoe hare])
EDI_{swim}	=	estimated daily intake of chemical from dermal contact and incidental ingestion of surface water during swimming (µg/d)
EDI_{dermal}	=	estimated daily intake of chemical from total dermal contact soil (µg/d)
EDI_{BM}	=	estimated daily intake of chemical from breast milk consumption (µg/d)

Example 63 Total estimated daily intake of benzo(a)pyrene for an agricultural from all routes of exposure

$$EDI_{total} = 1.00E-07 + 1.40E-03 + 2.60E-09 + 1.84E-06 + 1.1E-06 + 7.64E-06 + 1.1E-05 + 7.68E-04 + 2.68E-03 + 5.56E-06 + 5.89E-06 + 1.13E-05 + 8.09E-09 + 4.86E-09$$

$$EDI_{total} = 4.89E-03 \mu g/d$$

The total estimated daily intake was normalized to body weight as follows:

$$EDI_{total\_BW} = \frac{EDI_{total}}{BW}$$

Where:

EDI_{total_BW}	=	total estimated daily intake of chemical via all routes adjusted to body weight (µg/kg bw/d)
EDI_{total}	=	total estimated daily intake of chemical via all routes (µg/d)
BW	=	body weight (kg)

Example 64 Total estimated daily intake of benzo(a)pyrene for a toddler resident from all routes of exposure adjusted to body weight

$$EDI_{total\_BW} = \frac{4.89E-03}{16.5}$$

$$EDI_{total\_BW} = 2.96E-04 \mu g/kg\ bw/d$$

D13.0 HUMAN RISK CALCULATIONS

Risk quotient (RQ) values for non–carcinogens and incremental lifetime cancer risks (ILCRs) for carcinogens were estimated using the following equations and the calculated exposure estimates.

D13.1 Non-carcinogens

The following equation was used to calculate the risk quotients for non–carcinogens (Health Canada 2012):

$$RQ_i = \frac{EDI_{total\_BW}}{RfD}$$

Where:

RQ_i	=	risk quotient of chemical for the 'i' lifestage of the residents (unitless)
EDI_{total_BW}	=	total estimated daily intake of chemical via all routes adjusted to body weight for the 'i' lifestage (µg/kg bw/d)
RfD	=	chemical-specific reference dose (µg/kg bw/d)

The maximum RQ value of all the life stages (*i.e.*, infant, toddler, child, adolescent, and adult) was presented in the detailed HHRA report for non-carcinogens. The toddler lifestage had the highest RQ of all the lifestages.

Example 65 Risk quotient for formaldehyde for the Agricultural toddler life-stage in the Oil and Gas Scenario

$$RQ_i = \frac{3.01E-01}{150}$$

$$RQ_i = 2.0E-03$$

D13.2 Carcinogens

The following equation was used to calculate the incremental lifetime cancer risks for carcinogens (Health Canada 2012):

$$ILCR = \frac{EDI_{total\_BW-inf}}{RsD} x\, LAF_{-inf} + \frac{EDI_{total\_BW-tod}}{RsD} x\, LAF_{-tod} + \frac{EDI_{total\_BW-child}}{RsD} x\, LAF_{-child} + \frac{EDI_{total\_BW-adol}}{RsD} x\, LAF_{-adol} + \frac{EDI_{total\_BW-adult}}{RsD} x\, LAF_{-adult}$$

Where:

RQ	=	ILCR of chemical for the sum of the lifestages of the residents (unitless)
EDI_{total_BW-i}	=	total estimated daily intake of chemical via all routes adjusted to body weight for the 'i' lifestage (µg/kg bw/d)
RsD	=	chemical-specific risk-specific dose (µg/kg bw/d)
LAF-i	=	Lifetime adjustment factor for the 'i' lifestage for general population (yr-life stage/yr-total)

The sum of the ILCR values of all the life stages (*i.e.*, infant, toddler, child, adolescent, and adult) was presented in the detailed HHRA report for carcinogens.

Example 66 Risk quotient for benzo(a)pyrene for the Arras resident in the Oil and Gas scenario

$$ILCR = \frac{2.33E-04}{0.0014} x6.25E-03 + \frac{2.96E-04}{0.0014} x5.63E-02 + \frac{1.68E-04}{0.0014} x8.75E-02 + \frac{1.11E-04}{0.0014} x1.00E-01 + \frac{9.49E-05}{0.0014} x7.50E-01$$

$$ILCR = 8.2E-02$$

D14.0 REFERENCES

BC MOE (British Columbia Ministry of the Environment). 1985. Peace River Area, Charlie Lake Sub-Basin, Water Quality Assessment and Objectives. Technical Appendix. Resource Quality Section, Water Management Branch. February 1985.

BC OGC (British Columbia Oil and Gas Commission). 2014. North East Water Tool. Database inquiry for Charlie Lake. Available at: http://geoweb.bcogc.ca/apps/newt/newt.html.

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

Charlie Lake Conservation Society. 2005. Oil and Gas Well, Facility Site Erosion Management. Year 1 Summary. A 2-year Project of the Science and Community Environmental (SCEK) Fund. November 2005.

Grova N., Feidt, C., Laurent, C. and Rychen, G. 2002. [14C] Milk, urine and faeces excretion kinetics in lactating goats after and oral administration of [14C]polycyclic aromatic hydrocarbons. International Dairy Journal. 12: 1025-1031.

