SNC·LAVALIN

HUMAN HEALTH RISK ASSESSMENT

Fraser Surrey Docks Direct Transfer Coal Facility
Revised Final Report

Submitted to:

FRASER SURREY DOCKS
Pacific Rim Stevedoring

Prepared by:

SNC·LAVALIN

ENVIRONMENT & WATER

July 18, 2014

Internal Ref: 615850

SNC-LAVALIN INC.
8648 Commerce Court
Burnaby, British Columbia
Canada V5A 4N6
Tel.: 604-515-5151
Fax: 604-515-5150

EXECUTIVE SUMMARY

Fraser Surrey Docks LP (FSD) retained the Environment & Water business unit of SNC-Lavalin Inc. (SNC-Lavalin) to conduct a human health risk assessment (HHRA) of the Direct Transfer Coal Facility (the Project) proposed for the existing Fraser Surrey Docks (FSD) terminal site located on the Fraser River in Surrey, British Columbia (BC).

The HHRA is based largely on the results of an Air Quality Assessment (AQA) conducted by Levelton Consultants Ltd. (Levelton) (2014). As part of the AQA, Levelton, in consultation with Port Metro Vancouver and Metro Vancouver, developed a detailed air dispersion model to predict potential emissions from the Project. The AQA considered proposed emission sources related to the Project operations, in addition to the current emission sources from FSD's current agricultural operations. The results of the Levelton (2014) AQA were used to estimate exposures to the receptors of concern identified in the HHRA.

The Levelton (2014) AQA concluded, that with the exception of the maximum predicted annual NO_2, the maximum concentrations of Criteria Air Contaminants (CACs) plus background were below the most stringent of the municipal, provincial, national and international air quality objectives and guidelines. The higher annual NO_2 concentrations were predicted adjacent the FSD fenceline in the area of the berth, in a region concentrated over the waters of the Fraser River.

In addition to the results of the Levelton (2014) AQA, the HHRA considered the results of the analysis of the coal that is proposed to be transported as part of the Project, as well as the results of a background soil assessment that was conducted in the Study Area. The HHRA also considered the material safety data sheets for the binding and suppressing agents that will be used to control dust as part of the Project.

The HHRA was conducted using methods and guidance recommended by Health Canada, and using a series of conservative assumptions that will tend to overpredict exposures, and therefore risks, to receptors in the Study Area. Despite the conservative approach, no unacceptable risks have been predicted for the receptors in the Study Area (residents, commercial workers, urban park users, agricultural receptors, people involved in fishing activities), including those that have the potential to be exposed to the maximum Project emissions (the maximum North Delta residential receptor, the maximum rail corridor residential receptor and the industrial receptor).

In summary, no unacceptable health risks are predicted for exposures to the Project emissions in the Study Area.

Table of Contents

Table of Contents (Cont'd)

Page

Table of Contents (Cont'd)

Table of Contents (Cont'd)

Table of Contents (Cont'd)

Page

In-Text Tables (Cont'd)

Table of Contents (Cont'd)

Page

In-Text Tables (Cont'd)

Appendices

1 INTRODUCTION

Fraser Surrey Docks LP (FSD) retained the Environment & Water business unit of SNC-Lavalin Inc. (SNC-Lavalin) to conduct a human health risk assessment (HHRA) for the Direct Transfer Coal Facility (DTCP or the Project) proposed for the existing Fraser Surrey Docks (FSD) terminal site located on the Fraser River in Surrey, British Columbia (BC).

SNC-Lavalin understands that Port Metro Vancouver (PMV) has requested that a HHRA be conducted to further evaluate the potential human health impact of the Project. The HHRA, including methods and results, is presented in this report.

1.1 Scope

The scope of the HHRA was defined by SNC-Lavalin, in consultation with PMV, and Golder Associates Ltd. (Golder), who were retained by PMV as a third party reviewer, and to provide technical advisory services to PMV on the HHRA for the Project. The scope was developed considering the concerns raised and comments provided by the public and the local Health Authorities on the potential health effects associated with fugitive dust and air emissions from the Project. The scope of the HHRA was presented to PMV and Golder, and feedback was provided to SNC-Lavalin on certain aspects of the scope, in particular for aspects of the HHRA where SNC-Lavalin was required to exercise professional judgement.

The scope of the Project under the jurisdiction of the PMV includes the development of the coal handling facility at FSD, including new rail within the Port Authority Rail Yard (PARY), the direct transfer of coal from rail onto barge at FSD, and the barge transport of coal from the Project site to the mouth of the Fraser River. The physical works and activities undertaken during or preceding the loading of coal onto rail cars, the rail transport of coal from the mine site to PARY/FSD, barge transport from the mouth of the Fraser River to Texada Island and transport during and after the coal is unloaded at Texada Island are outside of the jurisdiction of PMV. Additionally, neither the mining of the coal nor the ultimate use of the coal, are within the scope of PMV or of this assessment.

The HHRA focused specifically on the potential health risks associated with emissions from the Project within the Study Area (defined below) and associated with the sources and activities under the jurisdiction of PMV; however, to determine whether exposures to emissions generated during rail transport of the coal within the BC lower mainland could be associated with potential health risks, at the request of FSD, the rail transport of coal from the Canada/US Border to FSD has also been included in the HHRA.

The Study Area includes the Project footprint and nearby land outside the FSD terminal lease boundaries including industrial, residential and commercial properties in the City of Surrey and the Corporation of Delta. Additionally, the Study Area also includes the rail transport of coal from the Canada/US Border to FSD, and the barge transport of coal from FSD to the mouth of the Fraser River, along the south arm of the Fraser River, which would include any nearby lands in any of the municipalities either the rail cars or barges transit through.

The air dispersion modeling Study Area considered a 20 km by 20 km domain with the FSD facility located at the center of the domain, as shown below in Figure 1. Fenceline and gridded receptors are indicated by the blue crosses, while the sensitive receptors are indicated by the red crosses. In total, the Levelton (2014) modelling domain contains over ten thousand receptors where ambient air concentrations were predicted, which included numerous sensitive receptors for: hospitals; schools; senior care residences; and, day care centres. The modelling domain is depicted in Figure 1.

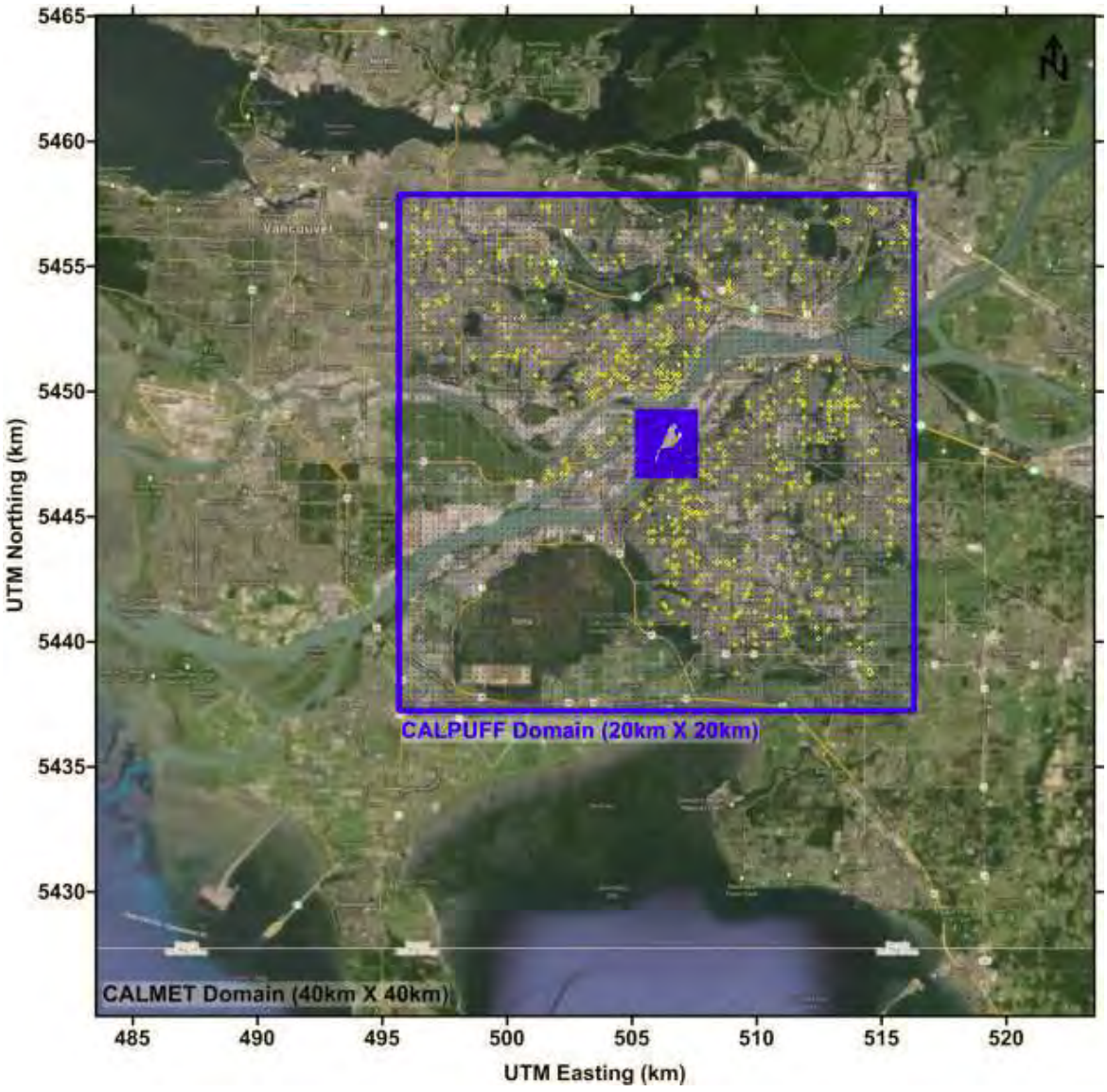

Figure 1-1: The Air Dispersion Modeling Domain (from Levelton, 2014).

The air dispersion modeling domain is presented in the above Figure in blue (20 km x 20 km area). Gridded receptors are indicated as blue crosses, while sensitive receptors are indicated as yellow crosses (Levelton, 2014).

1.2 Assessment Scenarios

The objective of the HHRA was to evaluate the potential human health risks for the following three scenarios:

- Baseline Scenario – includes potential health risks associated with the existing environmental conditions;
- Project Scenario – includes potential health risks associated with the Project alone, in order to assess incremental health risks associated with the Project; and
- Cumulative Scenario – includes potential health risks associated with the Baseline Scenario plus the Project Scenario to assess the total health risks associated with Project development.

The above described scenarios were assessed in the HHRA, and are discussed throughout the Problem Formulation.

2 HHRA APPROACH

The HHRA has been conducted using a series of highly conservative assumptions (i.e., reasonable worst-case), including the assumption that people in the area of the Project will be exposed to maximum predicted air emissions, to ensure that human health risks associated with the Project are not under predicted.

FSD is located on federal land under the jurisdiction of PMV. The direct transfer of coal from rail to barge and the movement of coal-laden barges down the Fraser River from FSD to the mouth of the river are within federal PMV jurisdiction. As such, guidance published by federal agencies, and specifically Health Canada's guidance on human health risk assessment, was followed when completing the HHRA. In addition to guidance collected through communication with Health Canada representatives, the following Health Canada guidance documents were considered and are referenced throughout the HHRA:

- Federal Contaminated Site Risk Assessment in Canada – Part V: Guidance on Complex Human Health Detailed Quantitative Risk Assessment. Version 1.0. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario (Health Canada, 2010a);
- Federal Contaminated Site Risk Assessment in Canada – Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors. Version 2.0. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario (Health Canada, 2010b); and
- Federal Contaminated Site Risk Assessment in Canada – Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). Version 2.0. May 2012. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario (Health Canada, 2012).

The HHRA was largely completed according to the above Health Canada guidance, and is consistent with methods commonly used by regulatory agencies across Canada and the United States (US), including the BC Ministry of Environment (MoE) and the US Environmental Protection Agency (US EPA). The HHRA consists of five main components, including the following:

- *Problem Formulation.* The Problem Formulation presents the location and description of the Project, the identification of chemicals of potential concern (COPCs) for the Project, the populations (also referred to as receptors of concern) that have the potential to be exposed to COPCs, and the relevant exposure pathways for the receptors of concern.

- *Exposure Assessment.* The Exposure Assessment involves the estimation of the dose of each COPC that the receptors of concern have the potential to be exposed to.
- *Toxicity Assessment.* The Toxicity Assessment is the compilation of toxicity data on the potential adverse health effects for each of the COPCs, as well as TRVs for each of the COPCs. For non-carcinogenic chemicals, TRVs represent an exposure dose or air concentration below which no adverse effects are expected to occur. For carcinogenic chemicals, the TRV is presented as an upper bound of the increased cancer risk from a lifetime exposure either a specified air concentration or intake rate of to the chemical.
- *Risk Characterization.* In the Risk Characterization the doses estimated in the Exposure Assessment are compared to/combined with the TRVs identified in the Toxicity Assessment to estimate potential health risks associated with receptor exposure to the COPCs under the assumptions of the HHRA.
- *Uncertainty Analysis.* The Uncertainty Analysis is conducted to evaluate the sources of uncertainty inherent in the HHRA, as well as how the uncertainty will affect the results of the HHRA.

As discussed above, the assessment has been conducted using a series of conservative assumptions to ensure that human health risks associated with the Project are not under predicted. This type of approach limits the likelihood of under predicting health risks and is likely to result in a considerable over prediction of risks. This will be further discussed in the Uncertainty Analysis.

3 PROBLEM FORMULATION

The Problem Formulation is the initial step of the HHRA and forms the basis of the assessment conducted in the remaining steps of the HHRA.

The main elements of Problem Formulation include the following:

1) **Project Location and Description:** Details on the FSD Facility and surrounding lands are described to provide context for the HHRA and to provide a basis for the identification of receptors of concern and potential operable exposure pathways.
2) **Data Evaluated in the HHRA:** Results of the air quality assessment, coal analysis, background soil assessment and dust palliatives review are presented and are evaluated in the COPC Screening.
3) **COPC Screening:** The results of the data evaluation are used to screen data and identify COPCs.
4) **Human Receptors of Concern:** The demographics of the project area, land uses occurring within the area, the locations with highest project emissions and Health Canada guidance are used to identify potential human receptors of concern.
5) **Identification of Exposure Pathways:** Using the findings of the previous sections (including the human receptor identification and COPC screening), potentially operable exposure pathways are identified.
6) **Conceptual Model:** The conceptual model provides a summary of the receptors of concern and the potentially operable exposure pathways carried forward for quantitative evaluation in the HHRA.

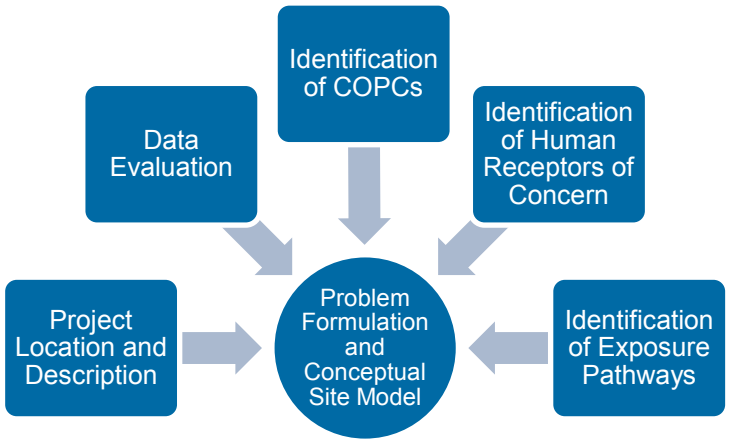

Figure 3-1: Main Elements of the Problem Formulation

The Problem Formulation is presented in the following sections.

3.1 Project Location and Description

The Project is located along the Fraser River in Surrey, BC at 11060 Elevator Road, on the border between the City of Surrey and the Corporation of Delta. The City of New Westminster is north of the Project on the opposite bank of the Fraser River. The geographic coordinates are approximately 49° 10' 42.5172 N, 122° 55' 1.7106" W. The location of FSD is presented on Figure 3-2.

Figure 3-2: Fraser Surrey Docks Location

The current FSD lease area (approximately 53.38 hectares) is the proposed location of land-based unloading and direct transfer of coal. The Project is proposed to be on the existing FSD marine terminal facility in Surrey, BC, which has been in operation for over 50 years. The Project includes construction and installation of direct coal transfer infrastructure, primarily consisting of new rail track, coal receiving pits and a conveyor system, on the

current FSD lease area. The Project also includes rail upgrades and installation of new rail track on the adjacent PARY licence area.

The Project is expected to last for 10 years, and FSD estimates that the Project will handle 2.0 million MT (metric tons) of coal in the first year of operations, with the volume increasing to 4.0 million MT in the subsequent years.

A detailed description of the coal operations proposed for the Project, including coal delivery, land-based operations traffic, coal conveyance, dust mitigation, barge loading and water-based traffic operations, as well as a description of the current agricultural operations at FSD, is provided in SNC-Lavalin (2013) and Levelton (2014). A summary of this information follows:

- FSD coal operations will include:
  - Receiving rail cars loaded with sub-bituminous coal. The coal will be transported from the mine site to the terminal via BNSF (Burlington Northern Santa Fe Corp.) controlled unit trains consisting of 125 to 135 bottom-dump cars. Loaded rail cars will be received into the covered unloading shed and gravity fed into the recieving pit.
  - FSD estimates that in the first year of operations, there will be a total of 160 unit train deliveries to FSD, with the number doubling to 320 in subsequent years. This would be approximately one train arriving and leaving from FSD every two days in the first year, and one train arriving and leaving per day in subsequent years.
  - Coal will be gravity fed from the rail cars into the receiving pits and moved along a series of covered conveyor segments and transfer points, treated with a binding agent and then directly transferred on to the waiting barge(s).
  - There will be a number of dust control methods utilized during operations including:
    - Wetting, which keeps coal moist and prevents dust generation, as well as fogging/misting to removes airborne dust particles;
    - Dust suppressing agents (i.e., dust palliatives) will be used at all stages of the coal transfer process; the agents will be applied at the mine site prior to rail transport, at a mid-point during rail transport and prior to barge transport from FSD to Texada Island;
    - Profiling coal into a rounded ‘bread loaf’ shape when it is loaded into rail cars to reduce the possibility for wind erosion and dispersion; and

  - The incorporation of physical barriers (i.e., fencing, enclosures, walls, etc.) in the Project design to control dust generation and dispersion.

- Loading of coal onto barges at existing FSD berth Nos. 2 and 3, with transport from FSD to Texada Island via single hulled coastal barges. The operation of barges will be avoided in wind conditions greater than 40 kilometres per hour (km/h).

The barge route is an established barge route to/from Texada Island. FSD estimates that in the first year of operations there will be 320 fully-loaded barges traveling to Texada Island, with the number doubling to 640 barges in subsequent years of the Project. It is expected that in the first year, on average there will be two barges arriving and departing FSD every second day; in subsequent years, there will be an average of two barges arriving and departing FSD every day.

FSD currently operates an on-site agricultural bulk facility in partnership with Parrish & Heimbecker. The facility is designed to handle a variety of agricultural products in bulk including canola meal, DDGs, malt, lentils, etc. The facility also has a gravity fed rail receiving area with conveyor belts and a weighing system. The system is designed so that agriculture products can either be stored in a shed or directly loaded onto vessels.

The Project operations, and in particular, the emissions predicted to be associated with the above summarized operations, were evaluated by Levelton (2014) in their Air Quality Assessment (AQA). The results of Levelton (2014) AQA are further discussed below, and are direct inputs into the HHRA.

3.1.1 FSD Facility Description

FSD is a multi-purpose marine terminal located on the Fraser River at the border of the City of Surrey (Whalley) and the Corporation of Delta (North Delta) and has been in operation since 1962. FSD is an active industrial operator in the South Westminster neighbourhood of Surrey. The terminal serves a variety of customers involved in containers, breakbulk, project cargo, forest products and bulk. The terminal currently has facilities to handle and transfer goods by rail, truck, barge and vessel, and has warehouses for cargoes requiring covered storage.

The Project operations will be confined to the existing footprint of the FSD facility and the adjacent PARY, areas that are zoned as Industrial lands. Access to the FSD facility is restricted by fencing and a gate at the entrance to the facility. The facility, including all berth and rail areas, is monitored through video and mobile security patrols on a 24 hour 7 days a week basis. SNC-Lavalin understands that signage will be placed along the berth face and breakwater on the west side (i.e., Fraser River side) of the facility to further discourage entry.

3.1.2 Description of Surrounding Lands

The Project is located in the South Westminster neighbourhood of Surrey; South Westminster is situated in the northwest quadrant of Surrey, with the neighbourhood bordered by the King George Highway to the north, the Fraser River to the northwest and the toe of the slope of the Whalley hillside to the south and southeast. South Westminster is currently zoned to support a variety of commercial, industrial and business park uses.

As described above, the FSD facility is located on industrial land, and the immediately adjacent surrounding lands are also used for industrial purposes. Residential lands, including several schools, childcare centres and parks and recreational facilities are located within the Study Area, as are several commercial operations. As will be discussed in subsequent sections of the HHRA, people living and working in the area of the FSD facility are those that have the potential to be exposed to some of the highest emissions from the Project; therefore, the below discussion of surrounding lands focuses on the areas within 3 km of FSD. In addition, this portion of the study area includes the primary area that PMV has jurisdiction over. The use of the lands in this area is summarized below. It is noted that while the AQA predicted maximum concentrations for receptors within this area, the assessment of air quality and associated potential exposures and associated risks along the rail corridor and barge route has also been conducted.

3.1.2.1 Industrial

Industrial land use dominates the land surrounding the immediate Project area, including all lands to the west of the South Fraser Perimeter Road. FSD is located in the industrial area of South Westminster, which includes approximately 1042 acres or 422 hectares of industrial lands located to the west of King George Boulevard and the Pattullo Bridge in Surrey, BC. In addition to FSD, industry in this area includes, among others, Interfor's Acorn Sawmill, Nap Steel, TMS Transportation, Rabanco Intermodal Distribution, Chemitron Rail Welding Services, CN Railway/Intermodal Yard, Van Kam Freightways, Day & Ross, Bekaert Industries, Vitran Distribution, Fed Ex Freight, Westran Stuffing Facility, Catalyst Paper Distribution Center, Western Cleanwood, Seaspan, Allied Blower, Pacific Coast Express and Mill & Timber. These companies represent steel suppliers and distributors, a manufacturing facility for fire suppressants, freight and distribution companies, tug operators, rail service and maintenance providers and several forest products company.

Annacis Island is located to the west of FSD, across the Fraser River. The island is used primarily for industrial purposes, and contains one of Metro Vancouver's secondary wastewater treatment plants. Further west of Annacis Island, is the New Westminster Queensborough neighbourhood; land use in Queensborough is mixed, with some industrial operations.

3.1.2.2 Residential

While the Project is situated on industrial lands, there are residential neighbourhoods in proximity. The South Westminster, Surrey neighbourhood has localized areas of residential lands, with the residential areas located primarily to the northeast of FSD. The North Delta residential neighbourhoods of Sunbury, Annieville and Nordel are located to the east of FSD and of the South Fraser Perimeter Road. The closest residential properties to FSD are located in North Delta, across the South Fraser Perimeter Road, approximately 400 m to the southeast of the FSD fenceline. As noted above, the New Westminster neighbourhood of Queensborough is located across the Fraser River from FSD. There are several residential areas in Queensborough.

3.1.2.2.1 Schools

The residential neighbourhoods include several elementary and secondary schools, daycares and community centres, all of which are considered residential land use. Five schools are located within 3 km of the FSD facility, and several childcare facilities are also present. The schools and child care facilities include the following:

In Surrey:

- Royal Heights Elementary School;
- Kirkbride Elementary School;
- L.A. Matheson Secondary School;
- S and C Tiny Town Daycare;
- St. Barnabas Daycare;
- Smart Baby Family Childcare;
- Treasure Chest Family Daycare;
- Little Safari Daycare; and
- Royal Heights Daycare and Preschool.

In Delta:

- Annieville Elementary School;
- Delview Secondary;
- Bean Sprouts Childcare Centre;

- Kangaroo Family Childcare Centre; and
- Delta Funtime Out of School Childcare Centre.

In Queensborough, New Westminster:

- Queen Elizabeth Community School;
- Queensborough Middle School;
- Queensborough Community Centre;
- Licensed Daycare, Salter Street, New Westminster;
- Merseyside Montessori;
- New West Montessori Daycare; and
- Bethany Childcare Centre.

3.1.2.3 Parks and Recreational Facilities

Recreational features and parks in Surrey and Delta, as well as in New Westminster, located within 3 km of FSD include:

- Ravine Park;
- Royal Heights Park;
- Tom Hopkins Ravine Park; and
- Tannery Park.

In New Westminster:

- Queensborough Community Centre;
- Old Schoolhouse Park;
- Port Royal Park and Community Garden; and
- Queensborough Skatepark.

3.1.2.4 Commercial

There is limited commercial land use within the immediate area of the Project; however, select commercial operations are present. The nearest commercial business to FSD is a convenience store located to the southeast, approximately 600 m from the FSD fenceline.

As noted above, Annacis Island is located to the west of FSD, across the Fraser River. The island is used primarily for industrial purposes; however, various commercial operations (e.g., restaurants, gas stations) are present. Additionally, several commercial operations, including the Queensborough Landing shopping complex, are located further west/northwest of FSD, in the Queensborough neighbourhood of New Westminster.

3.2 Data Evaluated in the HHRA

Levelton, in consultation with PMV and Metro Vancouver, developed a detailed air dispersion model; as the Project is not yet operational, the model was used to predict potential emissions from the Project, with the results presented in the Levelton (2014) Air Quality Assessment (AQA). The AQA considered proposed emission sources related to the Project operations, in addition to the current emission sources from FSD's current agricultural operations. The results of the Levelton (2014) AQA were used to estimate exposures to the receptors of concern identified in Section 3.4 of this report.

In addition to the results of the Levelton (2014) AQA, the HHRA considered the results of the analysis of the coal that is proposed to be transported as part of the Project, as well as the results of a background soil assessment that was conducted in the Study Area. The HHRA also considered the material safety data sheets (MSDS) for the binding and suppressing agents that will be used to control dust.

A summary of the results of these assessments, as well as how they were used in the HHRA, is presented below.

3.2.1 Air Quality Assessment Results

FSD retained Levelton to conduct an AQA (Levelton, 2014) to assess emissions from the Project, and to develop an Air Quality Management Plan to monitor air quality to determine the baseline, and to continue the monitoring program following the initiation of the Project to ensure mitigation measures are effective and air quality objectives are met.

The scope of the Levelton (2014) AQA included determining baseline air quality from existing Metro Vancouver monitoring stations near FSD and assessing the potential emissions associated with the various components of the Project including rail locomotives, tug-boats, barges and on site operations (i.e., rail unloading, material transfer points, barge loading) and potential emissions from agricultural handling operations.

Levelton modelled various air contaminants associated with potential emission sources related to the Project and FSD's agricultural handling facility for various averaging periods (1 hour to annual). The modelling was conducted assuming the coal throughput for year 2 and beyond (i.e., maximum throughputs), and air concentrations were estimated for particulate matter (PM) (PM_{10} and $PM_{2.5}$), diesel particulate matter (DPM), carbon monoxide (CO), sulphur dioxide (SO_2), nitrogen dioxide (NO_2) and total particulate matter (TPM). There is the potential for contribution to these emissions from various transportation equipment engine combustion sources associated with the Project; additionally, PM_{10} and $PM_{2.5}$ provide a measure of the potential for fugitive dust (i.e., suspended dust) from the coal handled/transported as part of the Project, as well as any suspended dust generated from the agricultural operations at FSD, to impact air quality. In addition, to facilitate the HHRA, the constituents of PM and VOC emissions were identified to allow for the assessment of health risks, if any, associated with exposures to the individual constituents.

Levelton (2014) presented the following conclusions of the AQA:

- There are predicted exceedences noted for annual NO_2 when combining the impacts of the proposed Project, agricultural goods operations, and ambient background concentrations.
- Predicted annual NO_2 exceedences are located immediate to the west of the modelled facility fenceline, over the Fraser River. This is an area where the tugs and vessels operate and public access is generally limited or controlled due to terminal marine operations. However, while the modelling results are likely to be conservative by nature, ambient air quality monitoring is recommended to validate that air quality exceedances will not occur.
- The majority of the maximum predicted modelled concentrations are located on the facility fenceline. The exception is the maximum 8-hour rolling CO concentration is located slightly beyond the west side of the fenceline.
- The predicted air contaminant concentrations quickly diminish as emissions disperse further away from FSD's facility or the in-transit emission sources.
- Predicted air quality impacts at sensitive receptors and within residential neighbourhoods in the vicinity of FSD with the ambient background added are generally low and remain below all AAQOs.
- Predicted air quality impacts at receptors adjacent to the in-transit study areas with the ambient background added are generally low and remain below all AAQOs.

Further details on the results of the Levelton (2014) AQA, including the assessment of baseline air quality, are presented in the following sections.

3.2.1.1 *Baseline Air Quality*

Baseline ambient air quality in the area surrounding the FSD facility was determined using data from three Metro Vancouver ambient air quality monitoring stations (T13 North Delta, T18 Burnaby South and T6 Second Narrows); these monitoring stations were selected in consultation with Metro Vancouver, with the T6 station selected as it is considered representative of air quality in an industrial area. The other monitoring stations were chosen based on their proximity to the FSD site and the air quality parameters monitored.

Background levels, presented as 1-hour maximums, 8-hour and/or 24-hour maximums, and annual average concentrations, of PM, CO, NO_2, SO_2, DPM and TPM are summarized in Table 3-1.

Table 3-1: Background Ambient Air Concentrations for PM, CO, NO_2 and SO_2

Pollutant	Averaging Time	Background (µg/m^3)
$PM_{2.5}$	24-hour	12
	Annual	4.4
PM_{10}	24-hour	27
	Annual	12
CO	1-hour	617
	8-hour	555
NO_2	1-hour	66
	Annual	27
SO_2	1-hour	28
	24-hour	18
	Annual	4
DPM[a]	24-hour	2.3
	Annual	0.8
TPM[b]	24-hour	56
	Annual	25

[a] estimated as 19% of the baseline PM2.5 concentrations as recommended in the report titled *Air Toxics Emissions Inventory and Health Risk Assessment – Summary Report* (Levelton, 2007), prepared for Metro Vancouver

[b] calculated based on the ratio of PM_{10} to TPM (Source: US EPA, 1986)

The data presented in Table 3-1 was used in the Baseline Scenario assessment, as well as in the Cumulative Scenario assessment (Baseline Scenario + Project Scenario).

3.2.1.2 *Air Quality Assessment Results*

The AQA conducted by Levelton (2014) predicted ambient air concentrations from the Project and from the agricultural operations at FSD, which were then added to the background ambient air concentrations for the Study Area. It should be noted that air dispersion modelling techniques and practices used by Levelton (2014) are considered to be conservative as they consider the combined effects of worst case emissions and meteorological conditions. This results in the ability to predict upper bound concentrations all within the context of atmospheric physics in the model that errs toward conservative air quality estimates. The conservatism in the air quality model is described in Levelton (2014), as is the associated uncertainty in the emission estimates. The Uncertainty Analysis of the HHRA (Section 7), also discusses the uncertainty in the estimates, and the resulting impact on the results of the HHRA. As indicated above, the air dispersion modelling conducted by Levelton (2014) predicted concentrations of PM_{10}, $PM_{2.5}$, CO, SO_2, NO_2, DPM and TPM resulting from the following Project sources:

- Coal dust;
- transportation equipment combustion emissions; and,
- the agricultural operations at FSD.

The results of the Levelton (2014) AQA, broken down by source and compared to the Metro Vancouver ambient air quality objectives (AAQO), are presented in Table 3-2 on the following page. To provide provincial, national and international context, the British Columbia Air Quality Objectives (AQO) (BC MoE, 2013), the Canadian Council for Ministers of the Environment (CCME) National Ambient Air Quality Objectives (NAAQO) (CCME, 1999) and Canada-wide Standards for Particulate Matter (CCME, 2000), as well as the World Health Organization (WHO) Air Quality Guidelines (AQG) (WHO, 2000, 2006 and 2010) are also provided for comparison purposes. The CCME has developed up to three objective values using the categories "maximum desirable", "maximum acceptable", and "maximum tolerable". The "maximum desirable" objective is the most stringent standard. British Columbia has established a similar set of objective values, designated as levels A, B and C, with level A being the most stringent. Level A is typically applied to new and proposed discharges to the environment, and is typically equivalent to the federal "maximum desirable" objective.

Table 3-2: Results of Levelton (2014) AQA: Maximum Predicted Concentrations at the FSD Fenceline and at the Maximum North Delta Residential Receptor

CAC	Averaging Time	Metro Vancouver[a] (µg/m³)	CCME[b]			British Columbia[d]			WHO (µg/m³)	Back-ground (µg/m³)	Maximum Concentration (µg/m³)								Maximum Concentration + Background (µg/m³)							
											Maximum Receptor				Nearest Residential Receptor				Maximum Receptor				Nearest Residential Receptor			
			Maximum Desirable (µg/m³)	Maximum Acceptable (µg/m³)	Maximum Tolerable (µg/m³)	Level A (µg/m³)	Level B (µg/m³)	Level C (µg/m³)			Coal	Agri	Combustion	Total	Coal	Agri	Combustion	Total	Coal	Agri	Combustion	Total	Coal	Agri	Combustion	Total
CO	1-hour	30,000	15,000	35,000	-	14,300	28,000	35,000	30,000[e]	617	-	-	236	236	-	-	76	76	-	-	854	854	-	-	694	694
	8-hour	10,000	6,000	15,000	20,000	5,500	11,000	14,300	10,000[e]	554	-	-	101	101	-	-	48	48	-	-	656	656	-	-	602	602
NO_2 (ARM*)	1-hour	200	-	400	1,000	-	-	-	200[f]	66*	-	-	111	111	-	-	100	100	-	-	111	111	-	-	100	100
NO_2 (100%**)	Annual	40	60	100	-	-	-	-	40[f]	27	-	-	21	21	-	-	5	5	-	-	**48**	**48**	-	-	32	32
SO_2	1-hour	450	450	900	-	450	900	900	-	28	-	-	1.0	1.0	-	-	0.4	0.4	-	-	29	29	-	-	28	28
	24-hour	125	150	300	800	160	260	360	20[f]	18	-	-	0.1	0.1	-	-	0.1	0.1	-	-	18	18	-	-	18	18
	Annual	30	30	60	-	25	50	80	-	4	-	-	0.01	0.01	-	-	0.003	0.003	-	-	4	4	-	-	4	4
PM_{10}	24-hour Rolling	50	-	-	-	50	-	-	50[f]	27	11.1	16.2	4.5	18.4	4.7	1.7	4.0	4.9	37.9	43.0	31.2	45.1	31.5	28.5	30.8	31.6
	Annual	20	-	-	-	-	-	-	20[f]	12	1.2	1.3	0.4	1.8	0.2	0.04	0.1	0.4	13.2	13.3	12.4	13.9	12.2	12.0	12.1	12.4
$PM_{2.5}$	24-hour Rolling	25	30[c] (2015: 28)[2] (2020: 27)[2]	-	-	25	-	-	25[f]	12	2.3	4.1	4.3[1]	5.0	0.7	0.4	3.9[1]	3.9	14.9	16.6	16.9[1]	17.6	13.2	12.9	16.4[1]	16.5
	Annual	8(6)	(2015: 10)[2] (2020: 8.8)[2]	-	-	8(6)	-	-	10[f]	4	0.3	0.3	0.3[1]	0.7	0.1	0.01	0.1[1]	0.2	4.7	4.8	4.8[1]	5.1	4.5	4.5	4.6[1]	4.7
TPM	24-hour	120	-	-	-	-	-	-	-	56	24.0	41.9	4.1	46.7	9.4	4.5	3.9	9.5	79.8	97.6	59.8	102.4	65.1	60.2	59.6	65.3
	Annual	60	-	-	-	-	-	-	-	25	3.1	3.5	0.4	4.7	0.5	0.1	0.1	0.7	28.2	28.6	25.5	29.8	25.6	25.2	25.3	25.9
Dustfall	Annual	1.7 (mg/dm²/day)	-	-	-	-	-	-	-	0.5	0.08	0.03	0.0002	0.08	0.01	0.002	0.00005	0.01	0.6	0.5	0.5	0.6	0.5	0.5	0.5	0.5

Notes:
a. Metro Vancouver Ambient Air Quality Objectives (2011); the annual $PM_{2.5}$ objective includes a planning goal (shown in brackets)
b. CCME National Ambient Air Quality Objectives (1999)
c. CCME Canada Wide Standards (2000)
d. B.C. Ambient Air Quality Objectives (2013); the annual $PM_{2.5}$ objective includes a planning goal (shown in brackets)
e. WHO Air Quality Guidelines for Europe (2000)
f. WHO Air Quality Guidelines (2006 and 2010)
[1] $PM_{2.5}$ from combustion sources is diesel particulate matter (DPM)
[2] CCME Proposed Air Quality Standards for $PM_{2.5}$ for 2015 and 2020
* - The Ambient Ratio Method (ARM) has been applied to the 1-hour NO_x results, which includes background in the calculation per the BC AQMG.
** NO_2 100% refers to 100% conversion of NOx to NO_2 and predicted concentrations from Project emissions include background
Bold – maximum concentration exceeds the AAQO

As summarized in Table 3-2, the maximum predicted concentrations were at the FSD fenceline (i.e., maximum receptor). In addition, Levelton (2014) predicted air concentrations for the maximum North Delta residential receptor. In their determination of the predicted air concentrations for the maximum North Delta residential receptor, Levelton (2014) evaluated the set of residential receptors depicted on the following figure. In the below figure, the FSD fenceline is depicted as a blue polygon, and the set of residential receptors are shown as yellow crosses. It is noted that a subset of these receptors includes the nearest sensitive receptors (e.g., schools, daycares) located nearest to the FSD facility, and predicted air concentrations at these sensitive receptors are less than the concentrations predicted at the maximum North Delta residential receptor.

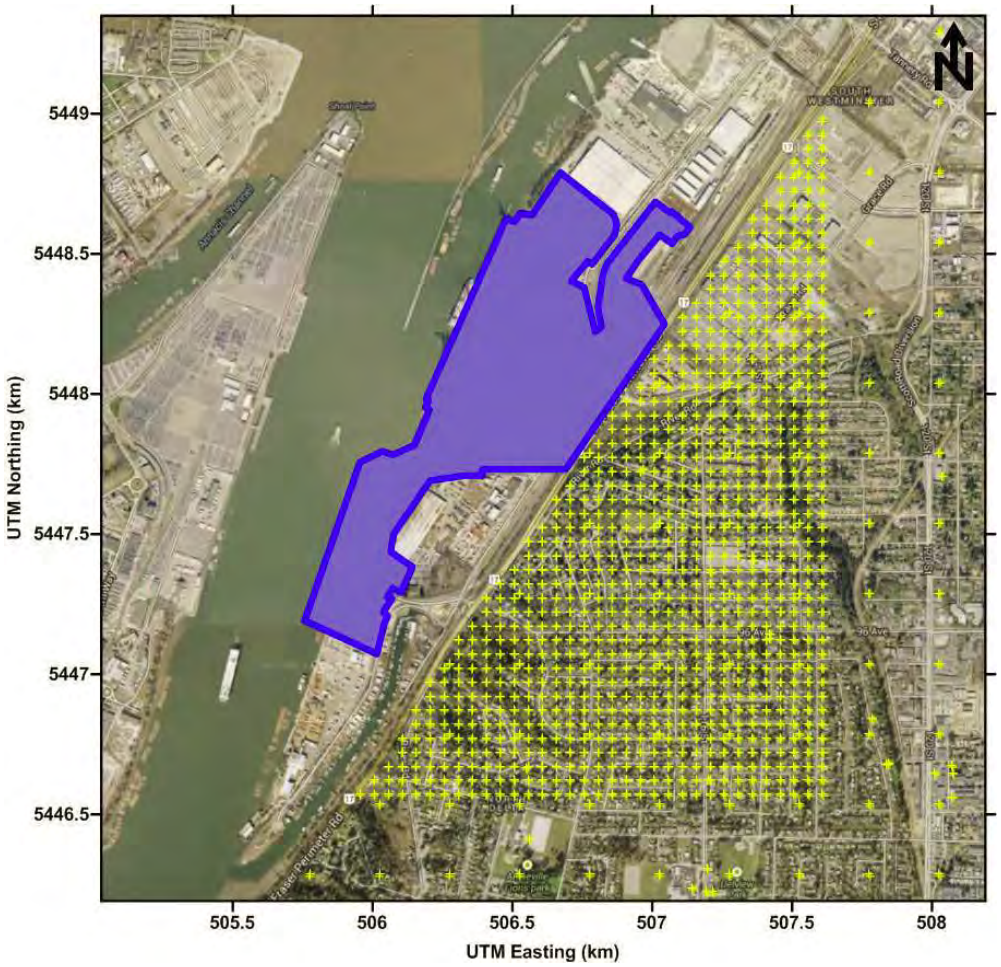

Figure 3-3: Residential Receptor Locations Considered by Levelton (2014) in the Maximum North Delta Residential Receptor Analysis.

The concentrations predicted at the FSD fenceline and at the maximum North Delta residential receptor are considered representative of maximum Project emissions in the immediate area of the facility; the predicted concentrations of the CACs in this area will decrease with distance from the facility. Results are presented as maximum concentrations associated with the Project, as well as maximum concentrations plus background at both the maximum receptor (along the FSD fenceline) and at the maximum North Delta residential receptor. For all parameters, with the exception of the maximum predicted annual NO_2, the maximum concentrations plus background were below the most stringent of the municipal, provincial, national and international air quality objectives and guidelines, including the CCME "Maximum Desirable" and the BC Level A levels, as well as the Metro Vancouver and BC $PM_{2.5}$ planning goal. The 24-hour and annual $PM_{2.5}$ concentrations were also below the CCME proposed Air Quality Standards for 2015 and 2020 of 28 µg/m^3 and 10 µg/m^3 (for 2015) and 27 µg/m^3 and 8.8 µg/m^3 (for 2020), respectively (CCME, 2012).

The model predicted higher annual NO_2 concentrations in the region concentrated over the Fraser River, including at the fenceline in the immediate area of the berth. Levelton (2014) indicates that there were a total of 21 receptors out of the total 10,043 modelled receptors surrounding FSD where predicted annual NO_2 concentrations exceeded the annual NO_2 AAQO. Eleven of these receptors were located at the FSD fenceline, while the remaining 10 are localized on the west side FSD, over the Fraser River in the area where the tugs operate. A location plot of these 21 receptors (shown as yellow crosses), as presented in Levelton (2014) is presented as Figure 3-4 on the following page. Predicted annual average concentrations of NO_2 decrease with distance from the FSD fenceline, and are predicted to be below the AAQO of 40 µg/m^3 approximately 200 m from the fenceline, in an area over the Fraser River.

It is noted that the predicted concentrations of select CACs presented in Table 3-2 differ from those presented in the EIA (SNC-Lavalin, 2013). The rationale for these differences is provided in Levelton (2014) and is summarized here:

- The Levelton (2014) AQA is based on FSD receiving one 135 car coal train a day, whereas the previous AQA (Levelton, 2013a) assumed one 125 car coal train per day;
- The design of the facility has been revised, with the Levelton (2014) AQA based on the re-design;
- The in-transit modeling approach (i.e., emissions from moving coal trains) was further refined; and
- As determined in consultation with Metro Vancouver, the fenceline for the FSD facility has been adjusted.

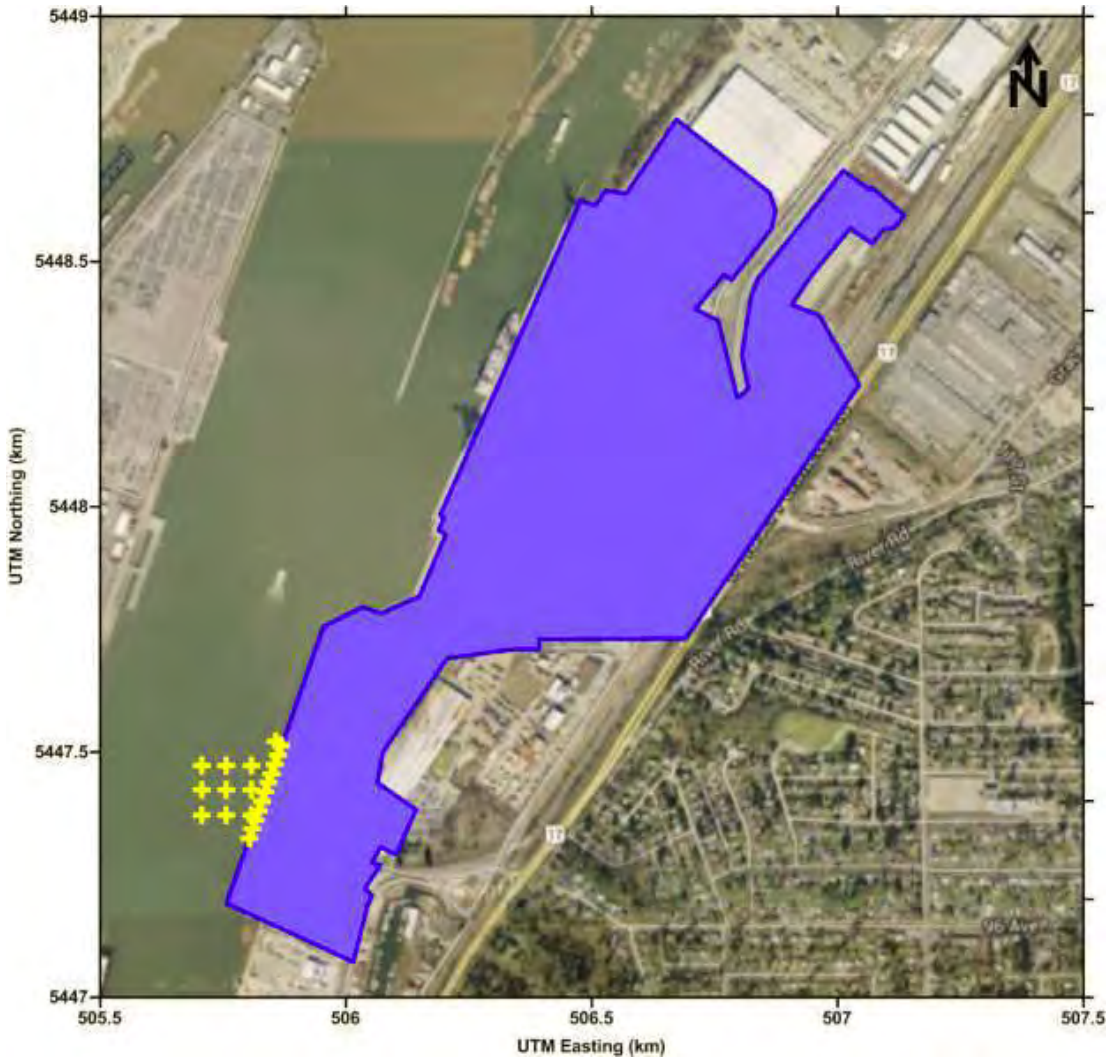

Figure 3-4: Map of Receptors with Predicted NO_2 Concentrations Greater than the Annual NO_2 AAQO of 40 µg/m^3 (from Levelton, 2014).

The Levelton (2014) AQA also predicted concentrations of the CACs along the rail corridor from the Canada-US Border, to FSD, and along the barge route, from FSD to the mouth of the Fraser River, by modelling a series of area sources representing either a passing train or barge at different locations along the transit route within Metro Vancouver. The modelled area sources along the rail corridor and barge route are depicted on Figure 3-5 below.

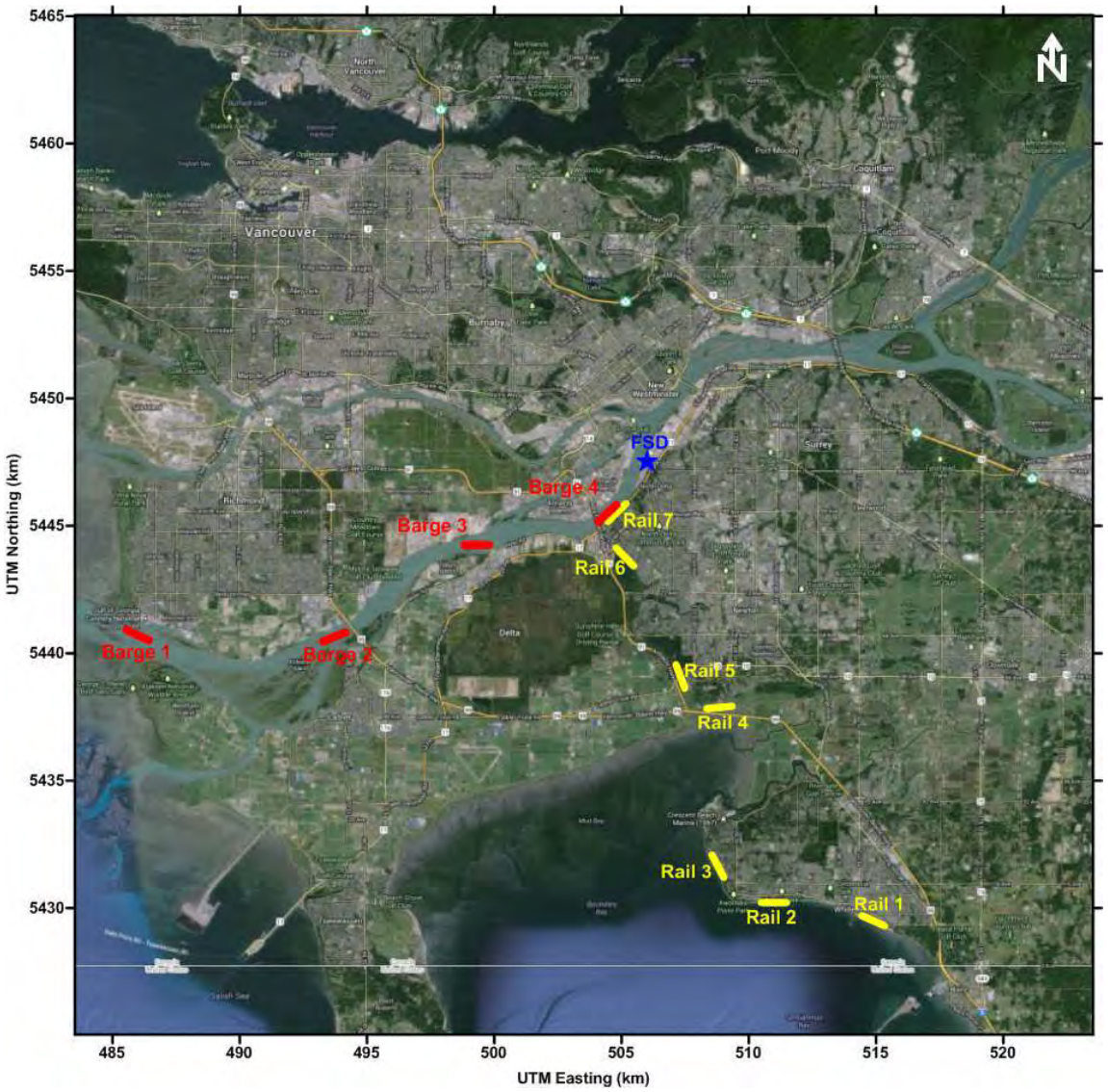

Figure 3-5: Location of the In-Transit Rail and Barge Study Areas (from Levelton, 2014).

The results of the in-transit modelling for the rail and barge study areas presented in Figure 3-5 are presented below in Table 3-3. The maximum predicted concentrations are given as ranges, where the low end represents the minimum predicted maximum concentration from all study areas and the high end represents the maximum predicted maximum concentration from all study areas. Table 3-3 also presents the maximum predicted concentrations plus background.

Table 3-3: Results of Levelton (2014) AQA: Maximum In-Transit Predicted Concentrations for Rail Corridor and Barge Route

CAC	Averaging Time	Metro Vancouver[a] (µg/m^3)	CCME[b]			British Columbia[d]			WHO (µg/m^3)	Back-ground (µg/m^3)	Maximum Concentration for Rail Corridor (µg/m^3)						Maximum Concentration for Barge Route (µg/m^3)					
											Maximum Receptor Along Rail Corridor			Maximum Receptor Along Rail Corridor + Background			Maximum Receptor Along Barge Route			Maximum Receptor Along Barge Route + Background		
			Maximum Desirable (µg/m^3)	Maximum Acceptable (µg/m^3)	Maximum Tolerable (µg/m^3)	Level A (µg/m^3)	Level B (µg/m^3)	Level C (µg/m^3)			Coal	Combustion	Total	Coal	Combustion	Total	Coal	Combustion	Total	Coal	Combustion	Total
CO	1-hour	30,000	15,000	35,000	-	14,300	28,000	35,000	30,000[e]	617	-	45.32 to 83.41	45.32 to 83.41	-	662.77 to 700.86	662.77 to 700.86	-	18.65 to 21.53	18.65 to 21.53	-	636.10 to 638.99	636.10 to 638.99
	8-hour	10,000	6,000	15,000	20,000	5,500	11,000	14,300	10,000[e]	554	-	26.07 to 55.44	26.07 to 55.44	-	580.91 to 610.27	580.91 to 610.27	-	10.80 to 12.71	10.80 to 12.71	-	565.63 to 567.55	565.63 to 567.55
NO_2 (ARM*)	1-hour	200	-	400	1,000	-	-	-	200[f]	66*	-	91.31 to 97.05	91.31 to 97.05	-	91.31 to 97.05	91.31 to 97.05	-	88.96 to 90.07	88.96 to 90.07	-	88.96 to 90.07	88.96 to 90.07
NO_2 (100%**)	Annual	40	60	100	-	-	-	-	40[f]	27	-	2.73 to 5.87	2.73 to 5.87	-	29.78 to 32.93	29.78 to 32.93	-	1.68 to 2.80	1.68 to 2.80	-	28.73 to 29.85	28.73 to 29.85
SO_2	1-hour	450	450	900	-	450	900	900	-	28	-	0.16 to 0.29	0.16 to 0.29	-	27.87 to 28.00	27.87 to 28.00	-	0.11 to 0.12	0.11 to 0.12	-	27.82 to 27.84	27.82 to 27.84
	24-hour	125	150	300	800	160	260	360	20[f]	18	-	0.005 to 0.01	0.005 to 0.01	-	17.84 to 17.84	17.84 to 17.84	-	0.003 to 0.004	0.003 to 0.004	-	17.84 to 17.84	17.84 to 17.84
	Annual	30	30	60	-	25	50	80	-	4	-	0.001 to 0.003	0.001 to 0.003	-	3.78 to 3.78	3.78 to 3.78	-	0.0008 to 0.001	0.0008 to 0.001	-	3.78 to 3.78	3.78 to 3.78
PM_{10}	24-hour Rolling	50	-	-	-	50	-	-	50[f]	27	1.14 to 2.47	0.25 to 0.54	1.39 to 3.01	27.89 to 29.22	27.00 to 27.30	28.14 to 29.77	0.00 to 1.08	0.12 to 0.16	0.14 to 1.09	26.75 to 27.84	26.88 to 26.92	26.89 to 27.84
	Annual	20	-	-	-	-	-	-	20[f]	12	0.31 to 0.67	0.07 to 0.15	0.38 to 0.81	12.32 to 12.67	12.08 to 12.15	12.39 to 12.82	0.01 to 0.04	0.03 to 0.05	0.04 to 0.07	12.01 to 12.04	12.04 to 12.06	12.05 to 12.07
$PM_{2.5}$	24-hour Rolling	25	30[c] (2015: 28)[2] (2020: 27)[2]	-	-	25	-	-	25[f]	12	0.46 to 0.99	0.24 to 0.53[1]	0.70 to 1.52	12.99 to 13.52	12.78 to 13.06[1]	13.23 to 14.05	0.00 to 0.16	0.11 to 0.15[1]	0.13 to 0.17	12.53 to 12.69	12.65 to 12.68[1]	12.66 to 12.70
	Annual	8(6)	(2015: 10)[2] (2020: 8.8)[2]	-	-	8(6)	-	-	10[f]	4	0.12 to 0.27	0.07 to 0.14[1]	0.19 to 0.41	4.57 to 4.71	4.51 to 4.59[1]	4.64 to 4.86	0.00 to 0.01	0.03 to 0.04[1]	0.03 to 0.04	4.45 to 4.45	4.47 to 4.49[1]	4.48 to 4.49
TPM	24-hour	120	-	-	-	-	-	-	-	56	2.16 to 4.23	0.24 to 0.46	2.40 to 4.69	57.90 to 59.96	55.98 to 56.20	58.13 to 60.43	0.00 to 2.09	0.13 to 0.16	0.13 to 2.10	55.74 to 57.83	55.86 to 55.90	55.86 to 57.84
	Annual	60	-	-	-	-	-	-	-	25	0.62 to 1.33	0.07 to 0.15	0.69 to 1.48	25.74 to 26.45	25.19 to 25.27	25.81 to 26.60	0.00 to 0.07	0.03 to 0.05	0.04 to 0.10	25.12 to 25.19	25.15 to 25.17	25.16 to 25.22
Dustfall	Annual	1.7 (mg/dm^2/ day)	-	-	-	-	-	-	-	0.5	0.01 to 0.02	0.00002 to 0.00008	0.01 to 0.02	0.51 to 0.52	0.50 to 0.50	0.51 to 0.52	0.00 to 0.001	3.9E-13 to 4.0E-04	2.5E-05 to 1.2E-03	0.50 to 0.50	0.50 to 0.50	0.50 to 0.50

Notes:

a. Metro Vancouver Ambient Air Quality Objectives (2011); the annual $PM_{2.5}$ objective includes a planning goal (shown in brackets)
b. CCME National Ambient Air Quality Objectives (1999)
c. CCME Canada Wide Standards (2000)
d. B.C. Ambient Air Quality Objectives (2013); the annual $PM_{2.5}$ objective includes a planning goal (shown in brackets)
e. WHO Air Quality Guidelines for Europe (2000)
f. WHO Air Quality Guidelines (2006 and 2010)
[1] $PM_{2.5}$ from combustion sources is diesel particulate matter (DPM)
[2] CCME Proposed Air Quality Standards for $PM_{2.5}$ for 2015 and 2020
* - The Ambient Ratio Method (ARM) has been applied to the 1-hour NO_x results, which includes background in the calculation per the BC AQMG.
** NO_2 100% refers to 100% conversion of NOx to NO_2
Bold – maximum concentration exceeds the AAQO

As summarized in Table 3-3, the maximum (high-end of the range) predicted concentrations of the CACs along both the rail corridor and barge route, as well as the maximum plus background, were below the most stringent of the municipal, provincial, national and international air quality objectives and guidelines, including the CCME "Maximum Desirable" and the BC Level A levels, as well as the Metro Vancouver and BC $PM_{2.5}$ planning goal. The 24-hour and annual $PM_{2.5}$ concentrations were also below the CCME proposed Air Quality Standards for 2015 and 2020 of 28 µg/m^3 and 10 µg/m^3 (for 2015) and 27 µg/m^3 and 8.8 µg/m^3 (for 2020), respectively (CCME, 2012).

As detailed in Levelton (2014), the in-transit modelling included the identification of maximum receptors along the sections of the rail corridor and the barge route. For the rail corridor, the concentrations presented in Table 3-3 represent a maximum receptor located at a distance of 5 m from the rail corridor. Given the proximity of residential properties to the railway tracks in some neighbourhoods along the rail corridor, although homes would not be present within 5 m of the rail corridor, it has been assumed that the predicted concentrations represent concentrations that residents along the rail corridor could be exposed to. This is further discussed in Section 3.4 of the report.

For the barge route, the concentrations presented in Table 3-3 represent a maximum receptor located over the water on the Fraser River. Levelton (2014) indicated that the predicted CAC concentrations along the barge route decrease with distances from the barge(s), with concentrations typically reaching background levels before or at the shoreline. In addition, the CAC concentrations predicted at the maximum receptor along the barge route (i.e., over the water) were lower than those predicted at the maximum North Delta residential receptor and/or the maximum rail corridor residential receptor.

In addition to the above, Levelton determined the constituents of the PM and volatile organic compounds (VOC) emissions predicted for the Project. Levelton conducted research to identify the most current transportation equipment engine combustion-related PM and VOC speciation profiles that are available from published sources and are relevant to the following key operations at FSD. The methods and results of the research were provided in Levelton (2014). The following sources were speciated:

- Marine – Tugboats;
- Rail - Line-haul locomotive;
- Rail - Yard-switcher locomotive; and,
- Non-road equipment - Front-end loaders.

The results indicated that several metals, metalloids, polycyclic aromatic hydrocarbons (PAHs), sulfates and carbon are constituents of the PM10 and PM2.5 emissions from the above sources. In addition, several VOCs, for example, acrolein, benzene, 1,3-butadiene, ethylbenzene, formaldehyde, n-hexane, toluene, xylenes, and several PAHs, were identified to be associated with the transportation equipment engine combustion emissions from the above sources. The transportation equipment engine combustion emission profiles for the above sources were considered in the determination of the COPCs for the Project.

The results of the AQA discussed above were considered in the identification of the COPCs for the Project (Section 3.3), as well as in the Project Scenario and Cumulative Scenario assessments presented in the subsequent sections of the HHRA.

3.2.2 Coal Analysis

The Project will transfer sub-bituminous coal. Sub-bituminous coal accounts for about 38 percent of Canada's coal production. Sub-bituminous coal is non-coking and has less sulphur but more moisture (approximately 10 to 45 percent) and volatile matter (i.e., components of coal, except for moisture, which are liberated at high temperature in the absence of air) (up to 45 percent) than bituminous coals. Carbon content is 35-45 percent and ash ranges up to 10 percent. Sulphur content is generally under 2 percent by weight. In addition to the major elements, sub-bituminous coal contains a large number of other minor elements (metals and metalloids) in trace amounts including, but not limited to, arsenic, cadmium, chromium, lead, mercury, selenium, and uranium. Further, sub-bituminous coal contains PAHs. Metals/metalloids and PAHs present in the coal could represent a concern with respect to the potential for health effects related to coal dust exposures. It should be noted that the metals/metalloids and PAHs in coal are generally not bioavailable under typical environmental conditions (Ahrens and Morrisey, 2005; Triton, 2013a).

To determine the concentrations of metals/metalloids and PAHs in the coal that is proposed to be transported as part of the Project, coal samples were obtained from the producers that will supply coal to FSD (i.e., the source coal) and submitted to ALS Environmental in Burnaby, BC (ALS) for laboratory analysis of metals/metalloids and PAHs, as agreed upon with PMV/Golder. A total of 12 coal samples were submitted for analysis of total metals (including metalloids) and chromium speciation (i.e., concentrations of both chromium III and chromium VI were determined). In addition, a total of 20 samples were submitted for analysis of PAHs. Finally, 12 samples were submitted for analysis of crystalline silica. Table 3-4 presents a summary of the metals/metalloids and PAHs analysed for in the coal samples submitted for analysis.

Table 3-4: Metals/Metalloids and PAHs Analysed in the Source Coal

Metals/Metalloids			PAHs	
Antimony	Lead	Tin	Acenaphthene	Chrysene
Arsenic	Lithium	Uranium	Acenaphthylene	Dibenz(a,h)anthracene
Barium	Manganese	Vanadium	Anthracene	Fluoranthene
Beryllium	Mercury	Zinc	Benz(a)anthracene	Fluorene
Cadmium	Molybdenum	Aluminum	Benzo(a)pyrene	Indeno(1,2,3-cd)pyrene
Chromium (+6)	Nickel	Bismuth	Benzo(b)fluoranthene	Naphthalene
Chromium (+3)	Selenium	Boron	Benzo(ghi)perylene	2-Methylnaphtalene
Cobalt	Silver	Iron	Benzo(j)fluoranthene	Phenanthrene
Copper	Strontium	Thallium	Benzo(k)fluoranthene	Pyrene
		Titanium		

As noted previously, the metals/metalloids and PAHs in coal are generally not bioavailable under typical environmental conditions (Ahrens and Morrisey, 2005; Triton, 2013a). To further assess the bioaccessibility of select metals, 6 samples of the source coal were submitted to Royal Road University for analysis using the Physiologically Based Extraction Test (PBET); PBET is an *in vitro* test system which simulates the human gastrointestinal tract conditions and is used estimate the bioaccessibility of metals from a solid matrix, in this case coal. Health Canada supports the use of PBET results in the estimation of oral/ingestion exposures to lead. Consequently the PBET results for lead have been used in the estimation of soil ingestion exposures to these metals in Section 4, the Exposure Assessment.

The laboratory results for metals/metalloids and PAHs provided the percent composition of each of the individual constituents in the source coal, and were subsequently used in the Exposure Assessment to estimate exposure point concentrations for each of these specific parameters in coal dust. The results of the PBET were also used in the Exposure Assessment in the in the estimation of soil and vegetation ingestion exposures.

The laboratory results for the coal samples for total metals, chromium speciation, crystalline silica and PAHs are presented in Tables VII-1 to VII-3 in Appendix VII, with the PBET results presented in Table VII-4. The mean concentrations of the metals/metalloids and PAHs measured in the coal samples were used to estimate the percent composition of each of the constituents in the coal.

Statistical analysis of the chemistry results for the coal samples indicates that the data for metals/metalloids and PAHs in the coal are normally distributed for 45 of the 51 parameters analysed for; these 45 parameters have concentrations within two standard deviations of the mean. Health Canada (2010a) indicates that when supported by the data, the mean or upper 95% confidence interval of the mean should be used to estimate contaminant

exposures. Given that the data for 88% of the metals/metalloids and PAHs are normally distributed, it was considered appropriate to use mean concentrations in the prediction of exposure concentrations in the HHRA. The results of the statistical analysis of the coal samples is presented in Appendix VI, with the mean concentrations of the metals/metalloids and PAHs in the coal presented below in Table 3-5.

Table 3-5: Mean Concentrations of Metals/Metalloids, PAHs and Crystalline Silica Measured in Coal

Constituent	Mean Concentration (µg/g)	Constituent	Mean Concentration (µg/g)	Constituent	Mean Concentration (µg/g)
Polycyclic Aromatic Hydrocarbons (PAHs)		***Metals/Metalloids & Crystalline Silica***			
Benzo(a)pyrene	0.24	Antimony	< DL (0.1)	Selenium	0.65
Benzo(a)anthracene	0.58	Arsenic	1.3	Strontium	221
Benzo(b)fluoranthene	0.85	Barium	352	Silver	< DL (0.05)
Benzo(g,h,i)perylene	0.06	Beryllium	0.2	Tin	0.2
Benzo(k)fluoranthene	0.18	Cadmium	0.1	Uranium	0.4
Chrysene	0.35	Chromium (+6)	< DL (0.1)	Vanadium	11
Dibenzo(a,h)anthracene	<DL (0.05)	Chromium (+3)	3	Zinc	14
Fluoranthene	1.64	Cobalt	1.3	Aluminum	2268
Indeno(1,2,3-cd)pyrene	< DL (0.09 – 0.2)	Copper	13	Bismuth	< DL (0.1)
Phenanthrene	0.40	Lead	2	Boron	18
Acenaphthene	0.36	Lithium	< DL (5)	Iron	2528
Acenaphthylene	0.10	Manganese	19	Thallium	< DL (0.05)
Anthracene	0.20	Mercury	0.08	Titanium	157
Fluorene	< DL (0.08 – 0.2)	Molybdenum	0.5	Crystalline Silica	< DL (0.75 – 1.0)
Naphthalene	0.07	Nickel	4		
2-Methylnaphthalene	0.07				
Pyrene	2.08				

< DL – parameter was not detected in any of the coal samples submitted for analysis; the detection limit/range of detection limits for the coal samples is presented in parentheses

The results of the coal analysis were used to estimate the concentrations of each of the metal/metalloid and PAH constituents in coal dust, which were subsequently used to estimate exposures in the Project Scenario and Cumulative Scenario assessments.

3.2.3 Background Soil Assessment

As indicated, metals/metalloids and PAHs are components of coal/coal dust; however, these parameters are also present in soil from both natural and anthropogenic sources. As per standard HHRA methods to assess overall

exposures to these parameters, the background soil concentrations of metals/metalloids (including chromium III and chromium VI) and PAHs in the Study Area were determined through a background soil assessment.

The assessment included the collection of surface soil samples from areas where sensitive receptors have the potential to be present near the FSD facility (e.g., the nearest residential neighbourhood), as well as along the rail corridor and barge route. Samples were collected from four municipalities in the Study Area, including the cities of Delta, Richmond, Surrey and White Rock. The number of samples collected from each municipality and submitted for analysis of total metals, chromium speciation and PAHs is summarized in Table 3-6.

Table 3-6: Background Soil Assessment: Number of Surface Soil Samples Collected

Parameter	Total Number of Samples from the Municipalities				Duplicate Samples	Number of analyses
	Delta	Richmond	Surrey	White Rock		
Total Metals and Cr (VI)	11	11	13	13	5	53
PAHs	11	11	13	13	5	53

The laboratory results for the background soil samples for total metals, chromium speciation and PAHs are presented in Tables VII-5 to VII-8 in Appendix VII. The mean concentrations of the metals/metalloids and PAHs measured in the background soil samples were used to estimate the background soil concentration for each of the parameters. The mean concentrations of the metals/metalloids and PAHs are presented in Table 3-7.

Table 3-7: Mean Concentrations of PAHs and Metals/Metalloids Measured in Background Soil Samples Collected in Surrey, Delta, Richmond and White Rock

Constituent	Mean Concentration (µg/g)
Polycyclic Aromatic Hydrocarbons (PAHs)	
Benzo(a)pyrene	0.40
Benzo(a)anthracene	0.34
Benzo(b)fluoranthene	0.60
Benzo(g,h,i)perylene	0.25
Benzo(k)fluoranthene	0.24
Chrysene	0.40
Dibenzo(a,h)anthracene	0.06
Fluoranthene	0.86
Indeno(1,2,3-cd)pyrene	0.27
Phenanthrene	0.52
Acenaphthene	0.02

Constituent	Mean Concentration (µg/g)
Polycyclic Aromatic Hydrocarbons (PAHs) (Cont'd)	
Acenaphthylene	0.03
Anthracene	0.08
Fluorene	0.04
Naphthalene	0.02
2-Methylnaphthalene	0.02
Pyrene	0.70
Metals & Metalloids	
Antimony	0.5
Arsenic	6
Barium	67
Beryllium	0.2
Cadmium	0.2
Chromium (+6)	0.2
Chromium (+3)	26
Cobalt	7.4
Copper	25
Lead	22
Lithium	9
Manganese	355
Mercury	0.05
Molybdenum	1.1
Nickel	21
Selenium	0.3
Strontium	68
Silver	0.1
Tin	2
Uranium	0.8
Vanadium	45
Zinc	83
Aluminum	13550
Bismuth	< DL (0.2)
Boron	---
Iron	18715
Thallium	0.06
Titanium	744

< DL – parameter was not detected in any of the coal samples submitted for analysis; the detection limit/range of detection limits for the coal samples is presented in parentheses.

It is noted that the mean concentrations of majority of the metals (antimony, arsenic, bismuth, cadmium, chromium (+3), chromium (+6), cobalt, copper, lead, lithium, manganese, molybdenum, nickel, silver, tin, uranium, vanadium, zinc, aluminum, iron, thallium and titanium) and select PAHs (benzo(a)pyrene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene and phenanthrene) in the background soil samples were higher than those measured in the coal samples. The significance of this is further discussed in Section 4 of the report.

The results of the background soil assessment were used to estimate direct and indirect soil exposures in the Baseline Scenario assessment, as well as in the Cumulative Scenario assessment (Baseline Scenario + Project Scenario).

3.2.4 Dust Palliatives Review

To support the HHRA, SNC-Lavalin conducted a review of the dust palliative or dust suppressant agents proposed for use at FSD, as well as those likely to be used on the coal transported to FSD by rail. Dust mitigation is an integral component of the overall Project design. Mitigation measures have been developed to address potential fugitive dust from several sources including:

1) Coal rail cars in transit;
2) The unloading of coal from rail cars at FSD;
3) Material transfer through covered conveying systems at FSD;
4) The loading of coal to barges at FSD; and
5) Coal barges in transit between FSD and Texada Island.

Dust palliatives are proposed for use at all stages of the coal transfer process, and use of these products will reduce the potential for coal dust (i.e., suspended dust) to be released to the surrounding environment (i.e., will reduce coal dust exposures). Dust control on rail will be applied by Burlington Northern Santa Fe Railway (BNSF) and the mine site operators and will consist of:

- Applying ‘body agent’ as required at the mine site to help bind coal particles to reduce dust losses;
- Addition of a secondary ‘body agent’ as required to reduce coal oxidation;
- Addition of ‘topping agent’ when coal is loaded into the railcar at the mine site to act as a sealant to prevent dust losses; and

- Reapplication of 'topping agent' approximately at midpoint of the rail movement from the mine site to FSD to address concerns regarding potential degradation of the topping agent during transit.

For the barge transport from FSD to Texada Island, FSD will apply a combination of two products to coal at the FSD site as the barge is loaded in order to minimize potential dust emissions during the barge journey and at the FSD facility, and therefore potential exposures to coal dust. General Electric Water and Process Technologies (GE) is the likely supplier of the products that will be applied on site at FSD, which will be applied using a technique that will allow application to the entire coal surface, rather than just the top layer of coal, which will provide greater effectiveness.

A review of the MSDS for dust palliatives proposed for use by BNSF and FSD was conducted. In addition, as presented in Section 5 of the report (Toxicity Assessment), a review of the TRVs, which represent acceptable levels of exposures, for the constituents of the dust suppressants was conducted to evaluate potential significance to human health. A summary of the composition of the products is presented in Table 3-8.

Table 3-8: Composition of Dust Palliatives

Trade Name/Manufacturer	Composition	
	Hazardous Component	Percentage (%)
DUSTREAT DC9138E / GE	Oxirane, Methyl- (Propylene Oxide)	7-13
	Linear Alkyl Sulfonate	Not Reported
	Diethylaminoethanol	1-5
DUSTREAT DC9144 / GE	None. MSDS indicates that 'Product is not hazardous as defined by OSHA regulations.'	
DUSTREAT DC9148 / GE	Adipic Acid, Diethylenetriamine, Epichlorohydrin polymer	30-60
DUSTREAT DC9149 / GE	Propylene Glycol	30-60
	Acid, Sulfo-1,4-bis (2-ethylhexyl) ester, sodium salt (succinic acid)	30-60
AKJ CTS-100 / AKJ Industries	Vinyl acetate-dioctyl maleate polymer	2-15
	Proprietary Additive	0.1-2
AKJ CTS-100C / AKJ Industries	Polyvinyl Acetate	25-55
	Proprietary Additive	<2
SOIL-SEMENT® Coal Car Topper / Midwest Industrial Supply	Acrylic & Vinyl Acetate Polymer	5-50
Dustbind Plus / Nalco	Alkyl Alcohol	30-60
Rantec® Capture 3000_ L Liquid Concentrate / Rantec	Guar Gum	40-50
	Soy Bean Oil	40-50
	Organophillic Clay	1-1.5
	Oleic Acid	1-1.5
	Surfactant, nonylphenyl	0.8-1
	Propylene Carbonate	<0.2
MINTOPPER_ S+0150 / Mintech Enterprises	None. MSDS indicates 'This product does not contain a toxic chemical subject to the reporting requirements of Section 313 of the Emergency Planning and Community Right-To-Know Act of 1986.'	