Health Canada. 1994. Human Health Risk Assessment for Priority Substances. Canadian Environmental Protection Act: Priority Substances List Assessment Report No. 41. Ottawa, ON: Health Canada. PSL-41E EN40-215/41E.

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 2010. Federal Contaminated Site Risk Assessment in Canada. PART II: Health Canada Toxicological Reference Values (TRVs) and Chemical-specific Factors, Version 2.0. Prepared by Contaminated Sites Division Safe Environments Directorate. September 2010.

Health Canada. 2012. Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA), Version 2.0. Prepared by; Contaminated Sites Division, Safe Environments Directorate, Ottawa, ON.September 2010, revised 2012.

Hofelt, CS, M Honeycutt, JT McCoy and LC Haws. 2001. Development of a Metabolism Factor for Polycyclic Aromatic Hydrocarbons for Use in Multipathway Risk Assessments of Hazardous Waste Combustion Facilities. Regul. Toxico. Pharm. 33: 60-65.

Laurent, C, C Feidt, E Lichtfouse, N Grova, F Laurent and G Rychen. 2001. Milk – Blood Transfer of (14C) Tagged Polycyclic Aromatic Hydrocarbons (PAHs) in Pigs. J Agr. Food Chem. 49: 2493-2496.

Laurent, C, C Feidt, N Grova, D Mpassi, E Lichtfouse, F Laurent and G Rychen. 2002. Portal adsorption of (14C) benzo(a)pyrene or (14C) TCDD in growing pigs. Chemosphere 48: 843-848.

McCrady, JK and SP Maggard. 1993. Uptake and photodegradation of 2,3,7,8-tetrachlorodibenzo-p-dioxin sorbed to grass foliage. Environ. Sci. Technol. 27(2):343-350. Cited in: US EPA OSW 2005.

McKone, TE. 1992. CalTOX, a Multimedia Total-exposure Model for Hazardous-waste Sites. Part III: The Multiple-pathway Exposure Model. Sacramento, CA: Office of the Science Advisor, Department of Toxic Substances Control, California Environmental Protection Agency.

Nagy, KA, IA Girard and TK Brown. 1999. Energetics of free-ranging mammals, reptiles and birds. Annu. Rev. Nutr. 19: 247-277.

Nagy, KA. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol. Monogr. 57: 111-128.

O'Connor Associates Environmental Inc. and G Mark Richardson. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment.1155-2720 Queensview Dr., Ottawa, Ontario.

Personal Communication. 2014. Karl Bresee, Intrinsik with Keith Carroll, P.Ag, Range Agrologist with the Peace River Forest District. May 22, 2014.

RAIS (Risk Assessment Information System). 2009. Chemical Specific Paramaters. Available at: http://rais.ornl.gov/cgi-bin/tools/TOX_search?select=chem_spef

Ramesh A, SA Walker DB Hood MD Guillen, K Schneider and EH Weyand. 2004. Bioavailability and Risk Assessment of Orally Ingested Polycyclic Aromatic Hydrocarbons. Int. J. Toxicol. 33: 301-333.

RTI (Research Triangle Institute). 2005. Methodology for Predicting Cattle Biotransfer Factors. September 23, 2005. Prepared for US Environmental Agency Office of Solid Waste.

Suter, GW, RA Efroymson, BE Sample and DS Jones. 2000. Ecological Risk Assessment for Contaminated Sites. Lewis Publishers, CRC Press LLC.

Swan, RL, PJ McCall and SM Unger. 1979. Volatility of Pesticides from Soil Surfaces. Unpublished, undated manuscript received as personal communication from Dow Chemical USA, Midland, Mich. In: WJ Lyman, WF Reehl and DH Rosenblatt (eds.) Handbook of Chemical Property Estimation Methods, Environmental Behaviour of Organic Compounds. 1990. Washington, DC: American Chemical Society.

Travis, CC and AD Arms. 1988. Bioconcentration of organics in beef, milk and vegetation. Environ. Sci. Technol. 22:271-274.

US EPA (United States Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook. Washington, DC: Office of Health and Environmental Assessment, Office of Research and Development. EPA/600/R-93/187. December 1993.

US EPA. (2004). Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment). Final. EPA/540/R/99/005. OSWER 9286.7-02RP. PN99-963312. July 2004.

US EPA. (2011). Estimation Programs Interface Suite™ for Microsoft® Windows, v 4.0. United States Environmental Protection Agency, Washington, DC, USA.

US EPA OSW (United States Environmental Protection Agency Office of Solid Waste). 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Volume I Peer Review Draft. US EPA Region VI. Multimedia Planning and Permitting Division. Center for Combustion Science and Engineering. Office of Solid Waste.

US EPA OSW. 2005. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Final. US EPA Region VI. Multimedia Planning and Permitting Division. Center for Combustion Science and Engineering. Office of Solid Waste.

Weast, RC. 1981. Handbook of Environmental Data on Organic Chemicals. Second Edition. Van Nostrand Reinhold Company. New York, NY. Cited in: US EPA OSW 2005.

Intrinsik Environmental Sciences | 736-8th Avenue SW, Suite 1060 | Calgary, Alberta T2P 1H4 | Tel: (403) 237-0275 | Fax: (403) 237-0291 | intrinsik.com