As previously discussed, dust palliatives will be applied to reduce potential exposures to coal. The HHRA evaluates the potential for exposures to both coal dust, and the constituents of the dust palliatives.

Only the constituents of the GE products are further evaluated in the HHRA. The GE products are those likely to be used at the FSD facility, and application rates for these products are available and were used in the determination of concentrations of these constituents in coal dust. It is acknowledged that other dust palliatives could be applied at the mine sites, with re-application at an in-transit mid-point location, however, exposures to these agents in the Study Area is anticipated to be lower than the GE products being applied at FSD. Furthermore, based on the composition of the GE products compared to that of the other dust palliatives and a comparison of the available TRVs for the constituents (as presented in Section 5 and Appendix V), evaluation of the GE products is considered to also be protective of the other agents based on their relative toxicity..

The chemical constituents of the dust palliatives were considered in the determination of the COPCs for the Project, as is further discussed in Section 3.3.

3.3 COPC Screening

The chemicals (or substances) of potential concern (COPCs) for the Project were identified through the development of an inventory of chemicals emitted from the Project to which humans may be exposed. Since the Project will not directly release chemicals to surface water or soil, the COPCs for the HHRA were based on air emissions only. According to the AQA (Levelton, 2014), atmospheric emissions associated with the Project include the following:

- The CACs, namely PM_{10}, $PM_{2.5}$, CO, NO_2, and SO_2, from coal dust, emissions from the agricultural operations at FSD, and/or from combustion emissions;
- TPM and DPM from dust and combustion emissions; and,
- Several metals, PAHs and carbon identified to be constituents of the PM_{10} and $PM_{2.5}$ from transportation equipment combustion emissions, and several VOCs were also identified to be associated with the transportation equipment combustion emissions.

The CACs, TPM, DPM and the metals, PAHs and VOCs identified as constituents of combustion emissions from transportation equipment were evaluated as COPCs for the Project, with the following exceptions:

- Calcium, elemental and organic carbon, magnesium, phosphorus, silicon and sodium identified as constituents of combustion emissions. These constituents were not included based on their innocuous nature.

Additionally, the metals, metalloids and PAHs constituents of coal/coal dust were identified as COPCs for the Project, with the exception of the following:

- Calcium, phosphorus and magnesium in coal dust. These constituents were not included based on their innocuous nature; and,
- Antimony, bismuth, chromium (VI), crystalline silica, lithium, silver, thallium, dibenzo(a,h)anthracene, fluorene, ideno(1,2,3-c,d)pyrene. These parameters were not detected in any of the coal samples submitted for analysis, and therefore were not retained as COPCs related to coal dust. It is noted some of these substances were predicted to be present in the combustion emissions and, therefore, were retained as COPCs for that source.

Furthermore, the chemical constituents of the GE dust palliatives, as summarized in Section 3.2.4, were identified as COPCs.

For the purpose of this HHRA, the COPCs were divided into two groups, based on physical and chemical properties of the COPC:

- Gaseous COPCs (e.g., CO, NO_2, SO_2, most VOCs). Gaseous COPCs will be present in air, with human exposures of toxicological relevance to these COPCs limited to inhalation; and
- Non-gaseous COPCs (e.g., metals, PAHs). Non-gaseous COPCs have the potential to be deposited from the atmosphere to soil or other surfaces (e.g., garden produce) in the Study Area. These COPCs have the potential to accumulate in the environment and humans may be exposed to these COPCs through the direct soil pathways and/or through the consumption of produce. The non-gaseous COPCs were included in the multi-media assessment (i.e., receptors of concern have the potential to be exposed to these COPCs in air, soil and vegetation).

All metals were considered to be non-gaseous COPCs, and the CACs, including $PM_{2.5}$, PM_{10}, CO, NO_2, SO_2 and DPM were considered to be gaseous COPCs. The organic COPCs (e.g., PAHs, VOCs) were evaluated using the following approach/criteria to determine whether they would be evaluated as gaseous or non-gaseous COPCs:

- As per the BC Ministry of Environment's (MoE) definition of a volatile substance, if the Henry's Law constant for the COPC is > 1.0 x 10-5 atm-m3/mol and the vapour pressure is > 0.05 Torr @ 25oC, the COPC was considered to be volatile (BC MoE definition available at http://www.env.gov.bc.ca/epd/wamr/labsys/bclqaac/sched11_recs_july30_08.pdf).

In addition, COPCs that were determined to be volatile (i.e., the gaseous COPCs) were further assessed for their bioaccumulation potential using the following BC MoE definition of a bioaccumulative substance:

- COPCs with a log octanol water partition coefficient (log Kow) > 5 were considered to be bioaccumulative substances (BC MoE, 2014b).

COPCs that were determined to be non-volatile were considered to be non-gaseous COPCs, COPCs that were determined to be volatile but not bioaccumulative were evaluated as gaseous COPCs only (i.e., present in air, with human exposures to these COPCs limited to inhalation), and COPCs that were determined to be volatile and bioaccumulative were included in the multi-media assessment. It is noted that two of the PAHs, naphthalene and 2-methylnaphthalene, were considered to be semi-volatile and, therefore, these have been evaluated as both gaseous (in air) and non-gaseous (in the multi-media assessment) COPCs.

Table 3-9 provides a summary of the COPCs identified for the Project. The sources of the COPCs (i.e., coal, combustion sources or agricultural operations) are indicated. In addition, whether the COPCs have been identified as a gaseous or non-gaseous COPC is indicated in the final column of the table.

Table 3-9: Summary of Project COPCs

Constituent	Sources			Gaseous or Non-Gaseous?
	Coal	Combustion	Agricultural Operations	
Polycyclic Aromatic Hydrocarbons (PAHs)				
Carcinogenic PAHs				
Benzo(a)pyrene	X	X		Non-Gaseous
Benzo(a)anthracene	X	X		Non-Gaseous
Benzo(b)fluoranthene	X	X		Non-Gaseous

Constituent	Sources			Gaseous or Non-Gaseous?
	Coal	Combustion	Agricultural Operations	
Polycyclic Aromatic Hydrocarbons (PAHs) (Cont'd)				
Carcinogenic PAHs (Cont'd)				
Benzo(g,h,i)perylene	X	X		Non-Gaseous
Benzo(k)fluoranthene	X	X		Non-Gaseous
Chrysene	X	X		Non-Gaseous
Dibenzo(a,h)anthracene		X		Non-Gaseous
Fluoranthene	X	X		Non-Gaseous
Indeno(1,2,3-cd)pyrene		X		Non-Gaseous
Phenanthrene	X	X		Non-Gaseous
Non-Carcinogenic PAHs				Non-Gaseous
Acenaphthene	X	X		Non-Gaseous
Anthracene	X	X		Non-Gaseous
Fluorene		X		Non-Gaseous
Fluoranthene	X	X		Non-Gaseous
Naphthalene	X	X		Gaseous (semi-volatile, therefore also included in multi-media assessment)
2-Methylnaphthalene	X	X		Gaseous(semi-volatile, therefore also included in multi-media assessment)
Pyrene	X	X		Non-Gaseous
Metals & Metalloids				
Aluminum	X			Non-Gaseous
Antimony		X		Non-Gaseous
Arsenic	X	X		Non-Gaseous
Barium	X	X		Non-Gaseous
Beryllium	X	X		Non-Gaseous
Boron	X			Non-Gaseous
Cadmium	X	X		Non-Gaseous
Chromium (+6)		X		Non-Gaseous
Chromium (+3)	X	X		Non-Gaseous
Cobalt	X			Non-Gaseous
Copper	X	X		Non-Gaseous
Indium		X		Non-Gaseous
Iron	X	X		Non-Gaseous
Lanthanum		X		Non-Gaseous

Constituent	Sources			Gaseous or Non-Gaseous?
	Coal	Combustion	Agricultural Operations	
Metals & Metalloids (Cont'd)				
Lead	X	X		Non-Gaseous
Manganese	X	X		Non-Gaseous
Mercury	X	X		Non-Gaseous
Molybdenum	X	X		Non-Gaseous
Nickel	X	X		Non-Gaseous
Selenium	X	X		Non-Gaseous
Strontium	X			Non-Gaseous
Tin	X	X		Non-Gaseous
Titanium	X	X		Non-Gaseous
Uranium	X			Non-Gaseous
Vanadium	X	X		Non-Gaseous
Zinc	X	X		Non-Gaseous
Criteria Air Contaminants (CACs)				
PM_{10}	X	X	X	Gaseous
$PM_{2.5}$	X	X	X	Gaseous
CO		X		Gaseous
NO_2		X		Gaseous
SO_2		X		Gaseous
DPM		X		Gaseous
TPM	X	X	X	Gaseous
Volatile Organic Compounds				
Acetaldehyde		X		Gaseous
Acrolein		X		Gaseous
Benzene		X		Gaseous
1,3-Butadiene		X		Gaseous
Ethylbenzene		X		Gaseous
Ethylene		X		Gaseous
Formaldehyde		X		Gaseous
Hexachlorobenzene		X		Gaseous/ Bioaccumulative*
n-Hexane		X		Gaseous
Propionaldehyde		X		Gaseous
Propylene (1-Propene)		X		Gaseous
Toluene		X		Gaseous
2,2,4-Trimethylpentane		X		Gaseous
Styrene		X		Gaseous
Xylenes		X		Gaseous

Constituent	Sources			Gaseous or Non-Gaseous?
	Coal	Combustion	Agricultural Operations	
Dust Palliatives Chemical Constituents				
Adipic Acid[a]	X			Non-Gaseous
Diethylaminoethanol[a]	X			Non-Gaseous
Diethylenetriamine[a]	X			Non-Gaseous
Epichlorohydrin	X			Gaseous
Linear Alkyl Sulfonate	X			Non-Gaseous
Propylene Glycol	X			Non-Gaseous
Propylene Oxide	X			Gaseous
Succinic acid	X			Non-Gaseous
Others				
Polychlorinated Biphenyls (PCB)		X		Non-Gaseous
Sulfate		X		Non-Gaseous

[a] chemical constituents of the GE product Dustreat DC9148; listed on the MSDS as adipic acid, diethylenetriamine, epichlorohydrin polymer

* The log Kow for hexachlorobenzene is > 5.0 and therefore, it was considered to be bioaccumulative, and was retained for evaluation in the multi-media assessment.

The COPCs presented in Table 3-9 were carried forward for evaluation in subsequent sections of the HHRA.

3.4 Human Receptors of Concern

People in the Study Area with the highest potential health risks associated with Project emissions include individuals who are exposed to the greatest Project air concentrations and/or who are more sensitive to the Project emissions.

Populations with the highest potential exposure to Project emissions will be those that spend time in areas with the highest predicted emission concentrations. Exposures are dependent on both the concentrations to which a population is exposed, as well as the duration of exposure; in HHRA, the exposure duration is typically characterized based on land use, with residential (and agricultural) land uses assumed to have the longest exposure duration (i.e., 24 hours a day, 7 days a week, 52 weeks a year, for 10 years for direct inhalation exposures and 80 years for routes where deposited material is the source of intake). Therefore, both the areas with the highest predicted Project emissions and the land uses within the Study Area were considered when identifying populations with the potential to receive the highest exposures to Project emissions.

Within a population, subpopulations have varying degrees of susceptibility to Project emissions. By evaluating the most sensitive or susceptible subpopulations in the region, it can be asserted that potential risks to the larger population are not underestimated; this approach is commonly used in HHRAs. The HHRA considered both the demographics of the Study Area, by municipality and overall region, as well as Health Canada guidance on the identification of receptors of concern for evaluation in the HHRA, to identify appropriate sensitive/susceptible subpopulations for evaluation in the HHRA. Rationale and discussion on receptor selection is presented in the sections following.

3.4.1 Demographics of Project Area

Demographic data for the region and detailed quantitative human health risk assessment guidance (Health Canada, 2012) was compiled, and was considered in the HHRA to identify the most sensitive and/or susceptible receptors within the region. As summarized in Table 3-10, Health Canada (2012) has determined characteristics of the general population based on the following age categories:

- Infant (0-6 months);
- Toddler (7 months to 4 years);
- Child (5-11 years);
- Teen (12-19 years); and
- Adult (≥ 20 years).

Table 3-10: Health Canada (2010a, 2012) Receptor Characteristics by Age Group

Characteristic	Infant 0-6mo.	Toddler 7 mo-4yrs	Child 5-11 yrs	Teen 12-19 yrs	Adult ≥20 yrs
Body weight (kg)	8.2	16.5	32.9	59.7	70.7
Soil ingestion rate (kg/day)	0.00002	0.00008	0.00002	0.00002	0.00002
Inhalation rate (m^3/day)	2.2	8.3	14.5	15.6	16.6
Time spent outdoors (h/day)	1.5 [1]	1.5 [1]	1.5 [1]	1.5	1.5
Skin surface area (cm^2)					
Hands	320	430	590	800	890
Arms (upper and lower)	550	890	1,480	2,230	2,500
Legs (upper and lower)	910	1,690	3,070	4,970	5,720
Total body	3,620	6,130	10,140	15,470	17,640

Characteristic	Infant 0-6mo.	Toddler 7 mo-4yrs	Child 5-11 yrs	Teen 12-19 yrs	Adult ≥20 yrs
Food ingestion[2] (kg/day)					
Root vegetables	0.083	0.105	0.161	0.227	0.188
Other vegetables	0.072	0.067	0.098	0.12	0.137

Notes:
[1] Data not available; however, time spent outdoors assumed to be equivalent to that of adults based on the assumption that the infant, toddler, or child is accompanied by a parent or guardian during outdoor activity.

[2] Data are for "eaters" only; those reporting zero intake were excluded from the estimate.

As per Health Canada guidance (Health Canada, 2010a), critical receptors (i.e., individuals within a population that may be at greater risk) are typically identified for each class of contaminants and each land use. The critical receptor is normally the member of the applicable receptor group that is expected to receive the highest exposure to a chemical, expressed as an average daily intake on a per unit body weight basis (Health Canada, 2010a). This would include children, or individuals consuming greater than average proportions of country foods and other natural foods (e.g., Aboriginal communities). For threshold (i.e., non-carcinogenic) chemicals where all age classes are present, the toddler is typically evaluated as the critical receptor based on their higher rate of exposure compared to their body weight. For non-threshold (i.e., carcinogenic) chemicals where all age classes are present, the adult is typically evaluated as the critical receptor.

Critical sub-groups, or sensitive subpopulations, may also be characterized as those with physical characteristics or conditions that may result in an increased likelihood of adverse effect to a given level of exposure (e.g., the elderly, persons suffering from existing medical conditions). These individuals are typically considered by health agencies (e.g., Health Canada) in the derivation of TRVs; when deriving TRVs, health agencies apply safety or uncertainty factors (i.e., an intraspecies/human variability uncertainty factor) to protect for sensitive subpopulations. Critical receptors are further discussed below in Section 3.4.3.

As will be presented in subsequent sections of the Problem Formulation, for the operable exposure pathways identified, higher exposures are not anticipated for Aboriginal communities; for example, it has been assumed that a residential receptor will consume 100% of their produce from a backyard garden impacted by Project emissions. Given the urbanized nature of the Study Area, there are no resources that would result in increased exposures for Aboriginal communities for the operable exposure pathways identified; it is noted that the fish/seafood ingestion pathway was determined to be inoperable (see discussion in Section 3.4.4.) . On this basis, Aboriginal communities were not specifically addressed in the HHRA.

Statistics Canada (2014) was used to determine the age distribution for communities with the region, specifically including the Project location (Surrey), adjacent municipalities (Corporation of Delta and City of New Westminster), municipalities located along the rail corridor between the Canada-US border and FSD (City of Surrey, Corporation of Delta and City of White Rock), as well as municipalities located along the barge route from FSD to the Strait of Georgia (Richmond). This information was based on the 2011 census data collected by Statistics Canada. Tables 3-11 and 3-12 provide a breakdown of the age characteristics for the region. The age groups differ slightly from those used by Health Canada (2012); however, they provide an indication of the age groups represented in the region.

Table 3-11: Age Group Distribution for Municipalities in the Study Area

Age Group	Greater Vancouver	Surrey	Delta	New Westminster	Richmond	White Rock
Total population	2,313,325	468,255	99,865	65,975	190,475	19,340
0 to 4 years	115,185	29,160	4,755	3,260	8395	575
5 to 9 years	114,390	28,800	5,745	2,655	8855	545
10 to 14 years	124,880	30,785	6,555	2,700	10110	680
15 to 19 years	145,190	33,130	7,540	3,180	12535	785
20 to 24 years	159,080	31,085	5,935	4,295	13680	730
25 to 29 years	170,065	32,275	4,645	5,315	12740	825
30 to 34 years	160,010	32,155	4,620	5,215	11160	840
35 to 39 years	161,245	32,900	6,050	4,915	11720	960
40 to 44 years	180,535	35,030	7,310	5,570	14,510	1,095
45 to 49 years	192,090	36,535	8,335	5,810	16,635	1,420
50 to 54 years	182,435	34,340	8,645	5,560	16,490	1,585
55 to 59 years	158,570	29,820	7,420	4,725	14,910	1,760
60 to 64 years	136,755	25,665	6,850	3,940	12,720	1,850
65 to 69 years	94,860	18,530	5,235	2,660	8,010	1,455
70 to 74 years	72,890	13,585	3,740	1,875	6,195	1,165
75 to 79 years	58,150	10,180	2,645	1,560	5035	955
80 to 84 years	44,235	7,400	1,990	1,325	3595	885
85 years and over	42,760	6,880	1,855	1,425	3,180	1,235

Source: Statistics Canada (2014)

Table 3-12: Age Group as Percentage of Total

Age Group	Greater Vancouver	Surrey	Delta	New Westminster	Richmond	White Rock
Infants and Toddlers (0-4yrs)	5%	6%	5%	5%	4%	3%
Children (5-9 years)	5%	6%	6%	4%	5%	3%
Teens (10-19 years)	12%	14%	14%	9%	12%	8%
Adults (20 years and over)	78%	74%	75%	82%	79%	87%

Source: Statistics Canada (2014), rounded to nearest percent.

As indicated in Table 3-12, age group distribution within each municipality is generally consistent with that of the Greater Vancouver population, with the City of White Rock having a slightly higher proportion of adults. The presented data, which indicates that each of the age classes considered in the Health Canada guidance represent a portion of the population in each of the municipalities in the Study Area, supports the use of the Health Canada recommended receptor characteristics.

The Health Canada recommended receptor characteristics presented in Table 3-10 were used in the Exposure Assessment to estimate receptor exposure to Project emissions for the various age group classes under the various assessment scenarios.

3.4.2 Areas of Study Area with the Highest Predicted Project Emissions

The results for the Levelton (2014) AQA indicated that the overall maximum predicted concentrations of CACs were identified at the FSD fenceline; as indicated previously, with the exception of NO_2, concentrations of the CACs at the fenceline and throughout the Study Area were less than the AAQO. In addition, Levelton (2014) predicted maximum concentrations of the CACs for the residential neighborhood adjacent FSD (e.g. maximum North Delta residential receptor), as well as for a receptor located 5 m from the rail corridor. As discussed above, residential properties are located in proximity to the rail corridor in some communities within the Study Area. Although homes are not present within 5 m of the corridor (for practical and safety reasons), there is the potential for residents to be exposed to emissions near the rail corridor. Consequently, the HHRA has conservatively evaluated a 'maximum rail corridor residential receptor' using the maximum concentrations of the CACs predicted by Levelton (2014) within 5 m of the rail corridor.

Given that there are in maximum predicted concentrations between the maximum North Delta residential receptor and the maximum rail corridor residential receptor, the HHRA will evaluate both residential receptors. As the findings of Levelton (2014) indicated that residents within the community adjacent FSD (North Delta) and along a

portion of railway would be exposed to the highest CACs predicted for any residents within the Study Area, the characterization of exposures for these two receptors address maximum residential exposures and is therefore protective of all residential exposures within the Study Area.

3.4.3 Land Uses and Receptor Identification

Land use is an important determinant of who is likely to be present in the area, the types of exposures that may occur and the duration of exposure. As discussed in Section 3.1, the land use surrounding the Project is diverse and includes industrial and commercial operations, as well as residential neighbourhoods, which include schools, childcare centres, senior's facilities and parks. Health Canada (2012) guidance for conducting HHRAs uses data sourced from demographics of the general Canadian population to determine typical exposure frequencies based on land use; the Health Canada recommended exposure frequencies and durations for various land uses are presented in Table 3-13.

Table 3-13: Health Canada (2012) Exposure Frequencies and Durations Based on Land Use

Characteristic	Residential	Agricultural	Commercial	Industrial	Urban Park
Hours per day on site	24	24	8	10	--
Days per week on site	7	7	5	5	--
Weeks per year on site	52	52	52	48	--
Days per year food ingested from site	365	365	0	0	--
Total years exposed	80	80	35[1]	35[1]	--
Life Expectancy	80	80	80	80	--

Notes: -- Not provided, professional judgement to be applied (Health Canada, 2012)
[1] 35 years based on assumption that employees, rather than members of the general public (i.e., patrons), will be the most repeatedly exposed
Source: Health Canada (2012)

The following sections discuss each of the land uses identified in the Study area, giving consideration to the Health Canada (2012) exposure duration and frequency assumptions recommended for the various land uses. In addition, receptors of concern for evaluation in the HHRA are identified for each of the land uses.

3.4.3.1 Residential Land Use

Residential land use is typically considered to be the most sensitive when evaluating human health exposures. Young children (toddlers) are assumed to be present, and as discussed in Section 3.4.1, are typically the most sensitive receptors when evaluating exposures to non-carcinogens (Health Canada, 2010a). Furthermore, in

deterministic HHRAs, such as that being conducted for the Project, exposures to residential receptors are estimated assuming that residents are present 24 hours per day, 7 days per week, 52 weeks per year (Health Canada, 2012) to account for the potential for individuals (e.g., seniors) that may spend the majority of their time at home. Furthermore, there is the potential for backyard gardens on residential lands, and deterministic HHRAs typically assume that all produce (root and above ground) consumed by a resident is grown on their property. It is acknowledged that these assumptions are overly conservative in most cases; for example, a portion of the day or week may be spent away from home at school or work, and not all homes have gardens and where gardens are present, commercial food sources are typically used as a supplemental food source.

Residential receptors in areas with the highest Project emissions were considered to be the most sensitive receptors. The concentrations of CACs for the maximum residential receptors were predicted for: the maximum North Delta residential receptor; and, the maximum residential receptor along the rail corridor. With the exception of receptors at the FSD fenceline (i.e., an area zoned for industrial use) these receptors would be exposed to the highest potential emissions predicted within the Study Area. As all age groups have the potential to be present, both toddlers and adults were identified as critical receptors and retained for quantitative evaluation in the HHRA. As summarized above, it was assumed that backyard gardens are present, and that all produce consumed by a resident is grown on their property.

Based on the predicted Project emissions, as well as the Health Canada recommended receptor characteristics (e.g., produce consumption rates), exposure frequency and duration, the residential toddler and adult are considered to be the most highly exposed receptors within the Study Area, and were evaluated as critical receptors in the HHRA.

3.4.3.2 Urban Park Land Uses

There are several urban parks located within the Study area including in the residential neighbourhoods adjacent the Project facility.

While young children may be present at these locations, exposures are much lower than those for the residential receptors, due to lower frequency and duration of exposures, and the lack of significant food production at these locations; therefore, characterization of exposures and associated risks for the maximum residential receptors is protective of urban park users; however, toddlers and adults using parks/recreational facilities in the Study area were identified as receptors of concern and qualitatively assessed (see Section 6, Risk Characterization).

3.4.3.3 Agricultural Land Uses

Other than where used as residences (i.e., farm homes), young children are not expected to frequent agricultural lands on a routine basis. The primary concern with these lands is the consumption of food produced on the lands, with the potential for consumption to occur within or outside of the Study area. The majority of the Greater Vancouver agricultural lands are located outside of the Study area; however, belts of agricultural lands used for produce production are located near the rail corridors in North Delta and Surrey. It is noted that livestock operations are not present in these areas.

Levelton (2014) indicates that predicted emissions for these areas (represented on Figure 3-5 as Rail 5 and Rail 6) are lower than those predicted for the residential areas adjacent the Project facility; however, to address concerns of individuals that consume food grown within the Study Area, risks associated with the consumption of produce grown on the agricultural lands in the Study Area has been considered. As agricultural areas are not located in areas of the highest Project emissions, consumption of food was assessed using the emissions data for the maximum residential receptor. As noted above, the Health Canada (2012) recommended exposure frequency for days per year that food is ingested from a site is the same for both agricultural and residential lands (i.e., 365 days/year). Food consumption exposures and associated risks for the residential receptors would therefore be protective of consumption exposures and associated risks for agricultural lands. Both toddlers and adults were identified as critical receptors for the evaluation of food consumption exposures and risks.

3.4.3.4 Commercial Land Uses

Commercial receptors are located in the vicinity of the Project Facility and therefore, may be exposed to the Project emissions. These receptors are not expected to be present for more than 8 hours a day, 5 days per week. Contact with soil is anticipated to be limited in commercial settings and produce is not expected to be grown on commercial properties for consumption purposes. On this basis, characterization of potential exposures and associated risks for the maximum residential receptors is protective of commercial receptors. However, to address concerns from members of the general public, commercial receptors (toddlers and adults) in the Study area were identified as receptors of concern and qualitatively assessed (see Section 6, Risk Characterization).

3.4.3.5 Industrial Land Uses

The Project facility is located within an area of industrial land use. While worker health and safety at FSD is under the jurisdiction of WorkSafe BC[1], workers at nearby industrial operations have the potential to be exposed to the Project emissions. Industrial workers are anticipated to have lower rates of exposure than residents in the area, with reduced amounts of time spent in the area and limited soil ingestion and no produce production anticipated for industrial lands. Based on the industrial nature of the activities that occur on industrial lands, access to industrial sites is typically limited due to safety requirements. Non-adult age groups are not expected to be present at these locations for significant durations. Consequently, the HHRA retained an adult industrial receptor in the Study Area, and specifically in the industrial area adjacent FSD, for quantitative evaluation.

The above land uses and Health Canada recommendations for characterization of exposures to people using the various land uses are considered in subsequent sections of the HHRA.

3.4.3.6 Other Receptor Groups

In addition to the above identified receptors, people involved in fishing activities (Aboriginal and others) have been retained as receptors of concern based on their potential to spend time on the river in the area of the Project. As summarized in previous sections of the report, levels of NO_2 exceeding the AAQO have been predicted adjacent FSD, in areas over the Fraser River. In addition, people involved in fishing activities on the Fraser River have the potential to be exposed to emissions predicted along the barge route.

The locations where NO_2 concentrations greater than the AAQO were predicted are depicted in Figure 3-4; as presented, the exceedances are localized over the Fraser River, and are confined to an approximate 400 m^2 area adjacent the FSD fenceline/berth. Fishing boats would not be expected to spend significant time in the area for the following reasons: 1) the area is located directly in the channel entrance to the FSD lower berths and therefore, there are safety concerns associated with boats maintaining a consistent position in the area; and 2). the area extends to the middle of the river channel between Surrey and Annacis Island and the strong currents in the area would prevent a fishing vessel from maintaining a consistent position in the area. On this basis, people involved in fishing activities would only be exposed to Project emissions on an acute basis.

Based on the above, evaluation of the industrial receptor is protective of people involved in fishing activities. Both acute and chronic exposures and associated risks have been characterized in subsequent sections of the report for the industrial receptor.

[1] *WorkSafeBC Occupational Health and Safety Regulation (OHSR),* BC Reg. 296/97, Amended by B.C. Reg. 230/2011, effective April 15, 2012.

3.4.3.7 *Summary of Identified Receptors of Concern*

As described above, the following receptors were retained for quantitative evaluation in the HHRA. Critical receptors identified for each receptor group are listed in parentheses.

- Residents (toddlers and adults); and
- Industrial workers (adults only).

Furthermore, as indicated, the evaluation of the residential receptor is considered protective of the agricultural receptor, urban park receptors and commercial receptors, and the evaluation of the industrial receptor is protective of people involved in fishing activities. These receptors will be qualitatively assessed in Section 6, Risk Characterization.

The pathways by which these receptor groups have the potential to be exposed to the Project COPCs is discussed below.

3.4.4 Identification of Exposure Pathways

Exposure to Project emissions may occur through contact with air, soil, dust and produce within the Study Area. The pathway of greatest public health concern in the region is the direct inhalation of Project emissions, and thus, the direct inhalation pathway is the focus of the HHRA. Based on predicted Project emissions, residents, commercial workers, industrial workers and any others spending time in the Study Area could be exposed to the Project COPCs via inhalation.

The above identified receptors of concern also have the potential to be exposed to the Project COPCs via secondary exposure pathways. Fate and transport processes may lead to the atmospheric deposition of non-gaseous COPCs onto near surface soil and vegetation, as well as subsequent plant uptake of COPCs from soil. Therefore, receptors in the Study Area also have the potential to be exposed to the COPCs via the direct soil pathways (i.e., incidental soil ingestion, dermal contact with soil and inhalation of dust generated from soil), as well as through the consumption of produce from areas where atmospheric deposition of the COPCs has occurred. In addition, hexachlorobenzene, a gaseous COPC, was identified to be bioaccumulative and, therefore, the potential for plants to uptake hexachlorobenzene vapour has also been evaluated.

Consideration was also given for the potential for Project COPCs to impact surface water and groundwater in the Study Area, with the subsequent potential for human exposure.

The Project is located within the Lower Fraser Watershed extending from Hope, BC, to the mouth of the river which is approximately 34 kilometres (km) from FSD. Watercourses in the vicinity of the Project are minor tributary streams

draining into the Fraser River (including Gunderson Slough). The streams at their lower reach have been highly modified from their natural condition in terms of drainage patterns and water quality due to the degree of urbanization in the immediate area. The surface flows for these watercourses into the Fraser River are mainly through drainage channels and culverts. Road run-off is a contributor to surface flow at the lower reaches.

Coal dust and other project emissions have the potential to enter surface water bodies in the Study Area as dustfall and could also enter the Fraser River during equipment or system failures and through vessel accidents resulting in spills (Triton, 2013). Surface water bodies in the immediate area of FSD, where project emissions are predicted to be the highest, are not used for recreational purposes (e.g., swimming), and the Fraser River in the industrial area of the Project is not used for seafood harvesting. The area from FSD to the mouth of the Fraser River is closed for shellfish harvesting based on the Fisheries and Oceans Canada year round sanitary closure in effect in the area (Closure 29.3; information available at http://www.pac.dfo-mpo.gc.ca/fm-gp/contamination/sani/area-secteur-29/29.3-eng.html). Furthermore, the ecological health effects assessment included in the Project EIA (SNC-Lavalin, 2013) concluded that no significant residual effects from coal dust, coal spill or accidental release of unprocessed coal wastewater into the aquatic environment are expected. In addition, the metals and PAHs are tightly bound to the coal matrix and therefore are not bioavailable under normal environmental conditions (Ahrens and Morrisey, 2005; Triton, 2013a). On this basis, no further evaluation of surface water will be conducted in the HHRA.

FSD is aligned over the Fraser River Junction Aquifer which is estimated at 9 square kilometres (km^2). The aquifer is shallow and unconsolidated, comprising of sand and gravel deposits. The Fraser River Junction Aquifer is not a local source of drinking water. The Newton Upland Aquifer is an upland sand and gravel aquifer underlying the City of Surrey. The aquifer is directly adjacent to the Project on the east side of River Road/South Fraser Way and is lightly developed (low demand relative to productivity), with low vulnerability to contamination.

As discussed, Project emissions, including coal dust (as $PM_{2.5}$ and PM_{10}) will undergo atmospheric deposition and be deposited on near-surface soils in the Study Area. Subsequent leaching of the metals and PAHs in the coal to groundwater is not anticipated under typical environmental conditions, as the metals and PAHs are tightly bound to the coal matrix. Furthermore, the Contaminated Sites Regulation (CSR) Schedule 5 Matrix Numerical Soil Standards include soil standards derived to be protective of soil leaching to groundwater used for drinking water and for groundwater flow to surface water used by aquatic life. Such standards are available for arsenic, barium, cadmium, chromium, copper, lead and zinc. The mean concentrations of these metals measured in the coal samples submitted for analysis, as presented in Table 3-4, are well below these standards and, in many cases, below the background metals concentrations measured in surface soil in the Study Area (i.e., deposition of these COPCs would not result in increased soil concentrations).

In addition to the above, the US EPA (2005) indicates that the ingestion of groundwater as drinking water pathway does not require evaluation when evaluating combustion emissions, as studies have indicated that it is very unlikely to be a significant exposure pathway.

Based on the above, Project emissions are not anticipated to impact groundwater in the region, and thus, no further evaluation will be conducted.

The operable exposure pathways evaluated in the HHRA for the above identified receptors of concern are summarized below:

- Inhalation of air (outdoor and indoor), including suspended dust;
- Incidental soil ingestion;
- Dermal contact with soil;
- Inhalation of soil particulate;
- Inhalation of indoor dust;
- Ingestion of above-ground plants (including fruit and vegetables); and
- Ingestion of below ground plants (root vegetables).

The above listed pathways have the potential to contribute to overall exposure to Project COPCs.

The potential exposure scenarios for the above identified receptors of concern are further discussed below.

3.4.4.1 Direct Inhalation of Project Emissions

The HHRA evaluated the direct inhalation of Project emissions in air. Exposures were determined for the maximum estimated Project emissions for each of the receptors evaluated, including the identified critical receptors for the residential and industrial areas within the Study Area (i.e., the HHRA will evaluate the most highly exposed individuals within the Study Area). Therefore, the evaluation of exposures to these receptors is considered protective of the lesser exposed individuals within the Study Area (e.g. agricultural, commercial and urban park receptors and people involved in fishing activities).

3.4.4.2 Inadvertent Ingestion of Soil

Select COPCs (i.e., the non-gaseous COPCs) associated with the Project emissions may undergo atmospheric deposition onto near surface soil; receptors in the Study Area therefore have the potential to be exposed to the Project COPCs deposited on surface soil via the direct soil pathways. Direct ingestion of soil is anticipated to be limited in most cases, as typical hygiene practices would include hand washing after handling soil; however, there remains the potential for hand to mouth contact soil exposures (i.e., ingestion of soil that has adhered to hands). Therefore, the HHRA evaluated incidental ingestion of soil impacted by Project emissions.

3.4.4.3 Direct Dermal Contact with Soil

In addition to the incidental ingestion of impacted soil from hand to mouth contact, there remains a potential for absorption of COPCs from soil though the skin in the event of soil contact. This pathway is of particular importance for the residential receptor and for urban park users, where soil to skin contact may occur during periods of play or gardening/landscaping. Dermal absorption of chemicals is dependent on a number of factors including the surface area and thickness of the exposed skin, the length of exposure and the physical properties of the COPC. Contributions of dermal exposures to overall soil exposure risks are typically low (US EPA, 2005). For example, dermal absorption exposure rates for PAHs are typically approximately 15% of those for oral exposures. However, in an effort to not underestimate potential risks associated with the Project, soil dermal contact was evaluated in the HHRA.

3.4.4.4 Inhalation of Re-Suspended Dust

Following the atmospheric deposition of Project COPCs to near-surface soils, there is the potential for dust to be generated from the soil, and therefore for the COPCs to be re-suspended and inhaled as soil particulate/dust. The inhalation of airborne respirable dust levels (particle matter of ≤ 10 µm in diameter) is typically an insignificant pathway relative to the soil ingestion and dermal contact pathways (Health Canada 2012; US EPA 2005); however, the HHRA will consider soil particulate inhalation. Based on the land uses and presence of paved roads within the Project vicinity, re-suspended dust concentrations for the land uses within the Study Area were assumed to be 0.76 µg/m^3 (as recommended by Health Canada 2012).

3.4.4.5 Inhalation of Indoor Dust

There is the potential for Project emissions, as well as for soil particulate generated from near-surface soils (where atmospheric deposition of Project COPCs has occurred) to migrate indoors and be deposited on hard surfaces. The HHRA assessed the inhalation of indoor dust for receptors in indoor spaces.

3.4.4.6 Ingestion of Produce

The Project Facility and portions of the rail corridor that will be used by trains transporting coal to FSD are located adjacent residential communities, where there is the potential for backyard gardens. In addition, agricultural lands are present along portions of the rail corridor evaluated as part of the Study Area. As indicated, there is the potential for atmospheric deposition of Project COPCs directly onto produce/vegetation in the Study area, as well as the potential for plants to uptake the Project COPCs from soil. On this basis, the HHRA will evaluate exposures to the residential receptor via the consumption of produce grown in the Study Area. As the highest potential emissions for lands that may be used to grow food (e.g., commercial and industrial lands in the Study Area are not anticipated to be used for food production) were for the maximum North Delta residential receptor and the maximum rail corridor residential receptor, the HHRA will evaluate the ingestion of home grown produce in these residential areas. As discussed, there are localized agricultural areas located along the rail corridor in the City of Surrey and Delta; however, the HHRA focused on food production in areas with the highest predicted Project emissions in an effort to be protective of both the general public and sensitive receptors. The evaluation of the residential receptor is considered protective of produce consumers throughout the Study Area.

Exposures via the consumption of both above-ground and below ground (i.e., root vegetables) produce were evaluated. Above-ground produce has the potential to be exposed to COPCs both through the deposition of Project COPCs onto above-ground, edible plant parts, as well as by root uptake. It is noted that some above-ground produce have a protective cover (e.g., peas, corn) and the outer layer of bulky produce (e.g., spinach, broccoli, celery and lettuce) is removed during food preparation (US EPA, 2005), therefore, the consumption of the edible portion of some above-ground produce is not subject to aerial deposition COPCs. Root uptake of COPCs was considered operable for both above- and below ground produce.

3.5 Conceptual Site Model

The final stage of the Problem Formulation is the development of a Conceptual Site Model (CSM); a CSM presents a summary of the Problem Formulation for the HHRA, including identified receptors of concern, the age group(s) to be evaluated in the HHRA, and the identified operable exposure pathways. A CSM has been developed for the Project emissions, and is presented below in tabular form, with a pictorial representation including COPC fate and transport processes presented in Figure 3-6.

Table 3-14: Conceptual Site Model, Human Health

Land Use	Receptor of Concern	Age Groups Considered	Operable Exposure Pathways
Residential	Residential Receptor	Infant Toddler Child Teen Adult	**Inhalation of Project emissions in air** **Incidental Ingestion of Soil** **Dermal Contact with Soil** **Inhalation of Soil Particulate** **Inhalation of indoor dust** **Ingestion of above-ground produce**** **Ingestion of below ground produce****
Urban Park	Urban Park User	Infant Toddler Child Teen Adult	*Evaluation of the Residential Receptor is Protective of the Urban Park Receptor*
Industrial	Industrial Workers	Adult	**Inhalation of Project emissions in air** **Incidental Ingestion of Soil** **Dermal Contact with Soil** **Inhalation of Soil Particulate** **Inhalation of indoor dust**
Commercial	Commercial Workers and Patrons	Infant Toddler Child Teen Adult	*Evaluation of the Residential Receptor is Protective of the Commercial Receptor*
Fraser River	People Involved in Fishing Activities	*Adult*	*Evaluation of the Industrial Receptor is Protective of People Involved in Fishing Activities*

BOLD – Retained for Quantitative Evaluation in the HHRA.
Underline – Critical Receptor
** - ingestion of above- and below ground produce will be evaluated for a residential receptor in the area of the highest Project emissions; the evaluation of exposures and associated risks for the residential receptor is considered protective of other produce consumers in the Study Area as it has been assumed that 100% of the fruit and vegetables consumed by the residential receptor has been grown in a backyard garden, and because predicted Project emissions in other areas of potential produce production are lower.

Figure 3-6: Conceptual Site Model

The identified COPCs, receptors of concern, and the operable pathways by which the receptors have the potential to be exposed to the COPCs, as identified in Table 3-14 and in Figure 3-6 are evaluated in the subsequent sections of the HHRA.

4 EXPOSURE ASSESSMENT

The Exposure Assessment is conducted to identify the concentrations of the COPCs in each exposure medium (i.e., the exposure point concentrations [EPCs]), as well to estimate the dose of each COPC that the receptors of concern have the potential to be exposed to. The EPCs for the various exposure media, the Health Canada (2012) exposure equations and the estimated doses for each receptor of concern are discussed in the following sections.

4.1. *Exposure Point Concentrations*

Exposure point concentrations for air, soil and vegetation for each of the Baseline, Project and Cumulative Scenarios are discussed in the following sections.

4.1.1 Air

4.1.1.1 Baseline Scenario Air EPCs

For the Baseline Scenario, baseline ambient air concentrations of the CACs were determined using data from three Metro Vancouver ambient air quality monitoring stations (T13 North Delta, T18 Burnaby South and T6 Second Narrows); the baseline ambient air concentrations of the CACs presented in Section 3.2.1.1, Table 3-1 were used as air EPCs for the Baseline Scenario.

In addition, the Burnaby South National Air Pollution Survey Program (NAPS) air monitoring data from 2008-2012 were reviewed to determine the baseline air concentrations for the various metal/metalloid, PAH and VOC COPCs. The baseline concentrations for these COPCs are presented in Appendix II, Table II-1A.

No baseline air data were available for select COPCs including all PAHs, with the exception of naphthalene, the dust palliatives chemical constituents, select VOC and metal COPCs, as well as PCBs. Based on the lack of baseline concentrations for these parameters, a literature review was conducted to determine typical background concentrations of these COPCs in Canadian urban areas. Table 4-1 presents a summary of the results of the literature review.

Table 4-1: Typical Ambient Air Concentrations of Select COPCs in Urban Areas

COPC	Typical Ambient Air Concentrations ($\mu g/m^3$)	Reference
PAHs	Acenaphthene 0.0022 Fluorene: 0.0051 Phananthrene: 0.016 Anthracene: 0.0016 Pyrene: 0.0049 Fluoranthene: 0.0068 Benzo(a)anthracene: 0.0008 Benzo(a)pyrene: 0.0004 Benzo(b)fluoranthene: 0.0007 Benzo(k)fluoranthene: 0.0003 Indeno(1,2,3-cd)pyrene: 0.0019 Total PAHs: 0.021 (Ambient air concentrations for Canadian cities; no data were available for Vancouver for the non-carcinogenic COPCs, and therefore data for Toronto were used)	Health Canada, 1994 (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/hydrocarb_aromat_polycycl/index-eng.php#t3)
Boron	No data	
Mercury	0.0037 (Total mercury concentrations in ambient air reported to range from 0.0012 to 0.0037 $\mu g/m^3$)	European Commission, Position Paper on Mercury (http://ec.europa.eu/environment/air/pdf/pp_mercury4.pdf)
Acetaldehyde	3.35 (max mean for individual sites characterized by the collection of 2805 24-hour samples from rural, suburban and urban locations at 14 sites in six provinces surveyed from August 1989 to June 1997)	Health Canada, 2000 (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl2-lsp2/acetaldehyde/index-eng.php#a2.3.2.1)
Acrolein	0.2 (typical ambient concentration in Canadian cities)	WHO, 2002 (http://www.who.int/ipcs/publications/cicad/en/cicad43.pdf)
Formaldehyde	8.76 (max long-term (1 month to 1 year) mean concentration for 14 sites, including urban sites with Vancouver BC listed as one of the sites, across Canada; mean concentrations at the 14 sites ranged from 0.78 to 8.76 $\mu g/m^3$)	Health Canada, 2001 (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl2-lsp2/formaldehyde/index-eng.php#a23211)
Epichlorohydrin	No data	See below discussion

Typical ambient air concentrations in urban areas, as summarized in Table 4-1, have been used in the HHRA as baseline air concentrations for these COPCs. Data for Canadian cities were available for PAHs, acetaldehyde, acrolein and formaldehyde. Although Canadian data were not available for mercury, the maximum of the range of typical ambient air concentrations reported by the European Commission (Position Paper on Mercury, available at

http://ec.europa.eu/environment/air/pdf/pp_mercury4.pdf) was used. As noted in Table 4-1, where reported as a range, the maximum concentrations were conservatively selected in an effort to ensure that baseline exposures are not underestimated.

Although SNC-Lavalin acknowledges that the use of literature values for the above COPCs is not ideal, it was determined that this approach was preferred over not addressing the Baseline Scenario for these COPCs. For some parameters (e.g., PAHs) the data are older; however, given the technological advances in reducing combustion emissions, the use of this data is considered conservative. Given the conservative approach used in the selection of the literature based concentrations, it is likely that the data are overestimates of current ambient concentrations in residential locations near the FSD facility.

No background air concentrations were available for boron and epichlorohydrin. Epichlorohydrin was retained as a COPC as it is a chemical constituent in the dust palliatives. As discussed throughout the report, the approach used to estimate exposures and associated risks to epichlorohydrin is highly conservative. Additionally, no other sources of epichchlorohydrin are anticipated in the Study Area, as it is typically associated with chemical manufacturing, and is formed during from the manufacture of various chemicals (e.g. glycerin), plastics and rubbers.

The lack of baseline data for select COPCs is further discussed in Section 7, the Uncertainty and Sensitivity Analysis.

4.1.1.2 Project Scenario Air EPCs

The results of the Levelton (2014) AQA were used to determine the concentrations of the COPCs in air for the Project Scenario. For the CACs, DPM and TPM, the maximum predicted concentrations associated with the Project at the FSD fenceline (i.e., maximum exposed receptor) were used as air EPCs in the estimation of exposures for industrial workers and people involved in fishing activities on the Fraser River. The maximum predicted concentrations for the maximum residential receptors (maximum North Delta residential receptor and maximum rail corridor residential receptor) were used as air EPCs for the residential receptors.

To determine the air concentrations of each of the metals/metalloids and PAHs (i.e., the constituents) in coal dust, the maximum predicted TPM concentrations at the FSD fenceline and at the maximum residential receptors were used, and adjusted by the average percent composition of each of these constituents found to be in the coal (based on the results of the coal analyses presented in Table 3-4).

In addition, for transportation equipment combustion emissions, the concentrations of the various species (metals/metalloids, PAHs, VOCs, sulfate and PCBs) were identified and presented by Levelton (2014). Levelton (2014) estimated the concentrations of these species at the maximum receptor (FSD fenceline) and at the maximum residential receptors by multiplying the either the maximum predicted PM_{10}, $PM_{2.5}$, or total VOC concentration by the speciation profile for each individual species. These adjusted concentrations were then summed up at each receptor to determine the maximum predicted concentration for each individual specie. These concentrations were used as EPCs for the various species in the Project Scenario.

Of the dust palliative constituents, epichlorohydrin was determined to have the highest relative toxicity (see discussion in Section 5, Toxicity Assessment) and therefore was conservatively selected as a surrogate for the characterization of risks associated with exposures to the dust palliatives. Epichlorohydrin is a constituent of the dust suppressant GE 9148; the MSDS indicates that 30-60% of the product is an adipic acid, diethylenetriamine, epichlorohydrin polymer. To estimate the air concentration of epichlorohydrin for the Project Scenario, the manufacturer's (i.e., GE's) recommended application rate was used along with the predicted coal dust concentration. It was conservatively assumed that 100% of the product is epichlorohydrin (vs. the 30-60% adipic acid, diethylenetriamine, epichlorohydrin polymer). A worked example for the estimation of the epichlorohydrin air concentration is presented in Appendix III.

For COPCs from multiple sources (e.g. PAHs and metals/metalloids from coal and from combustion emissions), the EPCs were determined by summing the predicted concentrations from the various sources.

Table II-1B (in Appendix II) presents the air EPCs for the Project Scenario.

4.1.1.3 Cumulative Scenario Air EPCs

The air EPCs for the Cumulative Scenario were calculated by adding the air EPCs for the Baseline Scenario (i.e., background air concentrations) to the air EPCs for the Project Scenario (i.e., emissions from the Project). The air EPCs for the Cumulative Scenario are provided in Appendix II, Table II-1C.

4.1.2 Soil

4.1.2.1 Baseline Scenario Soil EPCs

The soil EPCs for the Baseline Scenario were determined using the results of the background soil assessment described in Section 3.2.2.1. The assessment included the collection of surface soil samples from areas where sensitive receptors have the potential to be present near the FSD facility, as well as from municipalities along the rail

corridor and barge route. Samples were collected from four municipalities in the Study Area, including the cities of Delta, Richmond, Surrey, and White Rock. Each of the surface soil samples collected from these areas were submitted for analysis of metals/metalloids, chromium speciation and PAHs, with the mean concentrations of these parameters in background surface soils presented in Section 3.2.2.1, Table 3-6 and in Table II-1A (Appendix II); the mean concentrations were used as the soil EPCs for the Baseline Scenario.

The background deposition provided in Levelton (2014) was not used to determine baseline soil concentrations, as the measured soil concentrations include background contributions from deposition.

It is noted that background soil concentrations of select non-gaseous COPCs, including PCBs and the dust palliative chemical constituents, were not available. PCBs do not occur naturally in the environment, and no appreciable anthropogenic sources are anticipated in the Study Area. On this basis, background soil concentrations of PCBs are not expected to contribute significantly to exposures. Similarly, the potential for other sources of the chemical constituents of the dust palliatives in the Study Area is considered low to negligible and, therefore, it is unlikely that the dust palliative COPCs are present in background soils at appreciable concentrations, and if present, are unlikely to contribute significantly to exposures. This is further discussed in Section 7, the Uncertainty and Sensitivity Analysis.

4.1.2.2 Project Scenario Soil EPCs

As previously described, the non-gaseous COPCs have the potential to undergo atmospheric deposition and, therefore, be deposited on near surface soils in the Study Area. The soil EPCs for the Project Scenario were estimated using the predicted air concentrations of the various non-gaseous COPCs associated with the particulate matter emissions from the Project. This included:

- Metals/metalloids and PAHs in coal dust;
- The various COPCs (including metals/metalloids, PAHs, PCBs and sulfate) identified to be present in the combustion emissions from transportation equipment from the Project; and
- The non-gaseous constituents of the dust palliatives.

The soil concentrations of the non-gaseous COPCs were predicted primarily using models developed by the US EPA (US EPA OSW, 2005), along with deposition rates estimated by Levelton (2014) as part of their overall air quality assessment. The highest rates for coal and combustion deposition and dustfall from all rail segments modelled by Levelton (i.e., Rail 1 to 7 in Figure 3-5) were used to model soil concentrations for the maximum rail residential receptor. This is highly conservative as the locations of the maximum rates varied for the various

COPCs, and the assumption that they occur at a single location assumes that a single receptor is being exposed to maximum dustfall/deposition for each constituent along entire rail corridor.

The chemical-specific dustfall rates for the coal constituents (i.e., metals/metalloids, PAHs) were determined using the maxmum dustfall rates for coal dust reported by Levelton (2014), along with the mean concentrations of the coal, which were used to determine the percent composition of COPCs in coal samples. For each COPC, the coal dustfall rate was multiplied by the percent composition of the COPC in coal as per the following equation:

COPC specific dustfall rate = dustfall rate for coal dust × % composition of COPC in coal

Where applicable, the chemical-specific dustfall rate for the COPC in the coal was then added to the chemical-specific dustfall rate for that COPC from the combustion emissions (provided to SNC-Lavalin by Levelton) to determine an overall chemical-specific deposition rate for each COPC.

The deposition rates were used to predict chemical-specific deposition onto the top 2 cm of soil. The use of this shallow mixing depth is conservative as mixing is expected to range from 2 cm to 20 cm (US EPA OWS, 2005). Soil concentrations were predicted by taking into account chemical deposition onto soil and loss due to chemical degradation (biotic and abiotic) and volatilization. Parameters used in the prediction of chemical degradation for the COPCs were obtained from ORNL (2014). Although soil run-off would also contribute to chemical loss from soil, it is noted that soil concentrations were conservatively predicted assuming no run-off.

The resulting near surface soil concentrations were used as soil EPCs for the Project Scenario and were used to estimate soil exposures via the direct pathways (i.e., incidental ingestion of soil and dermal contact with soil). The modelled near surface soil concentrations were also used to estimate concentrations in soil dust, which were used as EPCs for the inhalation of soil particulate pathway.

As indicated in Section 3.2.2.1, the mean background soil concentrations of the majority of the metals (antimony, arsenic, cadmium, chromium (+3), chromium (+6), cobalt, copper, lead, lithium, manganese, molybdenum, nickel, silver, tin, uranium, vanadium, zinc, aluminum, iron, thallium and titanium) and select PAHs (benzo(a)pyrene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene and phenanthrene) were higher than the mean concentrations of these parameters measured in the coal; this is of significance as the deposition of coal dust on to surface soils will not result in increased soil concentrations for these parameters. This is further discussed in Sections 6 and 7 of the HHRA.

The soil EPCs for the Project Scenario are summarized in Appendix II, Table II-1B.

4.1.2.3 Cumulative Scenario Soil EPCs

The soil EPCs for the Cumulative Scenario were calculated by adding the soil EPCs for the Baseline Scenario (i.e., background soil concentrations) to the soil EPCs for the Project Scenario.

As described above, the concentrations of the majority of the metals and select PAHs were higher in the background surface soils than in the coal; for the metals with higher background soil concentrations, the deposition of coal containing these metals onto surface soils in the Study Area will result in decreased exposures/associated risks. On this basis, these metals were not assessed in the Cumulative Scenario. As discussed above for the Project Scenario soil EPCs, the PAHs with the higher concentrations in the background surface soils compared to the coal are further discussed in Section 6, the Risk Characterization.

The soil EPCs for the Cumulative Scenario are summarized in Appendix II, Table II-1C.

4.1.3 Vegetation

4.1.3.1 Baseline Scenario Vegetation EPCs

The vegetation EPCs for the Baseline Scenario were estimate through application of chemical uptake equations to the measured baseline (i.e., background) soil data. The HHRA evaluated uptake to both above (e.g., leafy vegetables) and below ground (e.g. root vegetables) vegetation. The main difference in the estimation of COPC concentrations for the two different vegetation types is that for above ground vegetation the modeling considers both direct deposition of the COPCs from the atmosphere to the surface of the vegetation, as well as root uptake from soil. The modeling of below ground vegetation concentrations only considers root uptake from soil.

In addition to the above, vegetation vapour uptake was estimated for hexachlorobenzene, a gaseous COPC that was considered to be bioaccumulative, and was therefore retained in the multi-media assessment.

Vegetation concentrations were estimated using models and parameters recommended by the US EPA (US EPA OWS, 2005). It is noted that it was assumed that vegetation would not be washed or peeled prior to consumption. This assumption is considered conservative; the US EPA OWS (2005) states that the majority of produce is washed or peeled prior to consumption, with the outer layer of above ground produce (e.g., broccoli, corn) removed and discarded prior to consumption.

The vegetation EPCs for the Baseline Scenario are summarized in Appendix II, Table II-1A.

4.1.3.2 Project Scenario Vegetation EPCs

The vegetation EPCs for the Project Scenario were estimated using the approach described above for the Baseline Scenario; however, the concentrations were modelled based on the soil concentrations predicted for the Project Scenario (as described above in Section 4.1.2.2). As described above, models and parameters recommended by the US EPA (US EPA OWS, 2005) were used along with the predicted soil concentrations to estimate both above and below ground vegetation concentrations. Root uptake was conservatively estimated using concentrations predicted for the top 2 cm of soil. This assumption is conservative as the majority of the plant root mass would be exposed to deeper soils with lower soil COPC concentrations.

For the metals and PAHs with higher concentrations in the background surface soils than in the coal, the deposition of coal dust containing these COPCs onto surface soils in the Study Area will not increase the soil concentrations and, therefore, assuming equivalent bioavailability, there will be less uptake of the COPCs by vegetation.

On this basis, similar to soil, although exposures via consumption of vegetation impacted by the potential Project emissions was estimated (i.e., for the Project Scenario), it is noted that the exposures for the metals and, therefore, the associated risks, were lower than those estimated for the Baseline Scenario. The PAHs are further evaluated in Section 6, the Risk Characterization.

The vegetation EPCs for the Project Scenario are summarized in Appendix II, Table II-1B.

4.1.3.3 Cumulative Scenario Vegetation EPCs

The vegetation EPCs for the Cumulative Scenario were calculated by adding the vegetation EPCs for the Baseline Scenario (i.e., estimated from background soil concentrations) to the vegetation EPCs for the Project Scenario (i.e., estimated from predicted soil concentrations).

For the metals with higher background soil concentrations (compared to the coal), the deposition of coal containing these metals onto surface soils in the Study Area will result in less uptake of the metals by vegetation. On this basis, these metals were not assessed in the Cumulative Scenario.

The vegetation EPCs for the Cumulative Scenario are summarized in Appendix II, Table II-1C.

4.2 Estimation of Exposures for Potential Receptors of Concern

Exposures for the receptors of concern (critical receptors) were estimated for the various operable exposure pathways identified in Table 3-14 and in Figure 3-6 using Health Canada (2010a, 2012) recommended receptor characteristics, exposure frequencies and durations and exposure equations.

Health Canada recommended receptor characteristics for the various age groups, as summarized in Section 3.4.1., Table 3-10, were used in the estimation of exposures. In addition, Health Canada recommended exposure frequencies and durations summarized in Section 3.4.3, Table 3-13, were used. As noted in Table 3-13, Health Canada does not provide a recommended exposure frequency or duration for an urban park receptor. Based on professional judgement an urban park receptor (toddler and adult) was conservatively assumed to frequent a park for 2 hours a day, 7 days a week, 52 weeks a year for a period of 35 years.

The Health Canada exposure equations are presented in section 4.2.1.

4.2.1 Exposure Equations

The Health Canada (2010a, 2012) recommended exposure equations have been used to quantify exposures via the identified operable exposure pathways. Details on the exposure equations for each exposure pathway considered in the HHRA are provided in below.

4.2.1.1 Incidental Ingestion of Soil

It has been assumed that the industrial, commercial, residential and urban park receptors may unintentionally ingest soil impacted by the Project COPCs. In order to estimate exposure from soil ingestion, the following Health Canada (2012) equation was applied:

$$EIG = \frac{C_s \times IR_s \times RAF_{Oral} \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EIG	=	exposure from the soil ingestion pathway (µg/kg bw/d)
C_S	=	soil chemical concentration (µg/g)
IR_S	=	soil ingestion rate of person (g/day)
RAF_{Oral}	=	relative absorption factor from gastrointestinal tract (unitless, chemical-specific)

D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
BW	=	body weight of person (kg)
LE	=	life expectancy (years) (only used for carcinogens)

4.2.1.2 Dermal Contact with Soil

Dermal contact with soil was assumed to be an operable exposure pathway for the industrial, commercial, residential and urban park receptor. Dermal exposure was estimated according to the following Health Canada (2012) equation:

$$EDS = \frac{[(C_s \times SA_H \times SL_H) + (C_s \times SA_o \times SL_o)] \times RAF_{Derm} \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EDS	=	exposure from the dermal pathway for soils (µg/kg bw/d)
C_S	=	soil chemical concentration (µg/g)
SA_H	=	surface area of hands exposed for soil loading (cm^2)
SA_O	=	surface area exposed other than hands (cm^2)
SL_H	=	soil loading rate to exposed skin of hands (g/cm^2/event)
SL_O	=	soil loading rate to exposed skin other than hands (g/cm^2/event)
RAF_{Derm}	=	relative dermal absorption factor (unitless, chemical-specific)
D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
BW	=	body weight of person (kg)
LE	=	life expectancy (years) (only used for carcinogens)

4.2.1.3 Inhalation of Suspended Soil (Dust)

The receptors have the potential to inhale soil particles originating from soils impacted by Project emissions. For the purpose of the HHRA, 100% of the inhalable soil particulates were conservatively assumed to originate from the portion of the Study Area containing the maximum predicted soil concentrations of each non-gaseous COPC. Indoor dust concentrations were assumed to be equivalent to outdoor dust, and it was assumed that the receptors of concern would be exposed to outdoor dust (from soil impacted by the Project emissions) for their entire exposure duration (e.g. for the residential receptor, 24 hours a day was assumed). Soil particulate concentrations were estimated by multiplying soil concentrations by a particulate emission factor of 0.76 µg/m^3 (as recommended by Health Canada 2012). Dust inhalation exposure was estimated as per the following equation:

$$EID = \frac{C_A \times RAF_{inh} \times D_1 \times D_2 \times D_3 \times D_4}{LE}$$

Where:

EID	=	exposure from the dust inhalation pathway for soil (µg/m^3; as an "amortized dust concentration")
C_A	=	airborne chemical concentration (µg/m^3)
RAF_{Inh}	=	relative absorption factor by inhalation (unitless, chemical-specific)
D_1	=	hours per day exposed/24 hours (unitless)
D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
LE	=	life expectancy (years) (only used for carcinogens)

Where inhalation TRVs (e.g. Tolerable Concentrations [TC]/Reference concentrations [RfC]) were not available for a COPC, the following equation (Health Canada, 2012) was used:

$$EID = \frac{C_A \times IR_A \times RAF_{Inh} \times D_1 \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EID	=	exposure from the dust inhalation pathway for soil (µg/kg bw/d)

C_A	=	airborne chemical concentration (µg/g)
IR_A	=	receptor air intake (inhalation) rate (m^3/day)
RAF_{Inh}	=	relative absorption factor by inhalation (unitless, chemical-specific)
D_1	=	hours per day exposed/24 hours (unitless)
D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
BW	=	body weight (kg)
LE	=	life expectancy (years) (only used for carcinogens)

4.2.1.4 *Inhalation of Air*

Exposures via the inhalation of Project emissions were estimated using the following Health Canada (2010a) equation:

$$EIA = \frac{C_A \times D_1 \times D_2 \times D_3 \times D_4}{LE}$$

Where:

EIA	=	exposure from the inhalation (µg/m^3; as an “amortized air concentration”)
C_A	=	air concentration (µg/m^3)
D_1	=	hours per day exposed/24 hours (unitless)
D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
LE	=	life expectancy (years) (only used for carcinogens)

Where inhalation TRVs (e.g. Tolerable Concentrations [TC]/Reference concentrations [RfC]) were not available for a COPC, the following equation (Health Canada, 2012) was used:

$$EIA = \frac{C_A \times IR_A \times RAF_{Inh} \times D_1 \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EIA	=	exposure from the dust inhalation pathway for soil (µg/kg bw/d)
C_A	=	air concentration (µg/m^3)
IR_A	=	receptor air intake (inhalation) rate (m^3/day)
RAF_{Inh}	=	relative absorption factor by inhalation (unitless, chemical-specific)
D_1	=	hours per day exposed/24 hours (unitless)
D_2	=	days per week exposed/7 days (unitless)
D_3	=	weeks per year exposed/52 weeks (unitless)
D_4	=	total years exposed to site (only used for carcinogens)
BW	=	body weight (kg)
LE	=	life expectancy (years) (only used for carcinogens)

4.2.1.5 Ingestion of Vegetation

Residential receptors were conservatively assumed to consume 100% of the produce from backyard gardens impacted by Project emissions; this highly conservative assumption will overpredict exposures for the general population, who supplement their garden produce supply with store-bought produce. Exposures via consumption of vegetation (above and belowground) were estimated using the following equation:

$$EIF = \frac{C_{food} \times SS \times BAIG \times NSSW \times NWSY \times AAF \times LAF}{BW \times NDW \times NWY}$$

Where:

EIF	=	exposure from the food ingestion pathway (µg/kg body weight/day)
C_{food}	=	chemical concentration in food source (above ground 4.7E-08 µg/g)
SS	=	serving size of vegetation for person (toddler: 67 g/serving)
BAIG	=	bioavailable fraction via the ingestion route (assumed to be 1.0)

NSSW = number of servings per week that food (7 serving/week)

NWSY= number of weeks vegetation is consumed per year (assumed to be 52 weeks/year)

NWY = number of weeks in a year (52 weeks/year)

AAF = annual amortization factor (assumed to be 1)

LAF = lifetime amortization factor (assumed to be 1 for non-carcinogens)

BW = body weight of person (kg) (toddler: 16.5 kg)

NDW = number of days in a week (7 days per week)

4.2.2 Bioavailability Assessment

Absorption (or bioavailability) factors allow for the comparison of exposures to the same chemical via multiple routes (e.g., dermal and oral). As a general rule, Health Canada (2010a, 2012) recommends a relative absorption factor (RAF) of 1 (100%) for oral and inhalation exposures, with the exception of cases where data are available to support the use of another value. These absorption factors are considered relative as oral and inhalation TRV are generally based on the response to an exposure (delivered or airborne) dose, as opposed to an absorbed dose, and therefore are relative to the exposure dose estimated for the oral and inhalation pathways (Health Canada, 2012).

As has been discussed throughout the HHRA, and is further discussed in Section 7, the metals/metalloids and PAHs in the coal are tightly bound to the coal matrix, and are not considered to be bioavailable under typical environmental conditions (Ahrens and Morrisey, 2005; Triton, 2013a). Despite this, the HHRA has conservatively used the Health Canada recommended RAF of 1 (100%) for all oral and inhalation exposures, with the exception of the RAF used in the estimation of ingestion exposures to lead, in which case, as is supported by Health Canada, the PBET results were used. The maximum bioaccessibility of lead (0.28 or 28%) measured for the six coal samples submitted for analysis was used. The assumption that the coal constituents, other than lead, have an RAF of 100% for all oral and inhalation exposures is highly conservative and will result in the over-prediction of exposures and therefore associated risks.

For all COPCs, dermal exposures to soil were adjusted by relative dermal absorption factors (RAF_{DERM}) for comparison to oral TRVs. Where available, the RAF_{DERM} recommended by Health Canada (2010b) were assumed in the multi-media assessment for the COPCs in soil (i.e., the non-gaseous COPCs).

Table 4-2: Relative Absorption Factors for Dermal Exposure (RAF_{DERM}) for Selected Substances

COPC	Relative Absorption Factor for Dermal Exposure
Carcinogenic and non-carcinogenic PAHs	0.148a
Metals & Metalloids	
Aluminum	0.1[e]
Antimony	0.1[e]
Arsenic	0.03[a]
Barium	0.1[e]
Beryllium	0.1[e]
Boron	0.1[e]
Cadmium	0.01[a]
Chromium III	0.1[e]
Chromium VI	0.1[b]
Cobalt	0.1[e]
Copper	0.06[b]
Indium	0.1[e]
Iron	0.1[e]
Lanthanum	0.1[e]
Lead	0.006[c]
Manganese	0.1[e]
Mercury	1[c]
Molybdenum	0.1[e]
Nickel	0.091[b]
Selenium	0.01[b]
Strontium	0.1[e]
Tin	0.1[e]
Titanium	0.1[e]
Uranium	0.1[e]
Vanadium	0.1[e]
Zinc	0.1[b]
Dust Palliative Chemical Constituents	
Epichlorohydrin	1.0[d]
Others	
PCBs	0.14[b]
Sulfate	0.1[e]

Notes:
[a] All PAHs assumed to have same RAF_{derm} as benzo(a)pyrene, as per Health Canada (2010b) guidance
[b] From Health Canada (2010b)
[c] Based on professional judgement; see below discussion
[d] Not available; a value of 1.0 conservatively assumed
[e] Not available, a value of 0.1 was assumed for all metals that lack Health Canada (2010b) recommended values

Although Health Canada (2012a) recommended a value of 1.0 for mercury dermal bioavailability, a value of 0.1 was considered to be more appropriate. Personal communications with Mark Richardson (i.e., one of the authors of the Moody et al. [2009] paper that is the cited source of the Health Canada value) has indicated that subsequent issues have indicated that this estimate is overly conservative and that a value of 0.1 (common upper bound relative dermal absorption value for most metals) may be a more reasonable estimate of the mercury dermal absorption. It is noted that a value of 0.1 is provided as the dermal absorption value in the most recent version of the Health Canada DQRA spreadsheet tool (Health Canada 2011). Consequently, a value of 0.1 has been assumed for the current HHRA.

In the case of lead (Pb) dermal bioavailability, a value of 0.006 was used. This value was previously recommended by Health Canada in the draft interim guidance for lead (Pb). It is recognized that Health Canada does not currently use this guidance; however, the withdrawal of the guidance was related to the TRV rather than the bioavailability information. This value of 0.006 was used by Wilson and Richardson (2013).

4.2.3 Exposure Estimates

The exposure estimates were combined with the TRVs identified in Section 5 (the Toxicity Assessment) to estimate non-cancer hazards and cancer risks, with the risk estimates presented in Section 6, the Risk Characterization.

5 TOXICITY ASSESSMENT

The next step of the assessment involved the identification of TRVs representing an acceptable dose or concentration of exposure for the constituents being assessed. TRVs are developed by recognized regulatory authorities such as Health Canada, the US EPA, and the WHO. Given the Project falls under federal jurisdiction, where available and scientifically defensible, Health Canada inhalation and oral TRVs have been used. In cases where Health Canada TRVs were not available, or were determined not to be suitable (i.e., another agency recommended a TRV based on toxicological data that would not have been available to Health Canada at the time they derived their TRV), other international agency TRVs have been considered. The key Health Canada sources of information included:

- TRVs reported in Health Canada (2010b) – Part II: Health Canada Toxicological Reference Values (TRVs) and Chemical-Specific Factors;
- TRVs obtained through communication with Chemical Health Hazard Assessment Division (CHHAD); and
- Residential indoor air quality guidelines for various substances.

In addition to Health Canada, the positions of other major health agencies were reviewed and considered in the selection of TRVs. Of the various sources, the US EPA was given general preference when Health Canada TRVs were not available and the US EPA recommended TRV was determined to be scientifically defensible. Nevertheless, in some cases, various agencies were found to have more appropriate TRVs (e.g., based on newer toxicological data not available to either Health Canada or the US EPA at the time of their review) and, in such cases, values other than those recommended by Health Canada or the US EPA were adopted. Various agencies/sources considered in the review and compilation of the available TRVs for the COPCs include:

US EPA sources including, but not limited to:

- Integrated Risk Information System (http://www.epa.gov/IRIS/);
- Provisional Peer Reviewed Toxicity Values for Superfund (PPRTV) (http://hhpprtv.ornl.gov/index.html); and
- The Voluntary Children's Chemical Evaluation Program (http://www.epa.gov/oppt/vccep/pubs/chemmain.html);
- World Health Organization (WHO) sources including, but not limited to:
  - http://www.inchem.org/;

- http://www.who.int/foodsafety/chem/jecfa/publications/en/index.html; and
- http://www.euro.who.int/data/assets/pdf_file/0009/128169/e94535.pdf.

- Netherlands National Institute of Public Health and the Environment (RIVM) Human Toxicological Maximum Permissible Risk Levels available at: http://www.rivm.nl/bibliotheek/rapporten/711701025.pdf;
- Agency for Toxic Substances and Disease Registry (ATSDR) (US) Maximum Risk Levels (MRLs) available at http://www.atsdr.cdc.gov/toxprofiles/index.asp; and,
- California Office of Environmental Health Hazard Assessment (OEHHA) Reference Exposure Levels (RELs), inhalation unit risk estimates and oral slope factors, available at:
  - http://www.oehha.ca.gov/air/allrels.html
  - http://www.oehha.ca.gov/air/hot_spots/2009/AppendixB.pdf
  - http://www.oehha.ca.gov/air/pdf/acutem-o.pdf
  - http://www.oehha.ca.gov/air/pdf/acutep-z.pdf
  - http://www.oehha.ca.gov/air/chronic_rels/pdf/22Summs.pdf
  - http://www.oehha.ca.gov/air/chronic_rels/pdf/acrol-cresol.pdf
  - http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=42.

A thorough review of the TRVs available from Health Canada and each of the above listed sources was conducted to ensure that the most scientifically defensible TRV was identified.

The HHRA has considered both acute and chronic inhalation exposures, and as such, TRVs for both exposure durations have been identified, where available. Acute and chronic inhalation TRVs are defined as follows:

- Acute TRV: the air concentration of a chemical that can be tolerated without appreciable health effects on a short term basis (e.g. 1 hour, 24 hours);
- Non-Cancer Chronic TRV: the air concentration of a chemical that can be tolerated without appreciable health effects for exposures that occur continuously for exposures over an entire lifetime; and,
- Cancer Chronic TRV: the air concentration associated with a specified increase in the incremental lifetime cancer risk for exposures that occur for exposures over an entire lifetime.

In addition to acute and chronic inhalation TRVs, chronic oral TRVs have also been identified for the characterization of non-cancer hazards and cancer risks associated with exposures to the Project COPCs.

The TRVs provided by the above listed agencies are protective of critical sub-groups, or sensitive subpopulations (i.e., those with physical characteristics or conditions that may result in an increased likelihood of adverse effect to a given level of exposure, for example, the elderly or persons suffering from existing medical conditions). These sensitive subpopulations are considered by the agencies in the derivation of TRVs; when deriving TRVs, health agencies apply safety or uncertainty factors (i.e., an intraspecies/human variability uncertainty factor) to protect for sensitive subpopulations.

For the CACs, a range of the available acute and chronic ambient air quality objectives (AQO) available from Metro Vancouver, the BC MoE, the CCME and the WHO were used. For all of the CACs evaluated, with the exception of $PM_{2.5}$ and PM_{10}, the AQO represent levels below which appreciable adverse health effects are unlikely. The AQOs for CO, NO_2 and SO_2 have been derived to be protective of adverse health effects in the general population, including sensitive individuals/sub-populations. The AQO for PM_{10} and $PM_{2.5}$ have been used as exposure limits as an initial screen; however, it is acknowledged that the AQO for these substances are based on mortality endpoints, and further evaluation of the potential health effects associated with PM is presented in Section 6 of the HHRA, the Risk Characterization.

For threshold substances, which are typically non-carcinogenic, the TRVs are presented as an acceptable air concentration (for air and soil particulate inhalation exposures) or dose level (for ingestion, dermal exposures, and dust inhalation of COPC with no acceptable air concentration) that was derived such that it is unlikely to be associated with appreciable risks, based on the assumption that these substances act in a threshold manner with an air concentration/exposure dose below which no adverse effects are expected to occur. For carcinogenic chemicals, the TRV was presented as a unit risk estimate or cancer potency factor (i.e., slope factor) based on the assumption that carcinogens act in a non-threshold manner with any exposure capable of producing carcinogenic effects.

As discussed in Section 3.3 of the report, the physical chemical properties of the COPCs were reviewed and the COPCs were divided into two groups: gaseous and non-gaseous. The gaseous COPCs will be primarily present in only air and, thus, exposure limited to inhalation. The non-gaseous COPCs have the potential to be deposited from the atmosphere to soil or other surfaces in the Study Area and were therefore retained in the multimedia assessment conducted as part of the HHRA. The bioaccumulation potential of the gaseous COPCs was also evaluated; the gaseous COPCs that were determined to be bioaccumulative were also retained in the multi-media

assessment. For the COPCs included in the multi-media assessment, both the available inhalation and oral TRVs were reviewed.

For all COPCs, use of a TRV derived specifically for the route of exposure (i.e., inhalation, oral) was preferred over route-to-route extrapolation (e.g., to estimate risks from the inhalation route, it was generally preferential to use an inhalation TRV rather than an oral TRV). Nevertheless, for COPCs where no appropriate inhalation TRVs were available, but oral TRVs were recommended by a recognized health agency, the oral TRVs were used to assess risks associated with inhalation exposures.

The acute and chronic duration inhalation TRVs selected for comparison to receptor exposures are summarized in Tables 5-1 and 5-2, respectively, with the oral/dermal TRVs presented in Table 5-3. Included within these tables is a brief description of the endpoint/target organ (i.e., potential effects) representing the toxicological basis of the TRV. The supporting documentation and rationale for the selection of TRVs for use in the HHRA is provided in Appendix V.

Table 5-1: Summary of Acute Inhalation TRVs

Chemical	Acute Duration Inhalation TRV	Endpoint/Target Organ	Reference
Carcinogenic PAHs			
Benzo(a)pyrene	-	-	-
Benzo(a)anthracene	-	-	-
Benzo(b)fluoranthene	-	-	-
Benzo(g,h,i)perylene	-	-	-
Benzo(k)fluoranthene	-	-	-
Chrysene	-	-	-
Dibenzo(a,h)anthracene	-	-	-
Fluoranthene	-	-	-
Indeno(1,2,3-cd)pyrene	-	-	-
Phenanthrene	-	-	-
Non-Carcinogenic PAHs			
Acenaphthene	-	-	-
Acenaphthylene	-	-	-
Anthracene	-	-	-
Fluorene	-	-	-
Fluoranthene	-	-	-
Naphthalene	-	-	-
2-Methylnaphthalene	-	-	-
Pyrene	-	-	-

Chemical	Acute Duration Inhalation TRV	Endpoint/Target Organ	Reference
Metals & Metalloids			
Aluminum	-	-	-
Antimony	-	-	-
Arsenic	1 hour REL: 0.2 µg/m³	Decreased fetal weight	OEHHA, 2008
Barium	-	-	-
Beryllium	-	-	-
Boron	Acute MRL: 300 µg/m³	Nasal and throat irritation	ATSDR, 2010a
Cadmium	Acute MRL: 0.03 µg/m^3	Respiratory effects	ATSDR, 2012
Chromium III	-	-	-
Chromium VI	-	-	-
Cobalt	-	-	-
Copper	1 hour REL: 100 µg/m³	Sweet taste consistent with the onset of metal fume fever	OEHHA, 2008
Indium	-	-	-
Iron	-	-	-
Lanthanum	-	-	-
Lead	-	-	-
Manganese	8 hour REL: 0.17 µg/m³	Nervous system effects	OEHHA, 2008
Mercury	1 hour REL: 0.6 µg/m³	Developmental neurotoxicity	OEHHA, 2008
Molybdenum	-	-	-
Nickel	1 hour REL: 0.2 µg/m³	Immunotoxicity	OEHHA, 2008
Selenium	-	-	-
Strontium	-	-	-
Tin	-	-	-
Titanium	-	-	-
Uranium	-	-	-
Vanadium	1 hour REL: 30 µg/m³	Respiratory effects (bronchial irritation)	OEHHA, 2008
Zinc	-	-	-
Criteria Air Contaminants			
PM_{10}	24-hour AQO: 50 µg/m^3	Reduction in life expectancy: increased cardio-pulmonary and lung cancer mortality	Metro Vancouver (2011) BC MoE (2013) WHO (2006)
$PM_{2.5}$	24-hour AQO: 25 µg/m^3	Reduced lung function and chronic obstructive pulmonary disease	Metro Vancouver (2011) BC MoE (2013) WHO (2006)
CO	1-hour AQO: 14,300 µg/m^3 8-hour AQO: 5,500 µg/m^3	Elevated carboxyhaemoglobin in blood	BC MoE (2013)

Chemical	Acute Duration Inhalation TRV	Endpoint/Target Organ	Reference
NO_2	1-hour AQO: 200 µg/m^3	Reduced lung function and airway responsiveness.	Metro Vancouver (2011) WHO (2010)
SO_2	1-hour AQO: 450 µg/m^3 24-hour AQO: 20 µg/m^3	Changes in pulmonary function and respiratory symptoms	*1 hour AQO:* Metro Vancouver (2011) BC MoE (2013) CCME (1999) *24-hour AQO:* WHO (2006)
Diesel Particulate Matter	-	-	-
VOCs			
Acetaldehyde	1 hour REL: 470 µg/m3	Respiratory effects (decreased Forced Expiratory Volume)	OEHHA, 2008
Acrolein	1 hour REL: 2.5 µg/m³	Eye and nasal irritation	OEHHA, 2008
	1 hour ReV: 11 µg/m³	Eye, nasal and throat irritation	TCEQ, 2014
Benzene	Subchronic PPRTV: 80 µg/m^3	Decreased lymphocyte count	US EPA, 2009a
1,3-Butadiene	1 hour REL: 660 µg/m³	Decreased fetal weight	OEHHA, 2013
Ethylbenzene	Acute MRL: 21,700 µg/m^3	Auditory threshold shifts	ATSDR, 2000
Ethylene	-	-	-
Formaldehyde	100 µg/m^3 (30 min)	Eye and nasal irritation	WHO (2010)
Hexachlorobenzene	-	-	-
n-Hexane	-	-	-
Propionaldehyde	-	-	-
Propylene (1-Propene)	-	-	-
Toluene	Short-term exposure limit (8 hour): 15,000 µg/m^3	Ocular and nasal irritation	Health Canada, 2011
2,2,4-Trimethylpentane	-	-	-
Styrene	1 hour REL: 21,000 µg/m^3	Nose and throat irritation	OEHHA, 2008
Xylenes	Acute MRL: 8,700 µg/m^3	Mild respiratory and neurological effects	ATSDR, 2007
Dust Palliatives Chemical Constituents			
Adipic Acid[a]	-	-	-
Diethylaminoethanol	-	-	-
Diethylenetriamine[a]	-	-	-
Epichlorohydrin[a]	Acute REL: 1300 µg/m^3	Respiratory and eye irritation	OEHHA (2008)
Linear Alkyl Sulfonate	-	-	-

Chemical	Acute Duration Inhalation TRV	Endpoint/Target Organ	Reference
Propylene Oxide	-	-	-
Propylene Glycol	-	-	-
Succinic acid	-	-	-
Others			
Polychlorinated Biphenyls (PCB)	-	-	-
Sulfate	1 hour REL: 120 µg/m^3	Respiratory effects (small changes in airway function tests)	OEHHA (2008)

Notes:
UR – unit risk
PEF – potency equivalence factor
RfC – reference concentration
REL – reference exposure level
MRL – minimal risk level
TCA – tolerable concentration in air
TC – tolerable concentration
AQO – air quality objective
Int. - Intermediate
µg/m^3 – micrograms per cubic meter
- indicates that no acute inhalation TRV was identified for the COPC
a - Chemical constituents of the GE product Dustreat DC9148; listed on the MSDS as adipic acid, diethylenetriamine, epichlorohydrin polymer

Table 5-2: Summary of Chronic Inhalation TRVs

Chemical	Inhalation TRV	Endpoint/Target Organ	Reference
Carcinogenic PAHs			
Benzo(a)pyrene	UR: 3.1 x 10^{-5} (µg/m^3)$^{-1}$	Cancer: respiratory tract	Health Canada (2010b)
Benzo(a)anthracene	UR: 3.1 x 10^{-6} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.1	Health Canada (2012)
Benzo(b)fluoranthene	UR: 3.1 x 10^{-6} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.1	Health Canada (2012)
Benzo(g,h,i)perylene	UR: 3.1 x 10^{-7} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.01	Health Canada (2012)
Benzo(k)fluoranthene	UR: 3.1 x 10^{-6} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.1	Health Canada (2012)
Chrysene	UR: 3.1 x 10^{-7} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.01	Health Canada (2012)
Dibenzo(a,h)anthracene	UR: 3.1 x 10^{-5} (µg/m^3)$^{-1}$	Based on benzo(a)pyrene UR and a PEF of 1	Health Canada (2012)

Chemical	Inhalation TRV	Endpoint/Target Organ	Reference
Fluoranthene	UR: 3.1 x 10^{-8} $(\mu g/m^3)^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.001	Health Canada (2012)
Indeno(1,2,3-cd)pyrene	UR: 3.1 x 10^{-6} $(\mu g/m^3)^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.1	Health Canada (2012)
Phenanthrene	UR: 3.1 x 10^{-8} $(\mu g/m^3)^{-1}$	Based on benzo(a)pyrene UR and a PEF of 0.001	Health Canada (2012)
Non-Carcinogenic PAHs			
Acenaphthene	-	-	-
Acenaphthylene	-	-	-
Anthracene	-	-	-
Fluorene	-	-	-
Fluoranthene	-	-	-
Naphthalene	RfC: 10 $\mu g/m^3$	Nasal lesions	Health Canada (2013a)
2-Methylnaphthalene	-	-	-
Pyrene	-	-	-
Metals & Metalloids			
Aluminum	-	-	-
Antimony	-	-	-
Arsenic	UR: 6.4 x 10^{-3} $(\mu g/m^3)^{-1}$	Lung cancer	Health Canada (2010b)
	TCA: 1 $\mu g/m^3$	Lung cancer (non-genotoxic mechanism)	RIVM (2001)
Barium	TCA: 1 $\mu g/m^3$	Cardiovascular effects	RIVM (2001)
Beryllium	RfC: 0.02 $\mu g/m^3$	beryllium sensitization and progression to chronic beryllium disease (CBD)	US EPA (1998a)
	UR : 2.4 x 10^{-3} $(\mu g/m^3)^{-1}$	Lung cancer	US EPA (1998a)
Boron	MRL: 300 $\mu g/m^3$	Nasal and throat irritation	ATSDR (2010) Acute MRL indicated to be protective of chronic exposures

Chemical	Inhalation TRV	Endpoint/Target Organ	Reference
Metals & Metalloids (Cont'd)			
Cadmium	UR: 9.8 x 10^{-3} $(\mu g/m^3)^{-1}$	Lung cancer	Health Canada (2010b)
	Chronic MRL: 0.01 $\mu g/m^3$	Renal toxicity	ATSDR (2012a)
Chromium III	Int. MRL: 5 $\mu g/m^3$	Trace to mild septal cell hyperplasia and chronic interstitial inflammation	ATSDR (2012b)
Chromium VI	UR: 7.6 x 10^{-2} $(\mu g/m^3)^{-1}$	Lung cancer	Health Canada (2010b)
	RfC: 0.1 $\mu g/m^3$	Respiratory effects: lactate dehydrogenase in bronchial alveolar fluid	US EPA (1998b)
Cobalt	Chronic MRL: 0.1 $\mu g/m^3$	Respiratory effects: interstitial lung disease	ATSDR (2004)
Copper	TCA: 1 $\mu g/m^3$	Respiratory and immunological effects	RIVM (2001)
Indium	-	-	-
Iron	-	-	-
Lanthanum	-	-	-
Lead	0.15 $\mu g/m^3$	IQ loss	US EPA (2008a)
Manganese	RfC: 0.05 $\mu g/m^3$	Neurotoxic effects	US EPA (1993a)
Mercury	RfC: 0.3 $\mu g/m^3$	Neurobehavioral effects	US EPA (1995)
Molybdenum	TCA: 12 $\mu g/m^3$	Body weight effects	RIVM (2001)
Nickel	TC: 3.5 x 10^{-3} $\mu g/m^3$ (for nickel sulphate)	Lung and nasal epithelium lesions	Health Canada (2010b)
	UR: 1.3 x 10^{-3} $(\mu g/m^3)^{-1}$ (for oxidic, sulphidic and soluble nickel)	Lung cancer	Health Canada (2010b)
Selenium	Chronic REL: 20 $\mu g/m^3$	Selenosis	OEHHA (2001 and 2008)
Strontium	-	-	-
Tin	-	-	-
Titanium	-	-	-
Uranium	Chronic MRL: 0.04 $\mu g/m^3$	Renal toxicity	ATSDR (2013)
Vanadium	Chronic MRL: 0.1 $\mu g/m^3$	Respiratory effects: degeneration of epiglottis respiratory epithelium	ATSDR (2012c)
Zinc	-	-	-

Chemical	Inhalation TRV	Endpoint/Target Organ	Reference
Criteria Air Contaminants			
PM_{10}	Annual AQO: 20 µg/m^3	Reduction in life expectancy: increased cardio-pulmonary and lung cancer mortality	Metro Vancouver (2011) BC MoE (2013) WHO (2006)
$PM_{2.5}$	Annual AQO: 8 µg/m^3 Annual AQO (planning goal): 6 µg/m^3	Reduced lung function and chronic obstructive pulmonary disease	Metro Vancouver (2011) BC MoE (2013)
CO	Long term AQO: 7000 µg/m^3	Increased ER visits for ischemic heart disease, congestive heart failure and cardiovascular disease	WHO, 2010
NO_2	Annual AQO: 40 µg/m^3	Reduced lung function and airway responsiveness.	Metro Vancouver (2011) WHO (2006)
SO_2	Annual AQO: 25 µg/m^3	Changes in pulmonary function and respiratory symptoms	BC MoE (2013)
Diesel Particulate Matter (DPM)	UR: 3 x 10^{-4} (µg/m^3)$^{-1}$	Lung cancer	OEHHA (2009)
	RfC: 5 µg/m^3	Pulmonary inflammation and histopathology	US EPA (2003a)
VOCs			
Acetaldehyde	TC: 390 µg/m^3	Nasal olfactory epithelial lesions	Health Canada (2000)
	TC_{05} (converted to a UR): 5.8 x 10^{-7} (µg/m^3)$^{-1}$	Nasal squamous cell carcinoma or adenocarcinoma	CEPA (1999a)
Acrolein	Chronic REL: 0.35 µg/m^3	Nasal epithelial lesions	OEHHA (2008)
	Chronic ReV: 2.7 µg/m^3	Nasal epithelial lesions	TCEQ (2014)
Benzene	RfC: 30 µg/m^3	Decreased lymphocyte count	US EPA (2003b)
	UR: 3.3x10^{-6} (µg/m^3)$^{-1}$	Hematoxicity	Health Canada (2010b)
1,3-Butadiene	RfC: 2 µg/m^3	Ovarian atrophy	US EPA (2002)
	UR: 5.9 × 10^{-6} (µg/m^3)$^{-1}$	Cancer: leukemia	CEPA (1999b)
Ethylbenzene	Chronic MRL: 260 µg/m^3	Nephropathy	ATSDR (2010b)
Ethylene	-	-	-
Formaldehyde	50 µg/m^3 (8 hour guideline considered protective of long-term exposure)	Eye and nasal irritation	Health Canada (2006)
Hexachlorobenzene	UR: 4.6 x 10^{-4} (µg/m^3)$^{-1}$	Hepatocellular carcinomas	US EPA (1996a)
n-Hexane	700 µg/m^3	Peripheral neuropathy	Health Canada (2010b)
Propionaldehyde	8 µg/m^3	Atrophy of the olfactory epithelium	US EPA (2008b)

Chemical	Inhalation TRV	Endpoint/Target Organ	Reference
VOCs (Cont'd)			
Propylene (1-Propene)	Chronic REL: 3000 µg/m^3	Squamous metaplasia, epithelial hyperplasia and inflammation of the nasal cavity	OEHHA (2008)
Styrene	TC: 92 µg/m^3	Developmental effects	Health Canada (2010b)
Toluene	Residential Long-Term Indoor Air Guideline: 2300 µg/m^3	Neurobehavioural effects	Health Canada (2011)
2,2,4-Trimethylpentane	-	-	-
Xylenes	TC: 180 µg/m^3	Maternal effects, developmental effects	Health Canada (2010b)
Dust Palliatives Chemical Constituents			
Adipic Acid[a]	-	-	-
Diethylaminoethanol	-	-	-
Diethylenetriamine[a]	-	-	-
Epichlorohydrin[a]	RfC: 1 µg/m^3	Changes in the nasal turbinates	US EPA (1992)
	UR: 1.2x10^{-6} (µg/m^3)$^{-1}$	Nasal cavity tumours	US EPA (1994b)
Linear Alkyl Sulfonate	-	-	-
Propylene Oxide	RfC: 30 µg/m^3	Development of nest-like fold in nasal respiratory epithelium	US EPA (1990)
	UR: 3.7 x 10^{-6} (µg/m^3)$^{-1}$	Nasal cavity hemangioma or hemangiosarcoma	US EPA (1994a)
Propylene Glycol	-	-	-
Succinic acid	-	-	-
Others			
Polychlorinated Biphenyls (PCB)	-	-	-
Sulfate	-	-	-

Notes:
UR – unit risk
PEF – potency equivalence factor
RfC – reference concentration
REL – reference exposure level
MRL – minimal risk level
TCA – tolerable concentration in air
TC – tolerable concentration
AQO – air quality objective
Int. - Intermediate
µg/m^3 – micrograms per cubic meter
- indicates that no chronic inhalation TRV was identified for the COPC
[a] - Chemical constituents of the GE product Dustreat DC9148; listed on the MSDS as adipic acid, diethylenetriamine, epichlorohydrin polymer

The inhalation TRVs selected for acrolein and diesel particulate matter are further discussed below.

Acute and chronic inhalation TRVs recommended for acrolein by both the OEHHA (2008) and the Texas Commission on Environmental Quality (TCEQ) (2014) were selected for use in the HHRA. Although the TCEQ was not selected as a preferred source in the TRV hierarchy, the agency has conducted a very recent (2014) and thorough toxicological review of acrolein, and has recommended by acute and chronic inhalation reference values (ReVs). As discussed in Appendix V, the same 2008 study was the basis of both the OEHHA and the TCEQ chronic inhalation TRV; however, the two agencies differed on their selection of uncertainty factors for the derivation of the TRV. The TCEQ documented their thorough assessment and selection of uncertainty factors, and the overall uncertainty factor selected by the agency is considered to be appropriate. Non-cancer risk estimates for acrolein presented in Section 6 of the HHRA are based on both the OEHHA acute and chronic RELs and the TCEQ acute and chronic ReVs.

Although diesel emissions are classified as a known carcinogen (IARC) or a likely human carcinogen (US EPA), few agencies, including the US EPA, have derived carcinogenic TRVs (e.g. inhalation unit risk, slope factor) for diesel emissions; the US EPA (http://www.epa.gov/iris/subst/0642.htm) indicates that a quantitative estimate of carcinogenic risk from inhalation exposure to diesel emissions has not been derived based on the absence of adequate data to develop a sufficiently confident dose-response relationship from the epidemiological studies. The inhalation UR included in Table 5-1 for DPM of $3.0x10^{-4}$ $(\mu g/m^3)^{-1}$ is recommended by the OEHHA, and is based on human occupational exposure lung tumour incidence in studies of US railroad workers (OEHHA, 2009). Personal communication with Health Canada suggests that the OEHHA (2009) DPM unit risk value is not widely accepted within Canada and may overestimate the carcinogenic potency of diesel particulates. Nevertheless, the OEHHA inhalation UR was used in the HHRA to characterize carcinogenic risks associated with DPM. Given the conservatism in the TRV, the uncertainty in the resulting cancer risk estimates was carefully considered.

The oral TRVs used in the HHRA for COPCs retained for evaluation in the multi-media assessment are presented below in Table 5-3.

Table 5-3: Summary of Oral and Dermal TRVs

Chemical	Oral TRV	Endpoint/Target Organ	Reference
Carcinogenic PAHs			
Benzo(a)pyrene	SF: 2.3×10^{-3} $(\mu g/kg\ bw/d)^{-1}$	Gastric tumours: mostly squamous cell papillomas with a few carcinomas	Health Canada (2010b)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V

Chemical	Oral TRV	Endpoint/Target Organ	Reference
Carcinogenic PAHs (Cont'd)			
Benzo(a)anthracene	SF: 2.3 x 10^{-4} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.1	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Benzo(b)fluoranthene	SF: 2.3 x 10^{-4} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.1	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Benzo(g,h,i)perylene	SF: 2.3 x 10^{-5} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.01	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Benzo(k)fluoranthene	SF: 2.3 x 10^{-4} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.1	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Chrysene	SF: 2.3 x 10^{-5} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.01	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Dibenzo(a,h)anthracene	SF: 2.3 x 10^{-3} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 1	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Fluoranthene	SF: 2.3 x 10^{-6} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.001	Health Canada (2012)
	RfD: 40 µg/kg bw/d	Nephropathy, increased liver weights, haematological alterations, clinical effects	Health Canada (2013b)
Indeno(1,2,3-cd)pyrene	SF: 2.3 x 10^{-4} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.1	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Phenanthrene	SF: 2.3 x 10^{-6} (µg/kg bw/d)$^{-1}$	Based on benzo(a)pyrene SF and PEF of 0.001	Health Canada (2012)
	TDI: 30 µg/kg bw/d	Based on TDI for pyrene	See Appendix V
Non-Carcinogenic PAHs			
Acenaphthene	RfD: 60 µg/kg bw/d	hepatotoxicity	Health Canada (2013b)
Acenaphthylene	-	-	-
Anthracene	RfD: 300 µg/kg bw/d	No observed effects	Health Canada (2013b)
Fluorene	RfD: 40 µg/kg bw/d	Decreased red blood cells, packed cell volume and haemoglobin	Health Canada (2013b)
Naphthalene	TDI: 20 µg/kg bw/d	Decreased body weight	Health Canada (2010b)
2-Methylnaphthalene	TDI: 4 µg/kg bw/d	Pulmonary alveolar proteinosis	Health Canada (2010b)
Pyrene	TDI: 30 µg/kg bw/d	Kidney effects: renal tubular pathology, decreased kidney weight	Health Canada (2010b)

Chemical	Oral TRV	Endpoint/Target Organ	Reference
Metals & Metalloids			
Aluminum	TDI : 300 µg/kg bw/d	Not available	Health Canada (2013b)
Antimony	TDI: 3 µg/kg bw/d	Decrease in thymus to body weight ratios	Health Canada (2013b)
Arsenic	SF: 1.8×10^{-3} (µg/kg bw/d)$^{-1}$	Bladder, liver, and lung cancer	Health Canada (2010b)
	RfD: 0.3 µg/kg bw/d	Skin lesions: hyper-pigmentation, keratosis	US EPA (1993b)
Barium	TDI: 200 µg/kg bw/d	Renal lesions	Health Canada (2010b)
Beryllium	RfD: 2 µg/kg bw/d	Small intestinal lesions	Health Canada (2013b)
Boron	ADI : 17.5 µg/kg bw/d	Testicular atrophy, infertility	Health Canada (2010b)
Cadmium	TDI: 0.83 µg/kg bw/d	Increased urinary excretion of β2-microglobulin	WHO JEFCA (2011)
Chromium III	RfD: 1500 µg/kg bw/d	No effects observed	Health Canada (2013b)
Chromium VI	Oral MRL: 0.9 µg/kg bw/d	Non-neoplastic lesions of the duodenum	ATSDR (2012b)
	SF: 4.2×10^{-4} (µg/kg bw/d)$^{-1}$	Gastric tumours	OEHHA (2009)
Cobalt	MRL: 10 µg/kg bw/d	Haematological effects	Health Canada (2013b)
Copper	Toddler UL: 91 µg/kg bw/d Adult UL: 141 µg/kg bw/d	Hepatotoxicity and gastrointestinal effects	Health Canada (2010b)
Indium	-	-	-
Iron	Provisional Maximum TDI: 800 µg/kg bw/d	Not reported	Health Canada (2013b)
Lanthanum	RfD: 500 µg/kg bw/d	Reduced serum or urine phosphate	NSF (2010)
Lead	Toddler: 0.6 µg/kg bw/d	Neurological effects: decreases in IQ	Based on WHO/JECFA (2011) Potency Estimate
	Adult: 1.3 µg/kg bw/d	Cardiovascular effects: increase in systolic blood pressure	
Manganese	Toddler UL: 136 µg/kg bw/d Adult UL: 156 µg/kg bw/d	Parkinsonian-like neurotoxicity	Health Canada (2010b)
Mercury	TDI: 0.3 µg/kg bw/d	Nephrotoxicity	Health Canada (2010b)
Molybdenum	Toddler UL: 23 µg/kg bw/d Adult UL: 28 µg/kg bw/d	Reproductive effects	Health Canada (2010b)
Nickel	TDI: 11 µg/kg bw/d for soluble nickel (nickel chloride and nickel sulphate)	Reproductive effects: post-implantation perinatal lethality	Health Canada (2010b)
Selenium	Toddler UL: 6.2 µg/kg bw/d Adult UL: 5.7 µg/kg bw/d	Selenosis	Health Canada (2010b)

Chemical	Oral TRV	Endpoint/Target Organ	Reference
Metals & Metalloids (Cont'd)			
Strontium	RfD: 600 µg/kg bw/d	Rhachitic bones (soft bones)	Health Canada (2013b) US EPA (1996b)
Tin	TDI: 2,000 µg/kg bw/d	Acute manifestations of gastric irritation	Health Canada (2013b)
Titanium	RfD: 3,000 µg/kd bw/d	No treatment related effects observed in study	NSF (2005)
Uranium	TDI : 0.6 µg/kg bw/d	Renal toxicity	Health Canada (2010b)
Vanadium	pTDI: 15 µg/kg bw/d	Not reported	Health Canada (2013b)
Zinc	Toddler UL: 500 µg/kg bw/d	Increased infant growth: length, weight and head circumference	Health Canada (2010b)
	Adult UL: 600 µg/kg bw/d	Reduced iron and copper status	
Volatile Organic Compounds			
Ethylene	-	-	-
Hexachlorobenzene	pTDI : 0.27 µg/kg bw/d	Not reported	Health Canada (2013b)
	Oral SF : 1.6×10^{-3} $(\mu g/kg\ bw/d)^{-1}$	Hepatocellular carcinomas	US EPA (1996a)
2,2,4-Trimethylpentane	-	-	-
Dust Palliatives Constituents			
Adipic Acid[a]	RfD:4,000 µg/kd bw/d	Reduced survival, diarrhea, decreased body weight and intestinal and liver pathology	NSF (2006)
Diethylaminoethanol	-	-	-
Diethylenetriamine[a]	-	-	-
Epichlorohydrin[a]	RfD : 6 µg/kg bw/d	Reduced male fertility	US EPA (2006b)
	SF: 9.9×10^{-6} $(\mu g/kg\ bw/d)^{-1}$	Forestomach papillomas and carcinomas	US EPA (1994)
Linear Alkyl Sulfonate	-	-	-
Propylene Glycol	RfD : 20,000 µg/kg bw/d	Reduced red blood cell counts and hyperglycemia	US EPA (2008)
Succinic acid	-	-	-
Others			
PCBs	Oral TDI: 2.3×10^{-6} µg/kg bw/d	Developmental effects	Health Canada (2010b)
Sulfate	-	-	-

Notes:
SF – slope factor
PEF – potency equivalence factor
RfD – reference dose
RsD – risk specific dose
REL – reference exposure level
TDI – tolerable daily intake
UL – tolerable upper intake level
PPRTV: US EPA provisional peer reviewed toxicity values
IQ – intelligence quotient
µg/kg bw/d – micrograms per kilogram body weight per day
- indicates that oral TRV was identified for the COPC
[a] chemical constituents of the GE product Dustreat DC9148; listed on the MSDS as adipic acid, diethylenetriamine, epichlorohydrin polymer

As noted in Tables 5-1 to 5-3, no TRVs were identified for select COPCs including acenaphthylene, ethylene, 2,2,4-trimethylpentane, diethylaminoethanol, diethylenetriamine, linear alkyl sulfonate and succinic acid.

Based on a lack of TRVs for several of the dust palliative constituents, an alternative approach has been used in the characterization of risks associated with exposures to the dust palliatives. Of the constituents with available TRVs, epichlorohydrin has the highest relative toxicity (based on a comparison of the available TRVs). As indicated above, no TRVs (inhalation or oral) were identified for diethylaminoethanol, diethylenetriamine, linear alkyl sulfonate and succinic acid, each of which is a constituent of the GE dust palliatives. To further evaluate the relative toxicity of these COPCs compared to epichlorohydrin, the Worksafe BC occupational exposure limits (8 hour Time Weighted Average [TWA] limits) were reviewed; it is emphasized that the review of these limits was conducted to evaluate the relative toxicity of the dust palliative constituents, and the TWA limits were not used as TRVs in the HHRA. Table 5-4 presents the available Worksafe BC TWA limit for epichlorohydrin, as well as for the constituents lacking TRVs.

Table 5-4: Worksafe BC 8 hour Time Weighted Average Limits for Dust Palliative Constituents

Chemical	Worksafe BC 8 hour TWA
Epichlorohydrin	0.1 ppm / 390 µg/m^3
Diethylaminoethanol	2 ppm / 9,600 µg/m^3
Diethylenetriamine	1 ppm / 4,200 µg/m^3
Linear alkyl sulfonate	Not Available
Succinic acid	Not Available

ppm – parts per million

Although no Worksafe BC limits were available for linear alkyl sulfonate and succinic acid, the limits available for epichlorohydrin, diethylaminoethanol and diethylaminotriamine support the assumption that epichlorohydrin has the highest relative toxicity for the dust palliative constituents. Despite the lack of TRVs and occupational exposure limits for linear alkyl sulfonate and succinic acid, the toxicity of these COPCs is unlikely to exceed that of epichlorohydrin based on how they are used. Succinic acid is a food additive (as an acidity regulator), is used in pharmaceuticals, and is on the US Food and Drug Administration's (FDAs) Generally Recognized as Safe (GRAS) list. Linear alkyl sulfonate is a surfactant that is widely used in household and personal care products.

The lack of TRVs for these COPCs is further discussed in Section 7, the Uncertainty and Sensitivity Analysis.

The TRVs identified in Tables 5-1 to 5-3 were combined with the exposure estimates from Section 4 to estimate cancer and non-cancer risks associated with exposures to the Project COPCs. The risk estimates are presented in the following section, Section 6, Risk Characterization.

6 RISK CHARACTERIZATION

Non-cancer and cancer risks for receptors of concern were estimated based on exposure estimates (from Section 4, Exposure Assessment) and TRVs (from Section 5, Toxicity Assessment). As presented in Section 5, TRVs were expressed as reference air concentrations (µg/m^3), reference doses (µg/kg body weight/day) or cancer potency factor estimates [(µg/m^3)$^{-1}$ or (µg/kg body weight/day)$^{-1}$].

Non-cancer risks associated with the inhalation of Project emissions and suspended soil particulates (i.e., dust from soil) were estimated as HQ values according to the following formula:

$$HQ = \frac{\text{Amortized Air/Dust Concentration (µg/m}^3\text{)}}{\text{Reference Concentration (µg/m}^3\text{)}}$$

Non-cancer risks as a result of oral/dermal exposures, and inhalation exposures where inhalation TRVs were not available, were estimated as HQ values according to the following formula:

$$HQ = \frac{\text{Estimated Exposure (µg/kg body weight/day)}}{\text{Reference Dose (µg/kg body weight/day)}}$$

A Total HQ (for all routes of exposure) was estimated for exposure to non-carcinogenic COPCs as the sum of the individual HQ for all applicable exposure pathways as follows:

$$HQ_{\text{all routes}} = HQ_{\text{inhalation air}} + HQ_{\text{ingestion soil}} + HQ_{\text{dermal soil}} + HQ_{\text{inhalation soil dust}} + HQ_{\text{ingestion produce}}$$

Cancer risks from the inhalation of Project emissions and suspended soil particulates were estimated as Incremental Lifetime Cancer Risks (ILCRs) as follows:

$$\text{ILCR} = \text{Amortized Air/Dust Concentration (µg/m}^3\text{)} \times \text{Inhalation Unit Risk (µg/m}^3\text{)}^{-1}$$

For oral and dermal exposures, and for inhalation exposures where unit risk estimates were not available, ILCRs were evaluated using estimated lifetime daily exposures. Cancer risks were estimated as ILCR values according to the following formulas:

$$\text{ILCR} = \text{Estimated Lifetime Daily Exposure (µg/kg/day)} \times \text{Cancer Potency Factor (µg/kg/day)}^{-1}$$

A Total ILCR (for all routes of exposure) was estimated for exposure to carcinogenic COPCs as the sum of the individual ILCR for the applicable exposure routes as follows:

$$ILCR_{\text{all routes}} = ILCR_{\text{inhalation air}} + ILCR_{\text{ingestion soil}} + ILCR_{\text{dermal soil}} + ILCR_{\text{inhalation soil dust}} + ILCR_{\text{ingestion produce}}$$

The resulting chronic HQs and ILCRs were compared to the Health Canada negligible risk levels.

Health Canada (2012) guidance indicates that total Hazard Quotients can be interpreted according to the following general guidelines:

- < 0.2 = negligible (i.e., acceptable) human health risks; and
- > 0.2 = potential unacceptable risks which may require mitigation or more detailed assessment.

Health Canada's negligible risk level of 0.2 (or 20% of the TRV) for non-carcinogens allows for 80% of the acceptable exposure level (i.e., as defined by the TRV) to come from other sources; this approach is based on the potential for exposures to a chemical in air, soil, water, food and consumer products (i.e., 20% of the acceptable exposure is typically allocated to each of these 5 media/sources). The non-cancer risk estimates associated with exposures to Project emissions were conservatively compared to the Health Canada negligible risk level of 0.2; this approach is considered conservative based on the multi-media assessment that was conducted for the non-gaseous COPCs, which assumed exposures to air, soil and vegetation.

In addition, Health Canada (2012) indicates that an ILCR less than 1×10^{-5} is generally considered to be acceptable, while an ILCR greater than 1×10^{-5} may indicate that some form of mitigation or more detailed site-specific analysis is required. Similar to non-cancer risks, interpretation of ILCR estimates greater than 1×10^{-5} requires consideration of the overall risk assessment process and assumptions.

In contrast to the estimation of HQs, the estimation of ILCRs is exclusive of background exposures; the HHRA predicted the incremental cancer risk, above background, from exposures associated with the Project emissions.

In the case of acute exposures for all COPCs, risk estimates were compared to an acceptable HQ of 1.0. An acceptable HQ of 1.0 was also applied to chronic inhalation exposures to the CACs and substances where irritation is the key concern (e.g., formaldehyde), as exposures to these COPCs in other media does not contribute to risks from inhalation.

6.1 COPCs with Additive Effects

Where evidence was available to suggest that the critical effects of two or more COPCs occur at the same target site (*i.e.*, tissue or organ system), an assessment of the potential for additive effects at the target site was conducted. If the available data suggested two or more COPCs exert (or could potentially exert) their critical

effects by similar mechanisms of action, then the risks associated with exposure to those COPCs were conservatively assumed to be additive.

Potential additive interactions were identified for COPCs with the following critical effects:

- Nasal, ocular or respiratory irritation associated with acute inhalation exposures;
- Nasal and respiratory irritation associated with chronic inhalation exposures;
- Lung cancer, leukemia, developmental effects, renal toxicity and neurotoxicity associated with chronic inhalation exposures; and,
- Renal toxicity, hepatotoxicity and reproductive toxicity associated with chronic oral exposures.

Table 6-1 presents a summary of the COPCs with potential additive effects.

Table 6-1: COPCs with Potential Additive Effects

Exposure Route and Duration	Critical Effect	COPCs Considered Additive
Acute inhalation	Nasal irritation	Acrolein, boron, formaldehyde, toluene, styrene
	Ocular irritation	Acrolein, epichlorohydrin, formaldehyde, toluene
	Respiratory irritation	Acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulphate
Chronic Inhalation	Nasal irritation/nasal lesions	Acetaldehyde, boron, acrolein, epichlorohydrin, formaldehyde, naphthalene, nickel, propionaldehyde, propylene oxide
	Respiratory irritation	Chromium III, chromium VI, cobalt, copper, formaldehyde, NO_2, SO_2, propylene, vanadium
	Lung cancer	Arsenic, beryllium, cadmium, chromium VI, nickel, PAHs
	Leukemia	Benzene, 1,3-butadiene
	Developmental effects	Styrene, xylenes
	Renal toxicity	Cadmium, ethylbenzene, uranium
	Neurotoxicity	n-Hexane, manganese, mercury, toluene
Chronic Oral	Renal toxicity	Barium, pyrene, mercury, uranium
	Hepatotoxicity	Acenaphthene, copper,
	Reproductive Toxicity	Molybdenum, nickel

In addition, given their structural similarities and Health Canada (2012) guidance, all carcinogenic PAHs were considered to act in an additive manner, as were all non-carcinogenic PAHs. On this basis, all Hazard Quotient values and Incremental Lifetime Cancer Risks for PAHs were summed.

The remaining COPCs evaluated in the HHRA were not considered to be additive.

6.2 Results of the HHRA

As described throughout the report, the HHRA has used a series of conservative assumptions, including that receptors are exposed to the maximum predicted concentrations of Project emissions, to estimate exposures and associated risks to receptors of concern. The conservative approach undertaken in the HHRA will tend to overestimate potential exposures and associated risks to receptors of concern.

Health Canada recommended methods and assumptions have been used, and for aspects of the HHRA where no Health Canada recommendations are available, guidance established by other agencies and/or best professional judgment have been used. Risk estimates for each receptor group are presented in the following sections.

As discussed in Section 4.1.2.2, the mean background soil concentrations of the majority of the metals (antimony, arsenic, cadmium, chromium (+3), chromium (+6), cobalt, copper, lead, lithium, manganese, molybdenum, nickel, silver, tin, uranium, vanadium, zinc, aluminum, iron, thallium and titanium) and select PAHs (benzo(a)pyrene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-cd)pyrene and phenanthrene) were higher than the mean concentrations of these parameters measured in the coal. Soil concentrations of these COPCs are not expected to increase due to deposition, and therefore, summing the deposition rates in for these COPCs is conservative and will tend to over predict environmental concentrations, exposures and associated risks to receptors of concern. Despite this, risk estimates for the Cumulative Scenario (i.e., Baseline + Project) have been conservatively presented in the following sections. As is further discussed in Section 7, the Cumulative Scenario risk estimates for the above COPCs are considered to overpredict risks to receptors of concern.

6.2.1 Health Risks Associated with Exposures to $PM_{2.5}$

As presented in earlier sections of the report, the predicted $PM_{2.5}$ concentrations for the Project have been compared to the lowest of the Metro Vancouver, BC MoE, CCME and WHO AAQO; the predicted concentrations have been compared to the Metro Vancouver and the BC MoE AAQO of 25 µg/m³ (24 hour maximum) and 8 µg/m³ (annual average), as well as the Metro Vancouver and BC MoE planning goal for $PM_{2.5}$ of 6 µg/m³. The

predicted $PM_{2.5}$ concentrations from the Project plus background were less than these AAQO for all receptors, with the maximum concentrations plus background at a residential receptor predicted to be 14.0 µg/m³ (24 hour maximum) and 4.9 µg/m³ (annual average). The current Metro Vancouver and BC MoE $PM_{2.5}$ air quality objective of 8 µg/m³ is among the lowest of the available guidelines across Canada and world-wide.

Appendix IV provides a discussion on the current human health in the region, with a focus on $PM_{2.5}$. The information contained in the Appendix is intended to provide a reference (or baseline) to evaluate the potential for the Project to impact human health, and was not used to adjust the quantitative risk estimates presented in the following sections for the receptors of concern, but rather to evaluate the current health status in the area with respect to $PM_{2.5}$.

In the information that was reviewed, WHO (2006) indicates that epidemiological studies on large populations have not identified a threshold concentration for non-mortality endpoints below which ambient PM has no effect on health. As some health effects are expected with any increase of $PM_{2.5}$, $PM_{2.5}$ objectives have been set to consider the context of continually improving air quality and establishing guidelines at concentrations where significant adverse effects have not been demonstrated. Due to the absence of a threshold, $PM_{2.5}$ concentrations to protect all individuals from all possible health outcomes cannot be derived (WHO, 2006), however, WHO (2006) indicates that the measureable health effects (i.e., morbidity endpoints) at ambient concentrations of 11 µg/m³ are similar to those observed at background concentrations of 3 µg/m³ to 5 µg/m³, and therefore have recommended a ambient air guideline for $PM_{2.5}$ of 10 µg/m³.

In addition to the above, consideration has also been given to WHO's recent classification of outdoor air pollution as a Group 1 carcinogen. The WHO's International Agency for Research on Cancer (IARC) issued a press release (available at: (information available at: http://www.iarc.fr/en/media-centre/pr/2013/pdfs/pr221_E.pdf) on October 17, 2013, announcing that they had classified outdoor air pollution as a Group 1 carcinogen. Additionally, a summary evaluation of IARC's findings was published in The Lancet Oncology (available on-line at http://www.thelancet.com/journals/lanonc/article/PIIS1470-2045%2813%2970487-X/fulltext). The IARC press release indicates that their findings for outdoor air pollution will be detailed in IARC Monograph Volume 109; however, as of the date of the HHRA, the document for air pollution is not yet available, and limited information is presented in the press release and summary evaluation. Although IARC Monograph Volume 109 is not yet available, the summary evaluation presented in The Lancet Oncology indicated that virtually all of the studies where positive exposure response relationships were consistently observed were done in areas where annual average levels of $PM_{2.5}$ range from 10 to 30 µg/m^3. In comparison, the maximum predicted annual average $PM_{2.5}$

concentration (Project emissions + background) for all of the thousands of receptors evaluated in the AQA (Levelton, 2014) is 4.48 µg/m^3.

Based on the above and the information presented in Appendix IV, no measureable health risks are anticipated for $PM_{2.5}$ concentrations below the WHO (2006) guideline of 10 µg/m³. Given that the maximum predicted annual average $PM_{2.5}$ concentrations + background for the Project are less than the Metro Vancouver and BC MoE planning goal of 6 µg/m³, as well as the HQs estimated for $PM_{2.5}$ in the subsequent sections of the report, no measureable health effects are predicted from $PM_{2.5}$ in Project emissions.

The risk estimates for $PM_{2.5}$ have been predicted using the above summarized AAQO; the estimates for the Baseline, Project and Cumulative Scenarios for the residential and industrial receptors are presented in the following sections.

6.2.2 Risk Estimates for Residential Receptors

As described throughout the HHRA, the results of the Levelton (2014) AQA indicated that residents within the community adjacent FSD (North Delta) and along a portion of the rail corridor have the potential to be exposed to the highest concentrations of predicted Project emission for any residents within the Study Area. The characterization of exposures for these two receptors addresses maximum residential exposures and is therefore protective of all residential exposures within the Study Area. Furthermore, based on the assumptions of the HHRA, including that residents are exposed to the Project COPCs for 24 hours a day, 7 days a week, 52 weeks a year for the duration of the Project, and that residents consume 100% of their produce from their backyard garden or other sources impacted by Project emissions, evaluation of the residential receptor is protective of agricultural receptors, commercial receptors and urban park receptors in the Study Area.

Potential operable exposure pathways for the residential receptors included inhalation of Project emissions, the direct soil pathways (soil ingestion, soil dermal contact and inhalation of soil particulate) and the ingestion of produce (both above and below ground).

The risk estimates for the maximum North Delta residential receptor and the maximum rail corridor residential receptor are presented below for the Baseline Scenario, Project Scenario and Cumulative Scenario.

6.2.2.1 Maximum North Delta Residential Receptor

The acute and chronic inhalation risk estimates, as well as the cancer and non-cancer risk estimates for multi-media exposures, for the maximum North Delta residential receptor are summarized in the following tables.

Table 6-2 summarizes the acute inhalation risk estimates for each of the Baseline, Project and Cumulative Scenarios for COPCs with acute inhalation TRVs; the risk estimates are also presented in Tables I-1A (Baseline Scenario), I-1B (Project Scenario) and I-1E (Cumulative Scenario) in Appendix I. The estimate acute inhalation risks have been compared to a target risk level of an HQ $\leq$ 1.0.

Table 6-2: Risk Estimates for Acute Inhalation Exposures – Maximum North Delta Residential Receptor (Toddler and Adult)

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Metals			
Arsenic	3.6E-02	1.6E-02	5.3E-02
Boron	ND	5.5E-05	NC
Cadmium	6.3E-01	7.0E-03	6.4E-01
Copper	1.1E-03	1.3E-04	1.2E-03
Manganese	1.9E-01	2.6E-01	4.4E-01
Mercury	ND	4.5E-04	NC
Nickel	6.9E-02	4.5E-02	1.1E-01
Vanadium	8.9E-04	1.0E-03	1.9E-03
VOCs			
Acetaldehyde	7.1E-03	2.0E-03	9.1E-03
Acrolein[a]	1.8E-02	1.4E-02	3.2E-02
Acrolein[b]	8.0E-02	6.0E-02	1.4E-01
Benzene	3.0E-02	2.3E-04	3.0E-02
1,3-Butadiene	1.6E-03	1.1E-04	1.7E-03
Ethylbenzene	2.2E-04	2.8E-06	2.2E-04
Formaldehyde	8.8E-02	2.3E-02	8.8E-02
Toluene	1.8E-03	1.9E-05	1.8E-03
Styrene	1.1E-03	8.4E-07	1.1E-03
Xylenes	2.1E-03	7.3E-06	2.1E-03
Dust Palliatives Chemical Constituents			
Epichlorohydrin	ND	1.8E-08	NC
Others			
Sulfate	6.1E-02	9.0E-03	7.0E-02

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Criteria Air Contaminants			
CO (1 hour)	4.3E-02	5.3E-03	4.8E-02
CO (8 hour)	1.0E-01	8.7E-03	1.1E-01
NO_2 (1 hour)	3.3E-01	****	5.0E-01
SO_2 (1 hour)	6.2E-02	8.0E-04	6.3E-02
SO_2 (24 hour)	9.0E-01	4.0E-03	9.0E-01
$PM_{2.5}$ (24 hour)	4.8E-01	2.0E-01	6.8E-01
PM_{10} (24 hour)	5.4E-01	7.8E-02	6.2E-01
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[a]	1.1E-01	3.7E-02	1.5E-01
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[b]	1.7E-01	8.4E-02	2.5E-01
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)** [a]	1.1E-01	3.7E-02	1.4E-01
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)** [b]	1.7E-01	8.3E-02	2.5E-01
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**	**1.2E+00**	4.3E-02	**1.2E+00**

Notes:
[a] – based on the TCEQ 1 hour ReV for acrolein
[b] – based on the OEHHA 1 hour REL for acrolein
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
** - no baseline air data were available for boron and epichlorohydrin; as discussed in previous sections of the report, due to a lack of sources, baseline air concentrations are not expected to appreciably contribute to exposures
**** - the Ambient Ratio Method (ARM) used by Levelton (2014) to predict the NO_2 emissions from the Project includes the background concentration. On this basis, COPCs from the Project alone could not be estimated; however, the HQ for the Cumulative Scenario includes Project + Background.
BOLD: indicates risks in excess of the target risk level of 1E+00 for acute exposures

As presented in Table 6-2, the risk estimates for the maximum North Delta residential receptor's acute exposures to the individual COPCs, as well as for the COPCs identified as nasal and ocular irritants, for each of the

Baseline, Project and Cumulative Scenarios, were below the target HQ of 1.0 for acute exposures. On this basis, unacceptable health risks are not predicted for acute exposures to these COPCs from Project emissions.

The maximum mixture HQ estimated for the COPCs identified as respiratory irritants exceeded the target risk level of 1.0 for the Baseline (HQ = 1.2) and Cumulative (HQ = 1.2) Scenarios. The HQ for the Cumulative Scenario (Project + Background) is equivalent to the HQ for the Baseline Scenario, with negligible contribution from the Project (3.5%). In addition, the HQs have been estimated using the maximum acute concentration, which would only occur for once a year, and therefore, HQs in excess of 1.0 would occur infrequently. On this basis, no unacceptable risks from Project emissions are predicted for the mixture of COPCs identified as being respiratory irritants.

Chronic inhalation risk estimates for the maximum North Delta residential receptor's (toddler and adult) exposures to the non-carcinogenic gaseous COPCs are presented in Table 6-3; the risk estimates have been compared to the Health Canada negligible risk level of 0.2 for all COPCs with the exception of the CACs and the irritants (i.e., formaldehyde), which were compared to a target HQ of 1.0. The risk estimates for each of the scenarios are also presented in Table I-2A and I-2B in Appendix I. The risks associated with chronic inhalation exposures to the COPC mixtures (i.e., the COPCs identified as having the potential to have additive effects) are presented in Table 6-3.

Table 6-3: Non-Cancer Risk Estimates for Chronic Inhalation Exposures to Gaseous COPCs – Maximum North Delta Residential Receptor (Toddler and Adult)

Chemical	Baseline Hazard Quotient	Project Hazard Quotient	Cumulative Hazard Quotient
VOCs			
Acetaldehyde	8.6E-03	1.2E-05	8.6E-03
Acrolein[a]	7.4E-02	1.3E-04	7.4E-02
Acrolein[b]	**5.7E-01**	9.8E-04	**5.7E-01**
Benzene	1.9E-02	2.8E-05	1.9E-02
1,3-Butadiene	**3.2E-01**	1.5E-04	**3.2E-01**
Ethylbenzene	1.4E-03	2.8E-06	1.4E-03
Ethylene*	--	--	--
Formaldehyde	1.8E-01	2.1E-04	1.8E-01
Hexachlorobenzene	ND	8.4E-11	NC
n-Hexane	7.4E-04	5.9E-07	7.4E-04
Propionaldehyde	ND	2.7E-04	NC
Propylene (1-Propene)	1.4E-04	7.0E-08	1.4E-04
Toluene	1.0E-03	1.5E-06	1.0E-03
2,2,4-Trimethylpentane*	--	--	--

Chemical	Baseline Hazard Quotient	Project Hazard Quotient	Cumulative Hazard Quotient
Styrene	1.5E-03	3.7E-06	1.5E-03
Xylenes	6.7E-03	4.2E-06	6.7E-03
Criteria Air Contaminants			
NO_2	6.8E-01	1.3E-01	8.0E-01
SO_2	1.6E-01	1.2E-04	1.6E-01
$PM_{2.5}$ (based on AAQO of 8 µg/m^3)	5.5E-01	2.5E-02	5.8E-01
$PM_{2.5}$ (based on planning AAQO of 6 µg/m^3)	7.3E-01	3.3E-02	7.7E-01
PM_{10}	6.0E-01	2.0E-02	6.2E-01
DPM	1.6E-01	2.6E-02	1.9E-01

Notes:
[a] – based on the TCEQ 1 hour ReV for acrolein
[b] – based on the OEHHA 1 hour REL for acrolein
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
* - risks could not be predicted for ethylene and 2,2,4-trimethylpentane as no TRVs were identified for these COPCs
BOLD: indicates risks in excess of the Health Canada negligible risk level of 0.2

As presented in Table 6-3, the non-carcinogenic risks associated with chronic inhalation exposures to VOCs from Project emissions are less than the Health Canada negligible risk level of 0.2 for the Baseline, Project and Cumulative Scenarios (i.e., risks are acceptable), with the exception of the HQs for acrolein (when the OEHHA chronic REL is used) and 1,3-butadiene for the Baseline and the Cumulative Scenarios. The HQs for these COPCs for the Cumulative Scenario (Baseline + Project) are equivalent to the HQs for the Baseline Scenario, with negligible contribution from the Project emissions (0.17% and 0.05%, respectively). Therefore, no unacceptable risks are predicted for the chronic inhalation of these VOCs in Project emissions. It is noted that the HQ estimated for acrolein using the TCEQ chronic ReV (which is considered to be more robust) is less than the Health Canada negligible risk level of 0.2.

In addition, the HQs for the CACs were less than the target HQ of 1.0 for all three scenarios, and therefore, no unacceptable non-cancer risks are predicted for the chronic inhalation of the individual CACs in Project emissions.

Chronic inhalation risks for the maximum North Delta residential receptor's (adult) exposure to the carcinogenic gaseous COPCs are presented in Table 6-4 and in Table I-4B, in Appendix I. The estimated ILCRs have been compared to the Health Canada negligible risk level of 1E-05. The risks associated with chronic inhalation exposures to the COPC mixtures (i.e., COPCs with additive effects) are presented in Table 6-8.

Table 6-4: Cancer Risk Estimates for Chronic Exposures to Gaseous COPCs – Maximum North Delta Residential Receptor (Adult)

Chemical	Project Scenario ILCR
VOCs	
Acetaldehyde	3.4E-10
Benzene	3.4E-10
1,3-Butadiene	2.3E-10
Hexachlorobenzene	3.9E-14
Criteria Air Contaminants	
DPM	4.9E-06

Notes:
ILCR – Incremental lifetime cancer risk

The ILCR estimates (cancer risks from air concentrations above background concentrations) associated with chronic inhalation exposures to the gaseous COPCs were less than the Health Canada negligible risk level of 1E-05. On this basis, no unacceptable cancer risks are predicted for the Maximum North Delta Residential Receptor exposed to the above carcinogenic COPCs in Project emissions.

As described in earlier sections of the report, the non-gaseous COPCs were retained for evaluation in the multi-media assessment. The non-cancer risk estimates for a toddler residential receptor (maximum North Delta residential receptor) for their multi-media exposure to the Project COPCs are presented in Table 6-5; the risk estimates for each of the Scenarios are also presented in Appendix I, Tables I-3A, I-3B and I-3C. The estimated HQs have been compared to the Health Canada negligible risk level of 0.2.

Table 6-5: Chronic Risks for Multi-Pathway Exposures, Maximum North Delta Residential Receptor (Toddler)

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Carcinogenic PAHs									
Benzo(a)pyrene	1.3E-05	3.8E-04	4.0E-04	6.4E-06	4.5E-07	6.9E-06	2.0E-05	3.8E-04	4.0E-04
Benzo(a)anthracene	6.7E-06	3.0E-04	3.1E-04	2.1E-07	5.3E-07	7.3E-07	6.9E-06	3.0E-04	3.1E-04
Benzo(b)fluoranthene	1.2E-05	6.6E-04	6.7E-04	4.4E-06	2.0E-07	4.6E-06	1.6E-05	6.6E-04	6.8E-04
Benzo(g,h,i)perylene	NA	1.2E-04	1.2E-04	3.4E-07	1.1E-07	4.5E-07	NA	1.2E-04	1.2E-04
Benzo(k)fluoranthene	5.0E-06	1.9E-04	1.9E-04	9.4E-07	4.5E-07	1.4E-06	6.0E-06	1.9E-04	1.9E-04
Chrysene	NA	4.3E-04	4.3E-04	1.9E-06	8.5E-07	2.7E-06	NA	4.3E-04	4.3E-04
Dibenzo(a,h)anthracene	NA	2.6E-05	2.6E-05	2.6E-07	7.6E-08	3.4E-07	NA	2.6E-05	2.6E-05
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	6.5E-06	4.8E-07	7.0E-06	9.2E-05	1.5E-03	1.6E-03

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Indeno(1,2,3-cd)pyrene	3.2E-05	1.2E-04	1.5E-04	9.1E-07	2.6E-07	1.2E-06	3.3E-05	1.2E-04	1.5E-04
Phenanthrene	2.7E-04	2.9E-03	3.1E-03	8.7E-06	5.9E-08	8.8E-06	2.8E-04	2.9E-03	3.1E-03
Carcinogenic PAH Mixture	4.2E-04	6.6E-03	3.9E-03	3.1E-05	3.5E-06	2.5E-05	4.5E-04	6.6E-03	7.0E-03
Non-Carcinogenic PAHs									
Acenaphthene	1.8E-05	1.1E-04	1.3E-04	9.7E-07	1.3E-08	9.8E-07	1.9E-05	1.1E-04	1.3E-04
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.7E-06	4.5E-05	4.7E-05	7.0E-07	2.5E-09	7.1E-07	3.4E-06	4.5E-05	4.8E-05
Fluorene	6.4E-05	2.4E-04	3.0E-04	9.6E-07	1.0E-08	9.7E-07	6.5E-05	2.4E-04	3.0E-04
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	6.5E-06	4.8E-07	7.0E-06	9.2E-05	1.5E-03	1.6E-03
Naphthalene	1.5E-02	7.5E-04	1.6E-02	1.8E-05	5.5E-08	1.8E-05	1.5E-02	7.5E-04	1.6E-02
2-Methylnaphthalene	NA	1.8E-03	1.8E-03	2.6E-06	2.7E-08	2.6E-06	NC	1.8E-03	1.8E-03
Pyrene	8.2E-05	2.3E-03	2.3E-03	1.1E-05	1.1E-06	1.2E-05	9.3E-05	2.3E-03	2.4E-03
Non-Carcinogenic PAH Mixture	1.5E-02	6.7E-03	2.2E-02	4.1E-05	1.7E-06	4.3E-05	1.5E-02	6.7E-03	2.2E-02
Metals & Metalloids									
Aluminum	2.5E-07	**5.2E-01**	**5.2E-01**	1.9E-04	1.3E-03	1.5E-03	1.9E-04	**5.2E-01**	**5.2E-01**
Antimony	NA	5.4E-02	5.4E-02	6.8E-06	1.6E-04	1.7E-04	NC	5.4E-02	5.4E-02
Arsenic	4.9E-04	**1.4E+00**	**1.4E+00**	4.1E-04	4.6E-03	5.0E-03	9.0E-04	**1.4E+00**	**1.4E+00**
Barium	3.6E-03	8.0E-02	8.3E-02	1.1E-01	6.5E-03	1.2E-01	1.1E-01	8.6E-02	2.0E-01
Beryllium	4.3E-05	2.5E-03	2.6E-03	3.5E-03	3.5E-05	3.5E-03	3.5E-03	2.6E-03	6.1E-03
Boron	NA	NA	NA	2.9E-06	9.6E-02	9.6E-02	NC	NC	NC
Cadmium	1.7E-02	**2.1E-01**	**2.3E-01**	1.2E-03	1.6E-03	2.8E-03	1.8E-02	**2.1E-01**	**2.3E-01**
Chromium III	1.3E-04	3.0E-04	4.2E-04	3.0E-05	1.8E-08	3.0E-05	1.6E-04	3.0E-04	4.5E-04
Chromium VI	6.3E-03	3.0E-03	9.3E-03	4.7E-06	8.8E-04	8.8E-04	6.3E-03	3.9E-03	1.0E-02
Cobalt	2.6E-04	2.7E-02	2.7E-02	6.3E-04	7.1E-05	7.0E-04	8.9E-04	2.7E-02	2.8E-02
Copper	3.4E-03	1.7E-01	1.7E-01	6.5E-04	1.4E-03	2.0E-03	4.0E-03	1.7E-01	1.8E-01
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	3.4E-05	**2.7E-01**	**2.7E-01**	7.9E-05	5.6E-04	6.4E-04	1.1E-04	**2.7E-01**	**2.7E-01**
Lanthanum	ND	ND	ND	1.5E-08	1.4E-09	1.6E-08	NC	NC	NC
Lead	2.1E-02	**6.7E-01**	**6.9E-01**	8.1E-04	1.0E-03	1.8E-03	2.2E-02	**6.7E-01**	**6.9E-01**
Manganese	5.4E-02	**1.0E+00**	**1.1E+00**	1.9E-02	8.6E-04	2.0E-02	7.3E-02	**1.0E+00**	**1.1E+00**
Mercury	1.2E-02	**2.4E-01**	**2.5E-01**	1.9E-05	5.7E-03	5.7E-03	1.2E-02	**2.4E-01**	**2.5E-01**
Molybdenum	1.9E-05	1.9E-02	1.9E-02	2.3E-06	1.3E-04	1.4E-04	2.1E-05	1.9E-02	1.9E-02

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Nickel	**4.0E-01**	1.9E-01	**5.9E-01**	5.7E-02	5.7E-04	5.8E-02	**4.6E-01**	1.9E-01	**6.5E-01**
Selenium	9.0E-06	2.1E-03	2.1E-03	1.6E-06	7.0E-05	7.2E-05	1.1E-05	2.2E-03	2.2E-03
Strontium	5.5E-07	**4.4E-01**	**4.4E-01**	9.3E-06	2.2E-02	2.2E-02	9.8E-06	**4.6E-01**	**4.6E-01**
Tin	1.0E-07	5.4E-05	5.4E-05	4.2E-09	9.1E-08	9.5E-08	1.0E-07	5.4E-05	5.4E-05
Titanium	NA	3.4E-03	3.4E-03	1.3E-06	1.1E-05	1.3E-05	NC	3.5E-03	3.5E-03
Uranium	NA	2.3E-02	2.3E-02	4.4E-04	1.7E-04	6.1E-04	NC	2.3E-02	2.4E-02
Vanadium	2.6E-02	4.2E-02	6.8E-02	5.4E-03	1.5E-04	5.5E-03	3.1E-02	4.2E-02	7.3E-02
Zinc	1.2E-05	**2.6E-01**	**2.6E-01**	7.1E-07	6.5E-04	6.5E-04	1.3E-05	**2.6E-01**	**2.6E-01**
Volatile Organic Compounds									
Hexachlorobenzene (total)	NA	NA	NA	2.5E-09	2.6E-10	3.0E-09	NC	NC	NC
Dust Palliatives Constituents									
Epichlorohydrin[a]	NA	NA	NA	1.3E-06	4.5E-08	1.3E-06	NC	NC	NC
Others									
PCBs	ND	ND	ND	1.8E-03	3.8E-04	2.2E-03	NC	NC	NC
Sulfate	ND	ND	ND	NA	NA	NA	NA	NA	NA

Notes:
NA – Not applicable; HQ could not be estimated based on a lack of TRVs for COPC
ND – no baseline data available for COPC
NC – not calculated based on a lack of baseline data for COPC
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

Table 6-5 presents the non-cancer risk estimates for the residential toddler (maximum North Delta residential receptor) exposed to the Project non-gaseous COPCs via inhalation, the direct soil pathways and ingestion of produce. Hazard quotients for all COPCs for the Project Scenario were less than the than the Health Canada negligible risk level of 0.2, indicating that there are no unacceptable risks associated with exposures to the Project emissions alone. HQs greater than 0.2 were predicted for the Cumulative Scenario (i.e., Baseline + Project) for aluminum, arsenic, cadmium, iron, lead, manganese, mercury, nickel, strontium and zinc. With the exception of nickel and strontium, the Cumulative Scearnio HQs are equivalent to the Baseline Scenario HQs, indicating negligible contribution from the Project. The HQs for nickel and strontium are further discussed below.

- For nickel, the Baseline Scenario HQ (all routes) is 0.59 and the Project Scenario HQ (all routes) is 0.058, resulting in a Cumulative Scenario HQ of 0.65. Of the exposure pathways considered, the inhalation route contributed the most significantly to the overall HQs (0.4 for the Baseline Scenario and 0.06 for the Project Scenario), resulting in a Cumulative Scenario HQ for the inhalation route of 0.46. If the inhalation route is not considered, the Cumulative Scenario HQ for the soil and vegetation pathways of 0.2 is equivalent to the Baseline Scenario HQ for the soil and vegetation pathways of 0.2 (i.e., negligible contribution from the Project), and is equivalent to the Health Canada negligible risk level.
- In the case of nickel inhalation, the relative contribution from the Project to the overall HQ is 10%, with 90% of the estimate associated with background exposures. A target HQ of 1.0 for inhalation exposures to nickel is appropriate as the mechanism of toxicity is specific to the inhalation route of exposure (i.e., the mechanism of toxicity differs for oral exposures). Furthermore, a conservative approach was used in the characterization of risks associated with nickel. The toxicity of nickel is highly dependent on the form of the metal, and Health Canada recommends different TRVs for the various forms of nickel. Because the form of the nickel in the coal and in the combustion emissions was not known, the most conservative of the available inhalation TRVs, a Tolerable Concentration (TC) for nickel sulphate, was used in the HHRA. This approach has likely overestimated risks associated with Project emissions as although nickel sulphate has the potential to be present in combustion emissions, it is unlikely to be present in unburnt coal. The relative contributions of combustion emissions and coal dust to the air EPC for the maximum North Delta residential receptor are 5E-08 µg/m3 and 2E-04 µg/m3, respectively (i.e., exposures are largely from coal). Based on the above, and given that the HQ is well below 1.0, unacceptable risks associated with inhalation exposures to nickel are not anticipated.
- For strontium, the Baseline Scenario HQ is 0.44, while the Project Scenario HQ is 0.02, for a Cumulative Scenario HQ of 0.46. The relative contribution of the Project to the overall HQ is 4% (96% from background). On this basis, and considering the conservatism in the estimated exposure and associated risk, the contribution from the Project is considered to be negligible.

Based on the above, no unacceptable risks are predicted for the toddler maximum North Delta residential receptor exposed to the COPCs included in Table 6-5 in project emissions.

It is noted that no baseline data were available for select COPCs, including baseline air data for select PAHs (e.g., 2-methylnaphthalene) and metals (e.g. antimony, boron, uranium), hexachlorobenzene, PCBs and epichlorohydrin, and baseline soil data for select COPCs including select metals (boron, indium and lanthanum),

PCBs and epichlorohydrin. This applies to the results of the multi-media results for all receptors (residential and industrial), and the uncertainty associated with the lack of this data is discussed in Section 7.

Table 6-6 presents the non-cancer risk estimates for an adult residential receptor (maximum North Delta residential receptor) for their multi-media exposure to the Project COPCs. The risk estimates for the various exposure pathways for each of the Baseline, Project and Cumulative Scenarios are presented in Appendix I, Tables I-4A, I-4B and I-4C, respectively. The risk estimates have been compared to the Health Canada negligible risk level of 0.2 for non-carcinogens.

Table 6-6: Chronic Risks for Multi-Pathway Exposures, Maximum North Delta Residential Receptor (Adult)

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Carcinogenic PAHs									
Benzo(a)pyrene	6.3E-06	1.5E-04	1.6E-04	3.0E-06	1.8E-07	3.2E-06	9.3E-06	1.5E-04	1.6E-04
Benzo(a)anthracene	3.1E-06	1.1E-04	1.1E-04	9.6E-08	1.9E-07	2.9E-07	3.2E-06	1.1E-04	1.1E-04
Benzo(b)fluoranthene	5.5E-06	2.6E-04	2.6E-04	2.1E-06	8.2E-08	2.1E-06	7.5E-06	2.6E-04	2.6E-04
Benzo(g,h,i)perylene	NA	3.8E-05	3.8E-05	1.6E-07	3.5E-08	1.9E-07	NA	3.8E-05	3.8E-05
Benzo(k)fluoranthene	2.3E-06	6.8E-05	7.0E-05	4.4E-07	1.7E-07	6.0E-07	2.8E-06	6.8E-05	7.1E-05
Chrysene	NA	1.6E-04	1.6E-04	8.7E-07	3.3E-07	1.2E-06	NA	1.6E-04	1.6E-04
Dibenzo(a,h)anthracene	NA	8.0E-06	8.0E-06	1.2E-07	2.4E-08	1.5E-07	NA	8.0E-06	8.0E-06
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	3.0E-06	2.0E-07	3.2E-06	4.3E-05	6.1E-04	6.5E-04
Indeno(1,2,3-cd)pyrene	1.5E-05	3.8E-05	5.2E-05	4.2E-07	8.3E-08	5.1E-07	1.5E-05	3.8E-05	5.3E-05
Phenanthrene	1.3E-04	1.2E-03	1.4E-03	4.1E-06	2.7E-08	4.1E-06	1.3E-04	1.2E-03	1.4E-03
Carcinogenic PAH Mixture	2.0E-04	2.7E-03	1.5E-03	1.4E-05	1.3E-06	1.1E-05	2.1E-04	2.7E-03	2.9E-03
Non-Carcinogenic PAHs									
Acenaphthene	8.6E-06	4.8E-05	5.7E-05	4.5E-07	6.1E-09	4.6E-07	9.1E-06	4.8E-05	5.8E-05
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	1.3E-06	1.9E-05	2.0E-05	3.3E-07	1.1E-09	3.3E-07	1.6E-06	1.9E-05	2.1E-05
Fluorene	3.0E-05	1.0E-04	1.3E-04	4.5E-07	4.7E-09	4.5E-07	3.0E-05	1.0E-04	1.3E-04
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	3.0E-06	2.0E-07	3.2E-06	4.3E-05	6.1E-04	6.5E-04
Naphthalene	1.5E-02	3.3E-04	1.5E-02	1.8E-05	2.6E-08	1.8E-05	1.5E-02	3.3E-04	1.5E-02
2-Methylnaphthalene	NA	7.9E-04	7.9E-04	1.2E-06	1.3E-08	1.2E-06	NC	7.9E-04	7.9E-04
Pyrene	3.8E-05	9.6E-04	9.9E-04	5.2E-06	4.9E-07	5.6E-06	4.4E-05	9.6E-04	1.0E-03
Non-Carcinogenic PAH Mixture	1.5E-02	2.9E-03	1.8E-02	2.9E-05	7.4E-07	3.0E-05	1.5E-02	2.9E-03	1.8E-02
Metals & Metalloids									
Aluminum	1.2E-07	**1.5E-01**	**1.5E-01**	8.9E-05	3.9E-04	4.7E-04	8.9E-05	**1.5E-01**	**1.5E-01**
Antimony	NA	2.4E-02	2.4E-02	3.2E-06	7.2E-05	7.5E-05	NC	2.4E-02	2.4E-02
Arsenic	4.9E-04	**5.7E-01**	**5.7E-01**	4.1E-04	1.9E-03	2.3E-03	9.0E-04	**5.7E-01**	**5.7E-01**
Barium	3.6E-03	3.5E-02	3.8E-02	1.1E-01	2.8E-03	1.1E-01	1.1E-01	3.7E-02	1.5E-01
Beryllium	4.3E-05	9.0E-04	9.4E-04	3.5E-03	1.2E-05	3.5E-03	3.5E-03	9.1E-04	4.4E-03
Boron	NA	NA	NA	2.9E-06	4.2E-02	4.2E-02	NC	NC	NC

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Cadmium	1.7E-02	9.1E-02	1.1E-01	1.2E-03	7.1E-04	1.9E-03	1.8E-02	9.2E-02	1.1E-01
Chromium III	1.3E-04	1.0E-04	2.3E-04	3.0E-05	6.1E-09	3.0E-05	1.6E-04	1.0E-04	2.6E-04
Chromium VI	6.3E-03	1.0E-03	7.3E-03	4.7E-06	2.9E-04	3.0E-04	6.3E-03	1.3E-03	7.6E-03
Cobalt	2.6E-04	1.1E-02	1.1E-02	6.3E-04	2.8E-05	6.6E-04	8.9E-04	1.1E-02	1.2E-02
Copper	3.4E-03	4.8E-02	5.2E-02	6.5E-04	3.9E-04	1.0E-03	4.0E-03	4.9E-02	5.3E-02
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	1.6E-05	7.7E-02	7.7E-02	3.7E-05	1.6E-04	2.0E-04	5.3E-05	7.7E-02	7.7E-02
Lanthanum	NA	NA	NA	7.0E-09	6.7E-10	7.6E-09	NC	NC	NC
Lead	2.1E-02	8.5E-02	1.1E-01	8.1E-04	1.3E-04	9.5E-04	2.2E-02	8.5E-02	1.1E-01
Manganese	5.4E-02	**4.0E-01**	**4.5E-01**	1.9E-02	3.3E-04	2.0E-02	7.3E-02	**4.0E-01**	**4.7E-01**
Mercury	1.2E-02	1.0E-01	1.2E-01	1.9E-05	2.5E-03	2.5E-03	1.2E-02	1.1E-01	1.2E-01
Molybdenum	1.9E-05	6.7E-03	6.7E-03	2.3E-06	4.8E-05	5.0E-05	2.1E-05	6.7E-03	6.7E-03
Nickel	**4.0E-01**	8.4E-02	**4.8E-01**	5.7E-02	2.5E-04	5.8E-02	**4.6E-01**	8.4E-02	**5.4E-01**
Selenium	9.0E-06	9.2E-04	9.3E-04	1.6E-06	3.1E-05	3.2E-05	1.1E-05	9.5E-04	9.6E-04
Strontium	2.6E-07	1.9E-01	1.9E-01	4.3E-06	9.7E-03	9.7E-03	4.6E-06	2.0E-01	2.0E-01
Tin	4.7E-08	2.2E-05	2.2E-05	2.0E-09	3.7E-08	3.9E-08	4.9E-08	2.2E-05	2.2E-05
Titanium	1.6E-05	7.7E-02	7.7E-02	3.7E-05	1.6E-04	2.0E-04	5.3E-05	7.7E-02	7.7E-02
Uranium	4.7E-08	2.2E-05	2.2E-05	2.0E-09	3.7E-08	3.9E-08	4.9E-08	2.2E-05	2.2E-05
Vanadium	NA	8.0E-03	8.0E-03	4.4E-04	5.8E-05	5.0E-04	NC	8.1E-03	8.5E-03
Zinc	2.6E-02	1.3E-02	3.9E-02	5.4E-03	4.8E-05	5.4E-03	3.1E-02	1.3E-02	4.5E-02
Volatile Organic Compounds									
Hexachlorobenzene	NA	NA	NA	5.9E-10	1.2E-10	7.1E-10	NC	NC	NC
Dust Palliatives Constituents									
Epichlorohydrin[a]	NA	NA	NA	1.3E-06	2.1E-08	1.3E-06	NC	NC	NC
Others									
PCBs	NA	NA	NA	8.6E-04	1.8E-04	1.0E-03	NC	NC	NC
Sulfate	NA	NA	NA	NA	NA	NA	NA	NA	NA

Notes:
NA – Not applicable; HQ could not be estimated based on a lack of TRVs for COPC
ND – no baseline data available for COPC
NC – not calculated based on a lack of baseline data for COPC
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

Table 6-7 presents the cancer risks for an adult residential receptor (maximum North Delta residential receptor) for their multi-media exposure to the Project COPCs; the risk estimates are also presented in Appendix I, Table I-4B. The ILCR estimates have been compared to the Health Canada negligible risk level of 1E-05 for carcinogens.

Table 6-7: Cancer Risks for Multi-Pathway Exposures, Maximum North Delta Residential Receptor (Adult)

Chemical	Project Scenario
	ILCR
Carcinogenic PAHs	
Benzo(a)pyrene	1.4E-09
Benzo(a)anthracene	1.3E-08
Benzo(b)fluoranthene	6.7E-10
Benzo(g,h,i)perylene	2.5E-11
Benzo(k)fluoranthene	1.2E-09
Chrysene	2.3E-10
Dibenzo(a,h)anthracene	1.7E-09
Fluoranthene	2.1E-11
Indeno(1,2,3-cd)pyrene	5.9E-10
Phenanthrene	3.9E-12
Carcinogenic PAH Mixture	1.9E-08
Metals & Metalloids	
Arsenic	1.3E-06
Beryllium	2.1E-08
Cadmium	1.4E-08
Chromium VI	1.2E-07
Nickel	3.3E-08
Volatile Organic Compounds	
Hexachlorobenzene	9.3E-14
Dust Palliatives Constituents	
Epichlorohydrin	1.4E-12

As presented in Table 6-6 and 6-7, the non-cancer and cancer risk estimates for the adult maximum North Delta residential receptor were less than the Health Canada negligible risk levels of 0.2 and 1E-05, respectively, with the exception of the Baseline and Cumulative Scenario HQs for aluminum, arsenic, manganese and nickel. The HQs for these COPCs are discussed below.

- For arsenic and aluminum, the Cumulative Scenario HQ is equivalent to the Baseline Scenario HQ; therefore, the Project contribution for these COPCs is considered to be negligible.

- For manganese, the Baseline Scenario HQ (all routes) is 0.45 and the Project Scenario HQ (all routes) is 0.02, resulting in a Cumulative Scenario HQ of 0.47. The contribution from the Project is considered to be negligible.
- Similar to the above discussion for nickel for the toddler receptor, the inhalation route contributes the most significantly to the overall HQs for the various scenarios, and if the inhalation route is not considered, the Cumulative Scenario HQ for the soil and vegetation pathways of 0.084 is equivalent to the Baseline Scenario HQ (i.e., negligible contribution from the Project) and is less than the Health Canada negligible risk level of 0.2.
- The HQ of 0.46 for the inhalation of nickel for the Cumulative Scenario was greater than 0.2 (0.4 from background and 0.06 from Project emissions). As discussed above for the toddler, a target risk level of 1.0 (versus 0.2) is appropriate for the evaluation of risks associated with the inhalation of nickel based on the mechanism of toxicity specific to this exposure route (i.e., risks from non-inhalation routes would not be additive). In addition, an HQ greater than 0.2 was predicted based on background concentrations, with minimal contribution from the Project emissions. On this basis, unacceptable risks associated with inhalation exposures to nickel are not anticipated.

Based on the above, no unacceptable risks are predicted for the adult maximum North Delta residential receptor exposed to the COPCs in project emissions.

Table 6-8 presents the total hazard quotients and ILCRs estimated for the COPCs identified as being additive. The non-cancer and cancer risk estimates have been compared the Health Canada negligible risk levels of 0.2 and 1E-05, respectively.

Table 6-8: Risk Estimates for Chronic Exposures to COPC Mixtures, Maximum North Delta Residential Receptor (Toddler and Adult)

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
Chronic Inhalation	Nasal irritation/nasal lesions	Acetaldehyde, boron, acrolein, epichlorohydrin, formaldehyde, naphthalene, nickel, propionaldehyde	HQ[a,b]: 6.7E-01 HQ[a,c]: **1.2E+00**	HQ[b]: 5.8E-02 HQ[c]: 5.9E-02	HQ[b]: **7.3E-01** HQ[c]: **1.2E+00**
	Respiratory irritation	Chromium III, chromium VI, cobalt, copper, formaldehyde, NO_2, SO_2, propylene, vanadium	HQ: **1.0E+00**	HQ: 1.6E-01	HQ: **1.2E+00**
	Lung cancer	Arsenic, beryllium, cadmium, chromium VI, nickel, PAHs	NA	ILCR: 1.6E-06	NA

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
	Leukemia	Benzene, 1,3-butadiene	NA	ILCR: 5.7E-10	NA
	Developmental effects	Styrene, xylenes	HQ: 8.2E-03	HQ: 7.9E-06	HQ: 8.2E-03
	Renal toxicity	Cadmium, ethylbenzene, uranium	HQ[a]: 1.8E-02	HQ: 1.6E-03	HQ: 2.0E-02
	Neurotoxicity	n-Hexane, manganese, mercury, toluene	HQ: 6.8E-02	HQ: 1.9E-02	HQ: 8.7E-02
Chronic Oral	Renal toxicity	Barium, pyrene, mercury, uranium	HQ Adult: 1.5E-01	HQ Adult: 5.3E-03	HQ Adult: 1.5E-01
			HQ Toddler: **3.4E-01**	HQ Toddler: 1.2E-02	HQ Toddler: **3.5E-01**
	Hepatotoxicity	Acenaphthene, copper	HQ Adult: 4.8E-02	HQ Adult: 3.9E-04	HQ Adult: 4.8E-02
			HQ Toddler: 1.7E-01	HQ Toddler: 1.4E-03	HQ Toddler: 1.7E-01
	Reproductive Toxicity	Molybdenum, nickel	HQ Adult: 9.1E-02	HQ Adult: 3.1E-04	HQ Adult: 9.5E-02
			HQ Toddler: **2.1E-01**	HQ Toddler: 7.0E-04	HQ Toddler: **2.1E-01**

Notes:
[a] – no baseline air concentrations were available for boron, epichlorohydrin, propionaldehyde and uranium and therefore, HQs for the Baseline Scenario do not include these COPCs. It is noted that these COPCs are included in the estimation of the HQ for the Project Scenario
[b] – based on the TCEQ chronic ReV for acrolein
[c] – based on the OEHHA chronic REL ofr acrolein
HQ: Hazard Quotient
ILCR: Incremental Lifetime Cancer Risk
NA: not applicable; risk estimates were not predicted for the Baseline and Cumulative Scenario for carcinogens, as the cancer risks are estimates as cancer risks above background
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

As presented in Table 6-8, HQs in excess of 0.2 were predicted for the Baseline and Cumulative Scenarios for the COPC mixtures identified as nasal and respiratory irritants for inhalation exposures, as well as the COPCs mixtures identified as renal and reproductive toxicants for oral exposures (toddler only). In the case of the nasal irritants (when the OEHHA chronic inhalation REL is used for acrolein vs. the TCEQ chronic inhalation ReV) and reproductive toxicants, the HQs for the Cumulative Scenario (Baseline + Project) are equivalent to the Baseline Scenario, with negligible contribution from the Project emissions. Therefore, no unacceptable risks are predicted for the chronic inhalation of these COPC mixtures in Project emissions.

The Project emissions contribute very little to the Cumulative HQs estimated for the respiratory irritants (Baseline HQ = 1.0 and Project HQ = 0.1), for the renal toxicants (Baseline HQ = 0.35 and Project HQ = 0.012) as well as for the nasal irritants when the TCEQ chronic inhalation ReV for acrolein is used (Baseline HQ = 0.67 and Project HQ = 0.058). In addition, the exposures to the respiratory irritants, and all COPCs, were estimated using the

maximum annual average concentrations for the numerous residential receptors modelled by Levelton (2014). Furthermore, the approach assumes that the maximum concentrations occur at the same location; however, a review of Levelton (2014) indicates that the maximum concentrations occur at different receptors, and thus, the approach overestimates actual risks. For example, the maximum concentration of the metals identified as respiratory irritants (which were predicted based on PM concentrations) and NO_2 were predicted at locations approximately 1 km away from each other. Finally, although the COPCs were identified as respiratory irritants, the mechanism of toxicity for the COPCs varies, and thus, the assumption of additivity is conservative. Given the above, no unacceptable risks are predicted for the chronic inhalation of these COPCs mixtures in Project emissions.

6.2.2.1.1 Maximum North Delta Residential Receptor Conclusions

As presented above, no unacceptable risks are predicted for the maximum North Delta residential receptor (toddler and adult). As described throughout the HHRA, the approach used to estimate exposures for this receptor, and all receptors, is highly conservative and will tend to overestimate exposures and therefore associated risks. In addition, the evaluation of the maximum North Delta residential receptor is protective of agricultural receptors, commercial receptors and urban park users in the area of the Project; this will be further discussed in Sections 6.2.4 to 6.2.6.

6.2.2.2 Maximum Rail Corridor Residential Receptor

The acute and chronic inhalation risk estimates, as well as the cancer and non-cancer risk estimates for multi-media exposures, for the maximum rail corridor residential receptor (toddler and adult) are summarized in the following tables.

Table 6-9 summarizes the acute inhalation risk estimates for each of the Baseline, Project and Cumulative Scenarios for COPCs with acute inhalation TRVs; the risk estimates are also presented in Tables I-1A (Baseline Scenario), I-1C (Project Scenario) and I-1F (Cumulative Scenario) in Appendix A. It is noted that based on a lack of baseline air concentrations for select COPCs (e.g., boron, epichlorohydrin), risks could not be estimated for the cumulative scenario. This is discussed in Section 7, the Uncertainty and Sensitivity Analysis.

Table 6-9: Risk Estimates for Acute Inhalation Exposures – Maximum Rail Corridor Residential Receptor (Toddler and Adult)

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Metals			
Arsenic	3.6E-02	6.7E-03	4.3E-02
Boron	ND	2.5E-05	NC
Cadmium	6.3E-01	1.5E-02	6.5E-01
Copper	1.1E-03	1.3E-04	1.2E-03
Manganese	1.9E-01	1.2E-01	3.0E-01
Mercury	ND	2.3E-04	NC
Nickel	6.9E-02	2.0E-02	9.0E-02
Vanadium	8.9E-04	3.6E-04	1.2E-03
VOCs			
Acetaldehyde	7.1E-03	1.1E-03	8.2E-03
Acrolein	--	--	--
Benzene	4.6E-03	3.5E-05	4.6E-03
1,3-Butadiene	--	--	--
Ethylbenzene	2.2E-04	3.9E-06	2.3E-04
Formaldehyde	8.8E-02	1.4E-02	1.0E-01
Toluene	1.8E-03	2.7E-05	1.8E-03
Styrene	1.1E-03	7.6E-07	1.1E-03
Xylenes	--	--	--
Dust Palliatives Chemical Constituents			
Epichlorohydrin	ND	8.1E-09	NC
Others			
Sulfate	--	--	--
Criteria Air Contaminants			
CO (1 hour)	4.3E-02	5.8E-03	4.9E-02
CO (8 hour)	1.0E-01	1.0E-02	1.1E-01
NO_2 (1 hour)	3.3E-01	****	4.9E-01
SO_2 (1 hour)	6.2E-02	6.4E-04	6.3E-02
SO_2 (24 hour)	9.0E-01	5.0E-04	9.0E-01
$PM_{2.5}$ (24 hour)	4.8E-01	6.0E-02	5.4E-01
PM_{10} (24 hour)	5.4E-01	6.0E-02	6.0E-01

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Nasal irritants (acrolein, boron, formaldehyde, toluene, styrene)**	9.1E-02	1.4E-02	1.0E-01
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)**	8.9E-02	1.4E-02	1.0E-01
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**	**1.1E+00**	3.1E-02	**1.2E+00**

Notes:
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
** - no baseline air data were available for boron and epichlorohydrin; as discussed in previous sections of the report, due to a lack of sources, baseline air concentrations are not expected to appreciably contribute to exposures
**** - the Ambient Ratio Method (ARM) used by Levelton (2014) to predict the NO_2 emissions from the Project includes the background concentration. On this basis, COPCs from the Project alone could not be estimated; however, the HQ for the Cumulative Scenario includes Project + Background.
BOLD: indicates risks in excess of the target risk level of HQ=1.0 for acute inhalation exposures

As presented in Table 6-9, similar to the maximum North Delta residential receptor, the risk estimates for the maximum rail corridor residential receptor's acute exposures to the individual COPCs, as well as for the COPCs identified as nasal and ocular irritants, for each of the Baseline, Project and Cumulative Scenarios, were below the target hazard quotient of 1.0 for acute exposures. On this basis, unacceptable health risks are not predicted for acute exposures to these COPCs from Project emissions.

The maximum mixture HQ estimated for the COPCs identified as respiratory irritants exceeded the target risk level of 1.0 for the Baseline (HQ = 1.1) and Cumulative (HQ = 1.2) Scenarios (i.e., negligible contribution from the Project). In addition, as the HQs are estimated based on the maximum acute concentrations, HQs greater than 1.0 would occur infrequently (e.g. once a year). On this basis, no unacceptable risks from Project emissions are predicted for the mixture of COPCs identified as being respiratory irritants.

Chronic inhalation risks associated with exposures to the non-carcinogenic gaseous COPCs, compared to the Health Canada negligible risk level of 0.2, are presented in Table 6-10. The risk estimates for the Baseline, Project and Cumulative Scenario are also presented in Tables I-2A, I-2C and I-2F. The risks associated with chronic inhalation exposures to the COPC mixtures identified as having the potential to be additive are presented in Table 6-15.

Table 6-10: Non-Cancer Risk Estimates for Chronic Inhalation Exposures to Gaseous COPCs – Maximum Rail Corridor Residential Receptor (Toddler and Adult)

Chemical	Baseline Hazard Quotient	Project Hazard Quotient	Cumulative Hazard Quotient
VOCs			
Acetaldehyde	8.6E-03	1.3E-05	8.6E-03
Acrolein**	--	--	--
Benzene	1.9E-02	2.3E-05	1.9E-02
1,3-Butadiene**	--	--	--
Ethylbenzene	1.4E-03	3.2E-06	1.4E-03
Ethylene*	--	--	--
Formaldehyde	1.8E-01	2.3E-04	1.8E-01
Hexachlorobenzene**	--	--	--
n-Hexane	7.4E-04	6.2E-07	7.4E-04
Propionaldehyde	NA	3.0E-04	NC
Propylene (1-Propene)**	--	--	--
Toluene	1.0E-03	1.8E-06	1.0E-03
2,2,4-Trimethylpentane*	--	--	--
Styrene	1.5E-03	1.8E-06	1.5E-03
Xylenes	6.7E-03	4.2E-06	6.7E-03
Criteria Air Contaminants			
NO_2	6.8E-01	1.5E-01	8.2E-01
SO_2	1.6E-01	1.2E-04	1.6E-01
$PM_{2.5}$ (based on AAQO of 8 µg/m^3)	5.5E-01	5.1E-02	6.0E-01
$PM_{2.5}$ (based on planning AAQO of 6 µg/m^3)	7.3E-01	6.8E-02	8.0E-01
PM_{10}	6.0E-01	4.1E-02	6.4E-01
DPM	1.6E-01	2.8E-02	1.9E-01

Notes:
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
* - risks could not be predicted for ethylene and 2,2,4-trimethylpentane as no TRVs were identified for these COPCs
** - acrolein, 1,3-butadiene, hexachlorobenzene and propylene were not COPCs for the maximum rail corridor residential receptor as they are associated with emissions from transportation equipment at FSD

The non-carcinogenic risks associated with chronic inhalation exposures to VOCs from Project emissions are less than the Health Canada negligible risk level of 0.2 for the Baseline, Project and Cumulative Scenarios for all VOC COPCs. In addition, the HQs for the CACs were less than the target HQ of 1.0 for all three scenarios. On this

basis, no unacceptable non-cancer risks are predicted for the chronic inhalation of gaseous COPCs in Project emissions.

Chronic inhalation risks associated with exposures to the carcinogenic gaseous COPCs are presented in Table 6-11 and in Table I-2C in Appendix I. The ILCRs have been compared to the Health Canada negligible risk level of 1E-05.

Table 6-11: Cancer Risk Estimates for Chronic Exposures to Gaseous COPCs – Maximum Rail Corridor Residential Receptor (Adult)

Chemical	Project Scenario ILCR
VOCs	
Acetaldehyde	3.7E-10
Benzene	2.9E-10
1,3-Butadiene**	--
Hexachlorobenzene**	--
Criteria Air Contaminants	
DPM	5.3E-06

Notes:
ILCR – Incremental lifetime cancer risk
** - hexachlorobenzene and 1,3-butadiene were not COPCs for the maximum rail corridor residential receptor as it is associated with emissions from transportation equipment at FSD

The ILCR estimates (cancer risks above background) associated with chronic inhalation exposures to the gaseous COPCs were less than the Health Canada negligible risk level of 1E-05. On this basis, no unacceptable cancer risks are predicted for the maximum rail corridor receptor exposed to the above carcinogenic COPCs in Project emissions.

The non-gaseous COPCs were retained for evaluation in the multi-media assessment. The non-cancer risk estimates for a toddler residential receptor (maximum rail corridor residential receptor) for their multi-media exposure to the Project COPCs is presented in Table 6-12; the risk estimates for the Baseline, Project and Cumulative Scenario are also presented in Tables I-5A, I-5B and I-5C in Appendix I. The estimated HQs have been compared to the Health Canada negligible risk level of 0.2 for non-carcinogens. It is noted that select COPCs that were included for the maximum North Delta residential receptor, including indium, lanthanum, hexachlorobenzene, PCBs and sulfate, were not COPCs for the maximum rail corridor residential receptor as they were present in emissions from transportation equipment used at FSD (i.e., at the Project facility).

Table 6-12: Chronic Risks for Multi-Pathway Exposures, Maximum Rail Corridor Residential Receptor (Toddler)

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Carcinogenic PAHs									
Benzo(a)pyrene	1.3E-05	3.8E-04	4.0E-04	1.2E-06	8.8E-05	8.9E-05	1.5E-05	4.7E-04	4.9E-04
Benzo(a)anthracene	6.7E-06	3.0E-04	3.1E-04	5.0E-07	3.6E-05	3.7E-05	7.2E-06	3.4E-04	3.5E-04
Benzo(b)fluoranthene	1.2E-05	6.6E-04	6.7E-04	1.8E-06	1.3E-04	1.3E-04	1.4E-05	7.9E-04	8.0E-04
Benzo(g,h,i)perylene	NA	1.2E-04	1.2E-04	1.4E-07	9.7E-06	9.8E-06	NC	1.3E-04	1.3E-04
Benzo(k)fluoranthene	5.0E-06	1.9E-04	1.9E-04	3.8E-07	2.7E-05	2.8E-05	5.4E-06	2.1E-04	2.2E-04
Chrysene	NA	4.3E-04	4.3E-04	7.6E-07	5.4E-05	5.5E-05	NC	4.8E-04	4.8E-04
Dibenzo(a,h)anthracene	NA	2.6E-05	2.6E-05	1.0E-07	7.6E-06	7.7E-06	NC	3.4E-05	3.4E-05
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	2.7E-06	1.8E-04	1.9E-04	8.8E-05	1.7E-03	1.7E-03
Indeno(1,2,3-cd)pyrene	3.2E-05	1.2E-04	1.5E-04	3.6E-07	2.6E-05	2.7E-05	3.2E-05	1.5E-04	1.8E-04
Phenanthrene	2.7E-04	2.9E-03	3.1E-03	8.7E-06	6.0E-05	6.8E-05	2.8E-04	2.9E-03	3.2E-03
Carcinogenic PAH Mixture	4.2E-04	6.6E-03	3.9E-03	1.7E-05	6.2E-04	5.7E-04	4.4E-04	7.2E-03	7.6E-03
Non-Carcinogenic PAHs									
Acenaphthene	1.8E-05	1.1E-04	1.3E-04	4.1E-07	2.7E-05	2.7E-05	1.9E-05	1.4E-04	1.6E-04
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.7E-06	4.5E-05	4.7E-05	7.1E-08	3.0E-06	3.0E-06	2.8E-06	4.8E-05	5.0E-05
Fluorene	6.4E-05	2.4E-04	3.0E-04	5.9E-07	1.9E-05	2.0E-05	6.5E-05	2.6E-04	3.2E-04
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	2.7E-06	1.8E-04	1.9E-04	8.8E-05	1.7E-03	1.7E-03
Naphthalene	1.5E-02	7.5E-04	1.6E-02	1.7E-06	1.6E-05	1.8E-05	1.5E-02	7.7E-04	1.6E-02
2-Methylnaphthalene	NA	1.8E-03	1.8E-03	1.0E-06	7.5E-05	7.6E-05	NC	1.9E-03	1.9E-03
Pyrene	8.2E-05	2.3E-03	2.3E-03	4.6E-06	3.1E-04	3.2E-04	8.7E-05	2.6E-03	2.7E-03
Non-Carcinogenic PAH Mixture	1.5E-02	6.7E-03	2.2E-02	1.1E-05	6.4E-04	6.5E-04	1.5E-02	7.3E-03	2.3E-02
Metals & Metalloids									
Aluminum	2.5E-07	**5.2E-01**	**5.2E-01**	4.7E-04	3.6E-02	3.6E-02	4.7E-04	**5.6E-01**	**5.6E-01**
Antimony	NA	5.4E-02	5.4E-02	2.1E-06	3.9E-04	3.9E-04	NC	5.4E-02	5.4E-02
Arsenic	4.9E-04	**1.4E+00**	**1.4E+00**	1.6E-04	2.7E-02	2.7E-02	6.5E-04	**1.4E+00**	**1.4E+00**
Barium	3.6E-03	8.0E-02	8.3E-02	4.3E-02	1.8E-02	6.1E-02	4.7E-02	9.7E-02	1.4E-01
Beryllium	4.3E-05	2.5E-03	2.6E-03	1.5E-03	5.3E-04	2.0E-03	1.6E-03	3.0E-03	4.6E-03
Boron	ND	ND	ND	7.2E-06	1.5E-01	1.5E-01	NC	NC	NC
Cadmium	1.7E-02	**2.1E-01**	**2.3E-01**	1.8E-03	3.0E-03	4.8E-03	1.9E-02	**2.1E-01**	**2.3E-01**
Chromium III	1.3E-04	3.0E-04	4.2E-04	7.4E-05	3.3E-07	7.4E-05	2.0E-04	3.0E-04	5.0E-04
Chromium VI	6.3E-03	3.0E-03	9.3E-03	3.0E-06	1.6E-02	1.6E-02	6.3E-03	1.9E-02	2.6E-02
Cobalt	2.6E-04	2.7E-02	2.7E-02	1.6E-03	6.7E-04	2.2E-03	1.8E-03	2.8E-02	3.0E-02
Copper	3.4E-03	1.7E-01	1.7E-01	1.6E-03	2.7E-03	4.3E-03	5.0E-03	1.7E-01	1.8E-01

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Iron	3.4E-05	**2.7E-01**	**2.7E-01**	2.0E-04	1.5E-02	1.5E-02	2.3E-04	**2.8E-01**	**2.8E-01**
Lead	2.1E-02	**6.7E-01**	**6.9E-01**	1.9E-03	4.7E-03	6.6E-03	2.3E-02	**6.7E-01**	**7.0E-01**
Manganese	5.4E-02	**1.0E+00**	**1.1E+00**	4.7E-02	1.9E-03	4.9E-02	1.0E-01	**1.0E+00**	**1.1E+00**
Mercury	1.2E-02	**2.4E-01**	**2.5E-01**	3.4E-05	9.8E-03	9.8E-03	1.2E-02	**2.5E-01**	**2.6E-01**
Molybdenum	1.9E-05	1.9E-02	1.9E-02	5.2E-06	3.0E-04	3.0E-04	2.4E-05	1.9E-02	1.9E-02
Nickel	**4.0E-01**	1.9E-01	**5.9E-01**	1.4E-01	2.5E-03	1.4E-01	**5.4E-01**	2.0E-01	**7.4E-01**
Selenium	9.0E-06	2.1E-03	2.1E-03	4.0E-06	5.7E-04	5.8E-04	1.3E-05	2.7E-03	2.7E-03
Strontium	5.5E-07	**4.4E-01**	**4.4E-01**	2.3E-05	3.5E-02	3.5E-02	2.3E-05	**4.8E-01**	**4.8E-01**
Tin	1.0E-07	5.4E-05	5.4E-05	7.0E-09	6.4E-07	6.5E-07	1.1E-07	5.5E-05	5.5E-05
Titanium	NA	3.4E-03	3.4E-03	3.2E-06	2.5E-04	2.5E-04	NC	3.7E-03	3.7E-03
Uranium	NA	2.3E-02	2.3E-02	1.1E-03	2.9E-03	4.0E-03	NC	2.6E-02	2.7E-02
Vanadium	2.6E-02	4.2E-02	6.8E-02	1.3E-02	3.4E-03	1.6E-02	3.9E-02	4.5E-02	8.4E-02
Zinc	1.2E-05	**2.6E-01**	**2.6E-01**	1.7E-06	1.1E-03	1.1E-03	1.4E-05	**2.6E-01**	**2.6E-01**
Dust Palliatives Constituents									
Epichlorohydrin[a]	ND	ND	ND	7.4E-02	5.5E-05	7.4E-02	NC	NC	NC

Notes:
NA – Not applicable; HQ could not be estimated based on a lack of TRVs for COPC
ND – no baseline data available for COPC
NC – not calculated based on a lack of baseline data for COPC
BG > Project – cumulative risks were not estimated as the background soil concentration of the COPC was greater than the concentration of the COPC in coal (see discussion in Section 4.1.2)
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

Table 6-12 presents the non-cancer risk estimates for the residential toddler (maximum rail corridor residential receptor) exposed to the Project non-gaseous COPCs via inhalation, the direct soil pathways and ingestion of produce. Hazard quotients for all COPCs for the Project Scenario were less than the Health Canada negligible risk level of 0.2, indicating that there are no unacceptable risks associated with exposures to the Project emissions alone. HQs greater than 0.2 were predicted for the Baseline (i.e., based on background) and Cumulative Scenarios for aluminum, arsenic, cadmium, iron, lead, manganese, mercury, nickel, strontium, and zinc. The HQs predicted for each of these COPCs are discussed below:

- For arsenic, cadmium, manganese and zinc, the Cumulative Scenario HQs are equal to the Baseline Scenario HQs, indicating negligible contribution from the Project.
- The Cumulative Scenario HQs are approximately equal to the Baseline Scenario HQs for iron (Cumulative Scenario HQ = 0.28, Baseline Scenario HQ = 0.27), lead (Cumulative Scenario HQ= 0.70, Baseline Scenario HQ = 0.69) and mercury (Cumulative Scenario HQ = 0.26, Baseline Scenario HQ = 0.25). There is negligible contribution from the Project for these COPCs.

- For aluminum, the Baseline Scenario HQ (all routes) is 0.52 and the Project Scenario HQ (all routes) is 0.04, resulting in a Cumulative Scenario HQ (all routes) of 0.56 (i.e., 93% of the Cumulative HQ is due to background exposures). The contribution from the Project is considered to be negligible.
- For nickel, as with the maximum North Delta residential receptor, the inhalation HQ contributes the most significantly the to the total HQ for all scenarios, and if the inhalation pathway is not considered, the Cumulative HQ for the soil and vegetation pathways of 0.2 is equivalent to the Health Canada negligible risk level, and approximately equivalent to the Baseline HQ for the soil and vegetation pathways (i.e., negligible contribution from the Project Scenario). In addition, the Cumulative Scenario HQ for the inhalation pathway is 0.54 and is well below an HQ of 1.0, which, as discussed for the maximum North Delta residential receptor, is an appropriate target HQ for inhalation exposures to nickel. As such, no unacceptable risks are predicted for nickel.
- For strontium, the Baseline Scenario HQ (all routes) is 0.44 and the Project Scenario HQ (all routes) is 0.04, resulting in a Cumulative Scenario HQ (all routes) of 0.48 (i.e., 96% of the Cumulative HQ is due to background exposures). The contribution from the Project is considered to be negligible.

Table 6-13 presents the non-cancer risk estimates for the adult maximum rail corridor residential receptor for their multi-media exposure to the Project COPCs. The risk estimates for the Baseline, Project and Cumulative COPCs are also presented in Tables I-6A, I-6B and I-6C in Appendix I, and have been compared to the Health Canada negligible risk level of 0.2 for non-carcinogens.

Table 6-13: Chronic Risks for Multi-Pathway Exposures, Maximum Rail Corridor Residential Receptor (Adult)

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Carcinogenic PAHs									
Benzo(a)pyrene	6.3E-06	1.5E-04	1.6E-04	5.8E-07	4.2E-05	4.2E-05	6.8E-06	1.9E-04	2.0E-04
Benzo(a)anthracene	3.1E-06	1.1E-04	1.1E-04	2.3E-07	1.7E-05	1.8E-05	3.4E-06	1.3E-04	1.3E-04
Benzo(b)fluoranthene	5.5E-06	2.6E-04	2.6E-04	8.2E-07	6.1E-05	6.1E-05	6.3E-06	3.2E-04	3.2E-04
Benzo(g,h,i)perylene	NA	3.8E-05	3.8E-05	6.5E-08	4.6E-06	4.7E-06	NC	4.3E-05	4.3E-05
Benzo(k)fluoranthene	2.3E-06	6.8E-05	7.0E-05	1.8E-07	1.3E-05	1.3E-05	2.5E-06	8.1E-05	8.3E-05
Chrysene	NA	1.6E-04	1.6E-04	3.5E-07	2.6E-05	2.6E-05	NC	1.9E-04	1.9E-04
Dibenzo(a,h)anthracene	NA	8.0E-06	8.0E-06	4.8E-08	3.6E-06	3.7E-06	NC	1.2E-05	1.2E-05
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	1.3E-06	8.8E-05	8.9E-05	4.1E-05	7.0E-04	7.4E-04
Indeno(1,2,3-cd)pyrene	1.5E-05	3.8E-05	5.2E-05	1.7E-07	1.3E-05	1.3E-05	1.5E-05	5.0E-05	6.5E-05

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Phenanthrene	1.3E-04	1.2E-03	1.4E-03	4.0E-06	2.9E-05	3.3E-05	1.3E-04	1.3E-03	1.4E-03
Carcinogenic PAH Mixture	2.0E-04	2.7E-03	1.5E-03	7.7E-06	3.0E-04	2.7E-04	2.0E-04	3.0E-03	3.2E-03
Non-Carcinogenic PAHs									
Acenaphthene	8.6E-06	4.8E-05	5.7E-05	1.9E-07	1.3E-05	1.3E-05	8.8E-06	6.1E-05	7.0E-05
Acenaphthylene	NA	NA	NA	NA	NA	NA	NC	NA	NA
Anthracene	1.3E-06	1.9E-05	2.0E-05	3.3E-08	1.4E-06	1.4E-06	1.3E-06	2.1E-05	2.2E-05
Fluorene	3.0E-05	1.0E-04	1.3E-04	2.8E-07	9.3E-06	9.6E-06	3.0E-05	1.1E-04	1.4E-04
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	1.3E-06	8.8E-05	8.9E-05	4.1E-05	7.0E-04	7.4E-04
Naphthalene	1.5E-02	3.3E-04	1.5E-02	1.7E-06	7.6E-06	9.3E-06	1.5E-02	3.4E-04	1.5E-02
2-Methylnaphthalene	NA	7.9E-04	7.9E-04	4.8E-07	3.6E-05	3.6E-05	NC	8.2E-04	8.2E-04
Pyrene	3.8E-05	9.6E-04	9.9E-04	2.1E-06	1.5E-04	1.5E-04	4.0E-05	1.1E-03	1.1E-03
Non-Carcinogenic PAH Mixture	1.5E-02	2.9E-03	1.8E-02	6.0E-06	3.0E-04	3.1E-04	1.5E-02	3.2E-03	1.8E-02
Metals & Metalloids									
Aluminum	1.2E-07	1.5E-01	1.5E-01	2.2E-04	1.7E-02	1.7E-02	2.2E-04	1.7E-01	1.7E-01
Antimony	NA	2.4E-02	2.4E-02	9.6E-07	1.8E-04	1.8E-04	NC	2.4E-02	2.4E-02
Arsenic	4.9E-04	**5.7E-01**	**5.7E-01**	1.6E-04	1.2E-02	1.3E-02	6.5E-04	**5.8E-01**	**5.8E-01**
Barium	3.6E-03	3.5E-02	3.8E-02	4.3E-02	8.0E-03	5.1E-02	4.7E-02	4.3E-02	8.9E-02
Beryllium	4.3E-05	9.0E-04	9.4E-04	1.5E-03	2.5E-04	4.0E-04	1.6E-03	1.2E-03	1.3E-03
Boron	ND	NA	NA	7.2E-06	6.5E-02	6.5E-02	NC	NC	NC
Cadmium	1.7E-02	9.1E-02	1.1E-01	1.8E-03	1.4E-03	3.1E-03	1.9E-02	9.3E-02	1.1E-01
Chromium III	1.3E-04	1.0E-04	2.3E-04	7.4E-05	1.6E-07	7.4E-05	2.0E-04	1.0E-04	3.0E-04
Chromium VI	6.3E-03	1.0E-03	7.3E-03	3.0E-06	7.8E-03	7.8E-03	6.3E-03	8.8E-03	1.5E-02
Cobalt	2.6E-04	1.1E-02	1.1E-02	1.6E-03	3.2E-04	1.9E-03	1.8E-03	1.1E-02	1.3E-02
Copper	3.4E-03	4.8E-02	5.2E-02	1.6E-03	7.8E-04	2.4E-03	5.0E-03	4.9E-02	5.4E-02
Iron	1.6E-05	7.7E-02	7.7E-02	9.1E-05	7.2E-03	7.2E-03	1.1E-04	8.4E-02	8.4E-02
Lead	2.1E-02	8.5E-02	1.1E-01	1.9E-03	8.4E-04	2.7E-03	2.3E-02	8.6E-02	1.1E-01
Manganese	5.4E-02	**4.0E-01**	**4.5E-01**	4.7E-02	7.7E-04	4.8E-02	1.0E-01	**4.0E-01**	**5.0E-01**
Mercury	1.2E-02	1.0E-01	1.2E-01	3.4E-05	4.4E-03	4.4E-03	1.2E-02	1.1E-01	1.2E-01
Molybdenum	1.9E-05	6.7E-03	6.7E-03	5.2E-06	1.1E-04	1.2E-04	2.4E-05	6.8E-03	6.8E-03
Nickel	**4.0E-01**	8.4E-02	**4.8E-01**	1.4E-01	1.2E-03	1.4E-01	**5.4E-01**	8.5E-02	**6.3E-01**
Selenium	9.0E-06	9.2E-04	9.3E-04	4.0E-06	2.9E-04	3.0E-04	1.3E-05	1.2E-03	1.2E-03
Strontium	2.6E-07	1.9E-01	1.9E-01	1.1E-05	1.5E-02	1.5E-02	1.1E-05	**2.1E-01**	**2.1E-01**

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Tin	4.7E-08	2.2E-05	2.2E-05	3.3E-09	3.0E-07	3.1E-07	5.0E-08	2.2E-05	2.2E-05
Titanium	NA	1.1E-03	1.1E-03	1.5E-06	1.2E-04	1.2E-04	NC	1.2E-03	1.2E-03
Uranium	NA	8.0E-03	8.0E-03	1.1E-03	1.4E-03	1.4E-03	NC	9.4E-03	9.4E-03
Vanadium	2.6E-02	1.3E-02	3.9E-02	1.3E-02	1.6E-03	1.5E-02	3.9E-02	1.5E-02	5.4E-02
Zinc	4.7E-06	9.5E-02	9.5E-02	6.5E-07	4.0E-04	4.0E-04	5.3E-06	9.5E-02	9.5E-02
Dust Palliatives Constituents									
Epichlorohydrin[a]	NA	NA	NA	7.4E-02	2.6E-05	7.4E-02	NC	NC	NC

Notes:
NA – Not applicable; HQ could not be estimated based on a lack of TRVs for COPC
ND – no baseline data available for COPC
NC – not calculated based on a lack of baseline data for COPC
BG > Project – cumulative risks were not estimated as the background soil concentration of the COPC was greater than the concentration of the COPC in coal (see discussion in Section 4.1.2)
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

Table 6-14 presents the cancer risks for an adult residential receptor (maximum North Delta residential receptor) for their multi-media exposure to the Project COPCs. The estimated ILCRs are also presented in Table I-6B in Appendix I, and have been compared to the Health Canada negligible risk level of 1E-05 for carcinogens.

Table 6-14 Cancer Risks for Multi-Pathway Exposures, Maximum Rail Corridor Residential Receptor (Adult)

Chemical	Project Scenario ILCR
Carcinogenic PAHs	
Benzo(a)pyrene	2.9E-07
Benzo(a)anthracene	1.2E-06
Benzo(b)fluoranthene	4.2E-07
Benzo(g,h,i)perylene	3.2E-09
Benzo(k)fluoranthene	8.9E-08
Chrysene	1.8E-08
Dibenzo(a,h)anthracene	2.5E-07
Fluoranthene	8.1E-09
Indeno(1,2,3-cd)pyrene	8.6E-08
Phenanthrene	2.0E-09
Carcinogenic PAH Mixture	2.3E-06
Metals & Metalloids	
Arsenic	6.7E-06
Beryllium	9.1E-09
Cadmium	2.2E-08
Chromium VI	2.9E-06
Nickel	8.0E-08
Dust Palliatives Constituents	
Epichlorohydrin[a]	1.3E-08

As presented in Table 6-13 and 6-14, the non-cancer and cancer risk estimates for the adult maximum rail corridor residential receptor were less than the Health Canada negligible risk levels of 0.2 and 1E-05, respectively, with the exception of the HQs for the Baseline and the Cumulative Scenarios for arsenic, manganese and nickel, and the Cumulative Scenario HQ only for strontium. The HQs for these COPCs are discussed below:

- The Cumulative Scenario HQs for arsenic (Cumulative Scenario HQ = 0.58, Baseline Scenario HQ = 0.57) and strontium (Cumulative Scenario HQ = 0.21 and Baseline Scenario HQ = 0.19) are approximately equivalent to the Baseline Scenario HQs. As such, there is negligible contribution from the Project for these COPCs.

- For manganese, the Baseline Scenario HQ is 0.45 and the Project Scenario HQ is 0.05, resulting in a Cumulative Scenario HQ of 0.50 (i.e., 90% of the Cumulative Scenario HQ is due to background). The contribution from the Project is therefore considered to be negligible.
- Similar to the above discussion for nickel for the other receptors, the inhalation route contributes the most significantly to the overall Cumulative HQ (Cumulative inhalation HQ = 0.54, Cumulative HQ (all routes) = 0.63), and if the inhalation route is not considered, the Cumulative Scenario HQ for the soil and vegetation pathways of 0.085 is approximately equivalent to the Baseline Scenario HQ (0.088) and is less than the Health Canada negligible risk level of 0.2. As discussed above for the toddler, use of a target risk level of 1.0 for inhalation exposures to nickel is appropriate based on the mechanism of toxicity being specific to the inhalation exposure route; the Cumulative inhalation HQ of 0.54 is well below 1.0. In addition, an HQ greater than 0.2 was predicted based on background concentrations, with minimal contribution from the Project emissions and the most conservative of the available TRVs for nickel (nickel sulfate) was used. On this basis, and as the HQ is less than 1.0, no unacceptable risks are predicted for the inhalation of nickel.

Table 6-15 presents the total hazard quotients and ILCRs estimated for the COPCs identified as being additive. The non-cancer and cancer risk estimates have been compared the Health Canada negligible risk levels of 0.2 and 1E-05, respectively.

Table 6-15: Risk Estimates for Chronic Exposures to COPC Mixtures, Maximum Rail Corridor Residential Receptor (Toddler and Adult)

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
Chronic Inhalation	Nasal irritation/nasal lesions	Acetaldehyde, boron, acrolein**, epichlorohydrin, formaldehyde, naphthalene, nickel, propionaldehyde	HQ[a]: **6.8E-01**	HQ: 1.4E-01	HQ: **8.2E-01**
	Respiratory irritation	Chromium III, chromium VI, cobalt, copper, formaldehyde, NO_2, SO_2, propylene, vanadium	HQ: **1.0E+00**	HQ: 1.6E-01	HQ: **1.2E+00**
	Lung cancer	Arsenic, beryllium, cadmium, chromium VI, nickel, PAHs	NA	ILCR: 1.2E-05	NA
	Leukemia	Benzene, 1,3-butadiene**	NA	ILCR: 2.9E-10	NA
	Developmental effects	Styrene, xylenes	HQ: 8.2E-03	HQ: 5.9E-06	HQ: 8.2E-03
	Renal toxicity	Cadmium, ethylbenzene, uranium	HQ[a]: 1.8E-02	HQ: 2.9E-03	HQ: 2.1E-02
	Neurotoxicity	n-Hexane, manganese, mercury, toluene	HQ: 6.8E-02	HQ: 4.8E-02	HQ: 1.2E-01

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
Chronic Oral	Renal toxicity	Barium, pyrene, mercury, uranium	HQ Adult: 1.5E-01	HQ Adult: 1.4E-02	HQ Adult: 1.6E-01
			HQ Toddler: **3.4E-01**	HQ Toddler: 3.1E-02	HQ Toddler: **3.7E-01**
	Hepatotoxicity	Acenaphthene, copper,	HQ Adult: 4.8E-02	HQ Adult: 9.6E-04	HQ Adult: 4.9E-02
			HQ Toddler: 1.7E-01	HQ Toddler: 2.7E-03	HQ Toddler: 1.7E-01
	Reproductive Toxicity	Molybdenum, nickel	HQ Adult: 9.1E-02	HQ Adult: 1.3E-03	HQ Adult: 9.2E-02
			HQ Toddler: **2.1E-01**	HQ Toddler: 2.8E-03	HQ Toddler: **2.1E-01**

Notes:
[a] – no baseline air concentrations were available for boron, epichlorohydrin, propionaldehyde and uranium and therefore, HQs for the Baseline Scenario do not include these COPCs. It is noted that these COPCs are included in the estimation of the HQ for the Project Scenario
HQ: Hazard Quotient
ILCR: Incremental Lifetime Cancer Risk
NA: not applicable; risk estimates were not predicted for the Baseline and Cumulative Scenario for carcinogens, as the cancer risks are estimates as cancer risks above background
** - not a COPC for the maximum rail corridor residential receptor as it is present in combustion emissions from transportation equipment used at the facility
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

As presented in Table 6-15, HQs in excess of 0.2 were predicted for the Baseline and Cumulative Scenarios for the COPC mixtures identified as nasal and respiratory irritants for inhalation exposures, as well as the COPCs mixtures identified as renal and reproductive toxicants for oral exposures (toddler only). In the case of the reproductive toxicants, the HQs for the Cumulative Scenario (Baseline + Project) are equivalent to the Baseline Scenario, with negligible contribution from the Project emissions. Therefore, no unacceptable risks are predicted for the chronic inhalation of this COPC mixture in Project emissions.

In addition, the Project emissions contribute very little to the Cumulative HQs estimated for the nasal irritants (Baseline HQ = 0.68 and Project HQ = 0.14), the respiratory irritants (Baseline HQ = 1.0 and Project HQ = 0.2) and for the renal toxicants (Baseline HQ = 0.35 and Project HQ = 0.03). Given this, and as the estimates are based on the conservative assumption that the maximum concentrations of each of the COPCs will occur at the same locations (which the results of the Levelton AQA (2014) indicates is not the case), no unacceptable risks are predicted for the chronic exposure to these COPC mixtures in Project emissions.

6.2.2.2.1 Maximum Rail Corridor Residential Receptor Conclusions

Similar to the maximum North Delta residential receptor, exposures for the maximum rail corridor residential receptor have been estimated using a series of conservative assumptions, including that the receptor is exposed to the maximum predicted COPC concentrations for 24 hours a day, 7 days a week, 52 weeks a year for the duration of the project, and that they consume 100% of their produce from their backyard garden, or from another source that has been impacted by the Project emissions. The use of these conservative assumptions will tend to overpredict exposures, and therefore associated risks. Despite this, based on the information summarized above, no unacceptable risks are predicted for the maximum rail corridor receptor exposed to Project emissions.

6.2.3 Industrial Receptor

The acute and chronic inhalation risk estimates, as well as the cancer and non-cancer risk estimates for multi-media exposures, for the industrial receptor (adult) are summarized in the following tables. The industrial receptor was assumed to be exposed to the maximum concentrations of the COPCs predicted at the FSD fenceline (i.e., the overall maximum predicted concentrations). Characterization of acute exposures for the industrial receptor is considered protective of people involved in fishing activities on the Fraser River, who were identified to have the potential to be exposed to Project emissions on an acute inhalation basis only.

The industrial receptor was assumed to be exposed to the Project COPCs for 10 hours a day, 5 days a week, 48 weeks a year for the duration of the Project (10 years); no amortization was assumed in the estimation of exposures for the criteria air contaminants. Operable exposure pathways for the industrial receptor included inhalation of Project emissions and the direct soil pathways (soil ingestion, soil dermal contact and inhalation of soil particulate).

The risk estimates for the maximum industrial receptor are presented below for the Baseline Scenario, Project Scenario and Cumulative Scenario.

Table 6-16 summarizes the acute inhalation risk estimates for each of the Baseline, Project and Cumulative Scenarios for COPCs with acute inhalation TRVs; the risk estimates are also presented in Tables I-1A (Baseline Scenario), I-1D (Project Scenario) and I-1G (Cumulative Scenario) in Appendix A. It is noted that based on a lack of baseline air concentrations for select COPCs (e.g., boron, epichlorohydrin), risks were not estimated for the Cumulative Scenario. This is discussed in Section 7, the Uncertainty and Sensitivity Analysis.

Table 6-16: Risk Estimates for Acute Inhalation Exposures – Industrial Receptor (Adult)

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Metals			
Arsenic	3.6E-02	4.3E-02	7.9E-02
Boron	ND	1.4E-04	NC
Cadmium	6.3E-01	1.7E-02	6.5E-01
Copper	1.1E-03	7.8E-04	1.9E-03
Manganese	1.9E-01	6.5E-01	8.4E-01
Mercury	ND	1.0E-03	NC
Nickel	6.9E-02	1.2E-01	1.9E-01
Vanadium	8.9E-04	2.8E-03	3.7E-03
VOCs			
Acetaldehyde	7.1E-03	5.3E-03	1.2E-02
Acrolein	8.0E-02	8.2E-01	9.0E-01
Benzene	4.6E-03	4.3E-04	5.0E-03
1,3-Butadiene	1.6E-03	1.4E-03	3.0E-03
Ethylbenzene	2.2E-04	5.2E-06	2.3E-04
Formaldehyde	8.8E-02	6.2E-02	1.5E-01
Toluene	1.8E-03	3.6E-05	1.9E-03
Styrene	1.1E-03	1.0E-06	1.1E-03
Xylenes	2.1E-03	3.7E-05	2.1E-03
Dust Palliatives Chemical Constituents			
Epichlorohydrin	--	4.6E-08	NC
Others			
Sulfate	6.1E-02	3.0E-02	9.0E-02
Criteria Air Contaminants			
CO (1 hour)	4.3E-02	1.7E-02	6.0E-02
CO (8 hour)	1.0E-01	1.8E-02	1.2E-01
NO_2 (1 hour)	3.3E-01	****	5.6E-01
SO_2 (1 hour)	6.2E-02	2.2E-03	6.4E-02
SO_2 (24 hour)	9.0E-01	4.4E-03	9.0E-01
$PM_{2.5}$ (24 hour)	4.8E-01	2.0E-01	6.8E-01
PM_{10} (24 hour)	5.4E-01	3.7E-01	9.1E-01
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**,[a]	1.1E-01	**2.5E-01**	**3.6E-01**

COPC	Hazard Quotient: Baseline Scenario	Hazard Quotient: Project Scenario	Hazard Quotient: Cumulative Scenario
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[,b]	1.7E-01	**8.9E-01**	**1.1E+00**
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)** [,a]	1.1E-01	**2.5E-01**	**3.6E-01**
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)** [,b]	1.7E-01	**8.9E-01**	**1.1E+00**
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**	**1.2E+00**	1.2E-01	**1.3E+00**

Notes:
[a] – based on the TCEQ 1 hour ReV for acrolein
[b] – based on the OEHHA 1 hour REL for acrolein
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
** - no baseline air data were available for boron and epichlorohydrin; as discussed in previous sections of the report, due to a lack of sources, baseline air concentrations are not expected to appreciably contribute to exposures
**** - the Ambient Ratio Method (ARM) used by Levelton (2014) to predict the NO_2 emissions from the Project includes the background concentration. On this basis, COPCs from the Project alone could not be estimated; however, the HQ for the Cumulative Scenario includes Project + Background.
BOLD: indicates risks in excess of the target risk level of 1E+00 for acute exposures

As presented in Table 6-16, the HQs for the industrial receptor's acute exposures to the individual COPCs for each of the Baseline, Project and Cumulative Scenarios were below the target hazard quotient of 1.0 for acute exposures. On this basis, unacceptable health risks are not predicted for acute exposures to these COPCs from Project emissions.

The maximum mixture HQ estimated for the COPCs identified as nasal and ocular irritants exceeded the target risk level of 1.0 for the Cumulative (Baseline + Project) Scenario. In addition, the HQ for the COPCs identified as respiratory irritants exceeded 1.0 for both the Baseline and Cumulative Scenarios.

For both the nasal and ocular irritants, the HQs for the Project Scenario (maximum HQ of 0.89 for both COPC mixtures when the OEHHA acute (1 hour) REL is used for acrolein) exceed the HQs predicted for the Baseline Scenario (maximum HQ of 0.17 for both COPC mixtures when the OEHHA acute REL is used for acrolein). Acrolein, with a predicted HQ of 0.82 for the Project Scenario, has the highest relative contribution to the overall HQ for the COPC mixtures. The HQs are considered to overpredict risks based on the following:

- The acute exposures to the respiratory irritants, and all COPCs, were estimated using the maximum overall predicted 1 hour (or for select COPCs 24 hour) concentrations from Levelton (2014). These concentrations would occur on an infrequent basis (i.e., 1 hour or 1 day of the year). Additionally, the approach assumes that the maximum concentrations occur at the same location; however, a review of Levelton (2014) indicates that the maximum concentrations occur at different receptors and, thus, the approach overestimates actual risks.
- Although identified as ocular or nasal irritants, the mechanism of toxicity varies between the COPCs and thus, the assumption of additivity is conservative. The HQs for each of the individual COPCs are less than the target risk level for acute exposures of 1.0.
- The OEHHA acute inhalation REL for arolein was derived based on the lowest observed adverse effect levels (LOAEL) for two studies reporting mild subjective ocular and nasal irritation in human volunteers. Uncertainty factors of 60 (10 for human variability and 6 for use of a LOAEL) were applied to the LOAELs to derive the TRV. The TRV is therefore considered to be protective. In addition, as presented in Table 6-16, when the more recent Texas Commission on Environmental Quality (TCEQ) acute (1 hour) ReV of 11 µg/m3 is used, the acute HQs of the ocular and nasal irritants are both equal to 0.25 (i.e., the contribution from acrolein drops to a HQ of 0.19 when the more recent TCEQ acute ReV is used). As has been discussed in Appendix V, although the TCEQ was not identified as a preferred source in the TRV hierarchy, given the very recent and comprehensive toxiclogical review conducted for acrolein by the TCEQ, their recommended inhalation ReVs have been considered in the HHRA.

Given the above and the overall conservative approach used in the estimation of exposures and associated risks, and as the HQs (1.1) only slightly exceeds the target HQ of 1.0 for acute exposures when the potentially overly conservative OEHHA acute REL for acrolein is used, no unacceptable risks are anticipated for the industrial receptor's acute exposures the nasal and ocular irritant COPC mixtures.

For the respiratory irritants, the Baseline Scenario HQ is 1.2 and the Cumulative Scenario HQ (Project + Baseline) is 1.3 (i.e., 92% of the HQ is based on background exposures). Over half of the incremental HQ (i.e., HQ of 0.062) is due to formaldehyde and there is very little indication that it would interact with the other substances as a respiratory irritant. On this basis, the Project emissions have negligible contribution to the overall HQ and, therefore, no unacceptable risks are predicted for the COPC mixture identified as respiratory irritants in Project emissions.

Chronic inhalation risks associated with exposures to the non-carcinogenic gaseous COPCs, compared to the Health Canada negligible risk level of 0.2, are presented in Table 6-17 and in Tables I-2A, I-2D and I-2G. The

risks associated with chronic inhalation exposures to the COPC mixtures identified as having the potential to be additive are presented in Table 6-21. It is noted that no amortization has been assumed for the CACs; this approach is conservative, and will tend to overestimate risks.

Table 6-17: Non-Cancer Risk Estimates for Chronic Inhalation Exposures to Gaseous COPCs – Industrial Receptor (Adult)

Chemical	Baseline Hazard Quotient	Project Hazard Quotient	Cumulative Hazard Quotient
VOCs			
Acetaldehyde	2.2E-03	1.9E-05	2.3E-03
Acrolein[a]	2.0E-02	1.2E-04	2.0E-02
Acrolein[b]	1.5E-01	9.6E-04	1.6E-01
Benzene	4.9E-03	6.7E-05	4.9E-03
1,3-Butadiene	8.3E-02	8.5E-05	8.3E-02
Ethylbenzene	3.7E-04	1.1E-06	3.7E-04
Ethylene*	--	--	
Formaldehyde	1.8E-01	1.1E-03	1.8E-01
Hexachlorobenzene**	NA	--	--
n-Hexane	1.9E-04	1.0E-06	1.9E-04
Propionaldehyde	NA	1.0E-04	NC
Propylene (1-Propene)	3.6E-05	2.2E-07	3.7E-05
Toluene	2.7E-04	5.9E-07	2.7E-04
2,2,4-Trimethylpentane*	--	--	--
Styrene	4.0E-04	1.5E-06	4.0E-04
Xylenes	1.7E-03	2.9E-06	1.7E-03
Criteria Air Contaminants			
NO_2	6.8E-01	5.3E-01	**1.2E+00**
SO_2	1.6E-01	3.8E-04	1.6E-01
$PM_{2.5}$ (based on AAQO of 8 µg/m^3)	5.5E-01	8.8E-02	6.4E-01
$PM_{2.5}$ (based on planning AAQO of 6 µg/m^3)	7.3E-01	1.2E-01	8.5E-01
PM_{10}	6.0E-01	9.0E-02	6.9E-01
DPM	4.2E-02	1.8E-02	5.9E-02

Notes:
[a] – based on the TCEQ 1 hour ReV for acrolein
[b] – based on the OEHHA 1 hour REL for acrolein
ND: no baseline air data were available for COPC (see discussion in Section 7)
NC: not calculated; cumulative risks could not be calculated based on a lack of baseline air data for COPC
* - risks could not be predicted for ethylene and 2,2,4-trimethylpentane as no TRVs were identified for these COPCs
** - evaluated as a carcinogen only

The non-carcinogenic risks associated with chronic inhalation exposures to VOCs from Project emissions are less than the Health Canada negligible risk level of 0.2 for the Baseline, Project and Cumulative Scenarios for all VOC COPCs.

In addition, the HQs for the CACs were less than the target HQ of 1.0 for all three scenarios, with the exception of the Cumulative Scenario HQ for NO_2. Exposures to the CACs, including NO_2, were conservatively estimated assuming no amortization (i.e., it has been assumed that industrial receptors would be exposed for 24 hours a day). As discussed in Section 3, the Problem Formulation, Levelton (2014) predicted concentrations of NO_2 (maximum concentration of 48 µg/m^3) in excess of the AAQO at and adjacent to the FSD fenceline, in a region concentrated over the Fraser River. The exceedances were confined to an approximate 400 m^2 area over the river, and Levelton (2014) indicated that predicted annual average concentrations of NO_2 decrease with distance from the FSD fenceline, with concentrations below the AAQO of 40 µg/m^3 approximately 200 m from the fenceline, in an area over the Fraser River. Given that the exceedances are located at the fenceline and over the river, the potential for exposures to the elevated NO_2 are limited to receptors that would spend time in this area. As discussed in Section 3, the Problem Formulation, people involved in fishing activities were identified as receptors of concern, however, it was determined that fishing boats would not spend significant time in the area for the following reasons: 1) the area is located directly in the channel entrance to the FSD lower berths and therefore, there are safety concerns associated with boats maintaining a consistent position in the area; and 2). the area extends to the middle of the river channel between Surrey and Annacis Island and the strong currents in the area would prevent a fishing vessel from maintaining a consistent position in the area. It was therefore concluded that people involved in fishing activities would only be exposed to Project emissions on an acute basis only. As presented in Table 6-16, and discussed above, no unacceptable risks are predicted based on acute exposures to the maximum Project emissions. Based on the above, no unacceptable non-cancer risks are predicted for the industrial receptor's (and for people involved in fishing activities) chronic inhalation of gaseous COPCs in Project emissions.

Chronic inhalation risks associated with exposures to the carcinogenic gaseous COPCs are presented in Table 6-18 and in Table I-2D. The ILCRs have been compared to the Health Canada negligible risk level of 1E-05.

Table 6-18: Cancer Risk Estimates for Chronic Exposures to Gaseous COPCs – Industrial Receptor (Adult)

Chemical	Project Scenario ILCR
VOCs	
Acetaldehyde	1.3E-10
Benzene	2.1E-10
1,3-Butadiene	3.2E-11
Hexachlorobenzene	5.2E-14
Criteria Air Contaminants	
DPM	3.3E-06

Notes:
ILCR – Incremental lifetime cancer risk

The ILCR estimates (cancer risks above background) associated with chronic inhalation exposures to the gaseous COPCs were less than the Health Canada negligible risk level of 1E-05, and therefore no unacceptable risks are predicted for the industrial receptor's chronic inhalation of the gaseous, carcinogenic COPCs in Project emissions.

The non-cancer risk estimates for the industrial receptor's (adult) multi-media exposure to the Project COPCs are presented in Table 6-19, and in Table I-7A, I-7B and I-7C. The estimated HQs have been compared to the Health Canada negligible risk level of 0.2 for non-carcinogens.

Table 6-19: Chronic Risks for Multi-Pathway Exposures, Industrial Receptor (Adult)

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Carcinogenic PAHs									
Benzo(a)pyrene	1.7E-06	4.8E-06	6.5E-06	9.9E-06	3.5E-08	9.9E-06	1.2E-05	4.8E-06	1.6E-05
Benzo(a)anthracene	8.6E-07	5.6E-06	6.5E-06	1.7E-07	6.4E-08	2.4E-07	1.0E-06	5.7E-06	6.7E-06
Benzo(b)fluoranthene	1.5E-06	8.5E-06	1.0E-05	6.2E-07	1.3E-08	6.3E-07	2.1E-06	8.5E-06	1.1E-05
Benzo(g,h,i)perylene	NA	3.5E-06	3.5E-06	5.0E-08	2.1E-08	7.1E-08	NC	3.5E-06	3.5E-06
Benzo(k)fluoranthene	6.5E-07	3.4E-06	4.0E-06	1.3E-07	5.4E-08	1.9E-07	7.8E-07	3.4E-06	4.2E-06
Chrysene	NA	5.6E-06	5.6E-06	2.6E-07	7.4E-08	3.3E-07	NC	5.7E-06	5.7E-06
Dibenzo(a,h)anthracene	NA	8.5E-07	8.5E-07	3.6E-08	1.6E-08	5.2E-08	NC	8.6E-07	8.6E-07
Fluoranthene	1.1E-05	9.1E-06	2.0E-05	9.1E-07	1.7E-08	9.2E-07	1.2E-05	9.1E-06	2.1E-05
Indeno(1,2,3-cd)pyrene	4.1E-06	3.8E-06	7.9E-06	1.3E-07	5.3E-08	1.9E-07	4.2E-06	3.9E-06	8.1E-06
Phenanthrene	3.4E-05	7.3E-06	4.2E-05	1.6E-06	4.0E-10	1.6E-06	3.6E-05	7.3E-06	4.3E-05
Carcinogenic PAH Mixture	5.4E-05	5.3E-05	6.5E-05	1.4E-05	3.5E-07	1.3E-05	6.8E-05	5.3E-05	1.2E-04

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Non-Carcinogenic PAHs									
Acenaphthene	2.4E-06	1.4E-07	2.5E-06	1.4E-07	1.5E-11	1.4E-07	2.5E-06	1.4E-07	2.6E-06
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	3.4E-07	1.1E-07	4.6E-07	9.6E-07	1.9E-11	9.6E-07	1.3E-06	1.1E-07	1.4E-06
Fluorene	8.2E-06	4.2E-07	8.6E-06	1.7E-07	3.0E-11	1.7E-07	8.4E-06	4.2E-07	8.8E-06
Fluoranthene	1.1E-05	9.1E-06	2.0E-05	9.1E-07	1.7E-08	9.2E-07	1.2E-05	9.1E-06	2.1E-05
Naphthalene	4.1E-03	4.2E-07	4.1E-03	1.5E-05	5.8E-13	1.5E-05	4.1E-03	4.2E-07	4.1E-03
2-Methylnaphthalene	NA	2.1E-06	2.1E-06	3.6E-07	5.0E-12	3.6E-07	NC	2.1E-06	2.1E-06
Pyrene	1.1E-05	9.9E-06	2.0E-05	1.5E-06	2.9E-08	1.6E-06	1.2E-05	9.9E-06	2.2E-05
Non-Carcinogenic PAH Mixture	4.2E-03	2.2E-05	4.2E-03	1.9E-05	4.6E-08	2.0E-05	4.2E-03	2.2E-05	4.2E-03
Metals & Metalloids									
Aluminum	3.2E-08	1.6E-02	1.6E-02	1.6E-04	2.7E-04	4.3E-04	1.6E-04	1.6E-02	1.6E-02
Antimony	NA	5.9E-05	5.9E-05	5.1E-06	1.2E-06	6.3E-06	NC	6.0E-05	6.5E-05
Arsenic	1.3E-04	4.7E-03	4.9E-03	1.2E-04	1.1E-04	2.3E-04	2.6E-04	4.9E-03	5.1E-03
Barium	9.9E-04	1.3E-04	1.1E-03	3.2E-02	7.0E-05	3.2E-02	3.3E-02	2.0E-04	3.3E-02
Beryllium	1.2E-05	4.4E-05	5.6E-05	1.1E-03	4.0E-06	1.1E-03	1.1E-03	4.8E-05	1.1E-03
Boron	NA	NA	NA	5.3E-06	2.1E-05	2.6E-05	NC	NC	NC
Cadmium	4.7E-03	5.8E-05	4.7E-03	3.2E-03	3.0E-06	3.2E-03	7.9E-03	6.1E-05	8.0E-03
Chromium III	3.5E-05	7.0E-06	4.2E-05	5.6E-05	2.8E-09	5.6E-05	9.0E-05	7.0E-06	9.8E-05
Chromium VI	1.7E-03	6.2E-05	1.8E-03	1.7E-05	1.2E-04	1.4E-04	1.7E-03	1.8E-04	1.9E-03
Cobalt	7.1E-05	2.7E-04	3.4E-04	1.2E-03	4.8E-06	1.2E-03	1.2E-03	2.8E-04	1.5E-03
Copper	9.3E-04	5.4E-05	9.9E-04	1.2E-03	2.9E-06	1.2E-03	2.1E-03	5.7E-05	2.2E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	4.4E-06	8.1E-03	8.1E-03	6.8E-05	1.1E-04	1.8E-04	7.2E-05	8.2E-03	8.3E-03
Lanthanum	NA	NA	NA	4.1E-08	3.9E-10	4.1E-08	NC	NC	NC
Lead	5.9E-03	2.7E-03	8.6E-03	1.5E-03	2.8E-05	1.5E-03	7.3E-03	2.7E-03	1.0E-02
Manganese	1.5E-02	2.3E-03	1.7E-02	3.5E-02	1.3E-05	3.5E-02	5.0E-02	2.3E-03	5.3E-02
Mercury	3.4E-03	5.8E-05	3.4E-03	2.7E-05	9.3E-06	3.6E-05	3.4E-03	6.7E-05	3.5E-03
Molybdenum	5.3E-06	7.8E-06	1.3E-05	4.9E-06	3.8E-07	5.3E-06	1.0E-05	8.2E-06	1.8E-05
Nickel	1.1E-01	1.9E-03	1.1E-01	1.1E-01	3.7E-05	1.1E-01	**2.2E-01**	1.9E-03	**2.2E-01**
Selenium	2.5E-06	1.1E-05	1.3E-05	3.0E-06	2.4E-06	5.3E-06	5.4E-06	1.3E-05	1.8E-05
Strontium	7.1E-08	3.9E-05	3.9E-05	7.9E-06	1.3E-05	2.1E-05	8.0E-06	5.2E-05	6.0E-05
Tin	1.3E-08	3.6E-07	3.7E-07	6.2E-09	4.1E-09	1.0E-08	1.9E-08	3.6E-07	3.8E-07
Titanium	NA	8.6E-05	8.6E-05	1.1E-06	1.9E-06	3.0E-06	NC	8.8E-05	8.9E-05
Uranium	NA	4.4E-04	4.4E-04	8.1E-04	2.1E-05	8.3E-04	NC	4.6E-04	1.3E-03
Vanadium	7.1E-03	1.1E-03	8.3E-03	1.0E-02	2.7E-05	1.0E-02	1.8E-02	1.2E-03	1.9E-02
Zinc	1.3E-06	4.8E-05	4.9E-05	5.6E-07	8.1E-07	1.4E-06	1.8E-06	4.9E-05	5.1E-05
VOCs									
Hexachlorobenzene	ND	ND	ND	7.6E-05	1.2E-11	7.6E-05	NC	NC	NC
Dust Palliatives Constituents									
Epichlorohydrin[a]	NA	NA	NA	1.8E-05	1.9E-13	1.8E-05	NC	NC	NC

Chemical	Baseline Scenario			Project Scenario			Cumulative Scenario		
	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}	HQ_{inh}	$HQ_{soil/veg}$	HQ_{all}
Others									
PCBs	ND	ND	ND	5.0E-03	5.2E-05	5.1E-03	NC	NC	NC
Sulfate	NA	NA	NA	NA	NA	NA	NA	NA	NA

Notes:
NA – Not applicable; HQ could not be estimated based on a lack of TRVs for COPC
ND – no baseline data available for COPC
NC – not calculated based on a lack of baseline data for COPC
BG > Project – cumulative risks were not estimated as the background soil concentration of the COPC was greater than the concentration of the COPC in coal (see discussion in Section 4.1.2)
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

Table 6-19 presents the non-cancer risk estimates for the industrial receptor exposed to the Project non-gaseous COPCs via inhalation and the direct soil pathways. The ingestion of produce pathway was not considered to be operable for the industrial receptor. The Cumulative Scenario HQs are less than the Health Canada negligible risk level of 0.2 with the exception of the Cumulative HQs for nickel for the inhalation pathway. The Cumulative Scenario HQ for the inhalation pathway is 0.22 and is well below the target HQ for inhalation exposures to nickel of 1.0 (as discussed above for the other receptors). On this basis, no unacceptable risks are predicted for the industrial receptor for the COPCs presented in Table 6-19.

Table 6-20 and Table I-7B present the cancer risks for an industrial receptor (adult) for their multi-media exposure to the Project COPCs. The estimated ILCRs have been compared to the Health Canada negligible risk level of 1E-05 for carcinogens.

Table 6-20: Cancer Risks for Multi-Pathway Exposures, Industrial Receptor (Adult)

Chemical	Project Scenario ILCR
Carcinogenic PAHs	
Benzo(a)pyrene	5.9E-10
Benzo(a)anthracene	2.0E-09
Benzo(b)fluoranthene	7.0E-11
Benzo(g,h,i)perylene	6.4E-12
Benzo(k)fluoranthene	1.7E-10
Chrysene	2.4E-11
Dibenzo(a,h)anthracene	5.0E-10
Fluoranthene	1.3E-12
Indeno(1,2,3-cd)pyrene	1.7E-10
Phenanthrene	8.2E-13
Carcinogenic PAH Mixture	3.6E-09

Chemical	Project Scenario ILCR
Metals & Metalloids	
Arsenic	1.2E-07
Beryllium	6.3E-09
Cadmium	4.0E-08
Chromium VI	3.8E-08
Nickel	6.0E-08
VOCs	
Hexachlorobenzene	5.0E-09
Dust Palliatives Constituents	
Epichlorohydrin	2.7E-12

As presented in Table 6-20, the cancer risk estimates for the industrial receptor (adult) were less than the Health Canada negligible risk level of 1E-05, and thus, no unacceptable cancer risks are predicted for the industrial receptor.

Table 6-21 presents the total HQs and ILCRs estimated for the COPCs identified as being additive. The non-cancer and cancer risk estimates have been compared the the Health Canada negligible risk levels of 0.2 and 1E-05, respectively.

Table 6-21: Risk Estimates for Chronic Exposures to COPC Mixtures, Industrial Receptor (Adult)

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
Chronic Inhalation	Nasal irritation/nasal lesions	Acetaldehyde, boron, acrolein, epichlorohydrin, formaldehyde, naphthalene, nickel, propionaldehyde	HQ[a]: **3.1E-01** HQ[b]: **4.4E-01**	HQ[a]: 1.1E-01 HQ[b]: 1.1E-01	HQ[a]: **4.2E-01** HQ[b]: **5.5E-01**
	Respiratory irritation	Chromium III, chromium VI, cobalt, copper, formaldehyde, NO_2, SO_2, propylene, vanadium	HQ: **1.0E+00**	HQ: **5.4E-01**	HQ: **1.5E+00**
	Lung cancer	Arsenic, beryllium, cadmium, chromium VI, nickel, PAHs	NA	ILCR: 2.2E-07	NA
	Leukemia	Benzene, 1,3-butadiene	NA	ILCR: 2.4E-10	NA
	Developmental effects	Styrene, xylenes	HQ: 2.1E-03	HQ: 4.4E-06	HQ: 2.1E-03
	Renal toxicity	Cadmium, ethylbenzene, uranium	HQ: 5.1E-03	HQ: 4.0E-03	HQ: 9.1E-03
	Neurotoxicity	n-Hexane, manganese, mercury, toluene	HQ: 1.9E-02	HQ: 3.5E-02	HQ: 5.4E-02

Exposure Route and Duration	Critical Effect	COPCs Considered Additive	Baseline Risk Estimate	Project Risk Estimate	Cumulative Risk Estimate
Chronic Oral	Renal toxicity	Barium, pyrene, mercury, uranium	HQ: 6.3E-04	HQ: 1.0E-04	HQ: 7.3E-04
	Hepatotoxicity	Acenaphthene, copper	HQ: 5.5E-05	HQ: 4.2E-06	HQ: 5.9E-05
	Reproductive Toxicity	Molybdenum, nickel	HQ: 1.9E-03	HQ: 3.8E-05	HQ: 1.9E-03

Notes:
HQ: Hazard Quotient
ILCR: Incremental Lifetime Cancer Risk
NA: not applicable; risk estimates were not predicted for the Baseline and Cumulative Scenario for carcinogens, as the cancer risks are estimates as cancer risks above background
BOLD: indicates risks in excess of Health Canada negligible risk level of 0.2

As presented in Table 6-21, HQs in excess of 0.2 were predicted for the Baseline and Cumulative Scenarios for the COPC mixtures identified as nasal irritants for inhalation exposures, and for the Baseline, Project and Cumulative Scenarios for the COPCs identified as respiratory irritants.

For the nasal irritants, the maximum Baseline HQ is 0.44 (based on the OEHHA chronic inhalation REL for acrolein) and the Project HQ is 0.11, resulting in a maximum Cumulative Scenario HQ of 0.55. The Project HQ is equivalent to the inhalation HQ estimated for nickel (0.11). As discussed above, in the case of nickel, a target HQ of 1.0 for risks associated with inhalation exposures is appropriate as the mechanism of toxicity is specific to the inhalation route of exposure. The remaining COPCs in the mixture contribute negligibly to the Project HQ for the mixture and, therefore, no unacceptable risks are predicted for the COPC mixture of nasal irritants in Project emissions. When the HQ for nickel is not included in the estimation of the Baseline HQ (based on the use of a target risk level of 1.0), the estimated HQ ranges from of 0.3 (when the OEHHA REL for acrolein is used) to 0.2 (when the TCEQ ReV, which is considered to be more robust, for acrolein is used). It is noted that although the COPCs in the group are nasal irritants, the mechanism of toxicity varies, and thus the assumption of additivity is conservative. In addition, given the conservative approach used to estimate exposures, including the assumption that all predicted maximum concentrations occur at the same location, no unacceptable risks are predicted for cumulative (Baseline + Project) exposures to the nasal irritants.

For the respiratory irritants, NO_2 has the highest relative contribution to the HQ for the Project Scenario (HQ for NO_2 = 0.53 compared to the Project HQ = 0.54). The Project Scenario HQ for NO_2 is based on the maximum predicted annual average NO_2 concentration. As discussed above, the maximum NO_2 concentrations were predicted at the FSD fenceline and in areas over the Fraser River. Based on the locations of the maximum concentrations, it was determined that persons would be at these locations for only short durations and exposures

to these concentrations would only be on an acute basis, and therefore, no unacceptable risks were predicted for NO_2. When NO_2 is not included in the COPC mixture, the Cumulative HQ for the respiratory irritants is essentially equivalent to the Baseline HQ, indicating negligible contribution from the Project. On this basis, no unacceptable risks are predicted for the COPCs identified as respiratory irritants in Project emissions.

6.2.3.1 Conclusions for the Industrial Receptor

Exposures and associated risks for the industrial receptor were predicted using conservative assumptions, including exposure to the maximum predicted concentrations at the fenceline. As presented above, no unacceptable risks are predicted for the industrial receptor.

6.2.4 Agricultural Receptor

Belts of agricultural lands are located near the rail corridors in North Delta and Surrey. As agricultural areas are not located in areas of the highest Project emissions, consumption of food was assessed using the emissions data for the maximum residential receptors. The Health Canada (2012) recommended exposure frequency for days per year that food is ingested from a site is the same for both agricultural and residential lands (i.e., 365 days/year). Food consumption exposures and associated risks for the residential receptors are therefore be protective of consumption exposures and associated risks for agricultural lands.

Based on the above, and as no unacceptable risks were predicted for the residential receptors exposed to the maximum Project emissions predicted outside of the industrial area of the Project, no unacceptable risks are estimated for agricultural receptors exposed to Project emissions in the Study Area.

6.2.5 Commercial Receptor

As discussed in earlier sections of the report, evaluation of exposures and associated risks for the residential receptors is protective of the commercial receptor. Commercial receptors are not expected to be present for more than 8 hours a day, 5 days per week, and produce is not expected to be grown on commercial properties for consumption purposes. As such, and based on the assumptions used to characterize exposures for residential receptors, including an exposure frequency and duration of 24 hours a day, 7 days a week, 52 weeks a year, with 100% of their produce consumed from backyard gardens in the Study Area, exposures and, therefore, risks to commercial receptors would be well below those predicted for the residential receptors.

Based on the above, and as no unacceptable risks were predicted for the residential receptors exposed to the maximum Project emissions predicted outside of the industrial area of the Project, no unacceptable risks are estimated for commercial receptors in the Study Area.

6.2.6 Urban Park User

There are several urban parks located within the Study area including in the residential neighbourhoods adjacent the Project facility. While young children may be present at these locations, exposures would be much lower than those for the residential receptors, due to lower frequency and duration of exposures, and the lack of significant food production in parks. Although Health Canada (2012) does not recommend exposure frequencies or durations for an urban park user, the HHRA would conservatively assume that they would be present at a park in the Study Area for 2 hours a day, 7 days a week, 52 weeks a year. As such, and considering the assumptions used in the prediction of exposures for residential receptors (i.e., higher exposure frequency, assumption that 100% of their produce is consumed from a backyard garden in the Study Area), exposures and, therefore, risks for an urban park user would be much lower than those estimated for a residential receptor.

Based on the above, evaluation of a residential receptor is protective of an urban park user. As no unacceptable risks were predicted for residential receptors exposed to Project emissions, no unacceptable risks are estimated for urban park users in the Study Area.

The overall conclusions of the HHRA are presented in Section 8.

7 UNCERTAINTY AND SENSITIVITY ANALYSIS

Some degree of uncertainty is inherent to the prediction of any health risk, regardless of the source of the COPCs or the methods used in the assessment. In an effort to be health protective and not under predict potential risks associated with exposures to the Project emissions, the HHRA has been conducted using a series of conservative assumptions intended to reflect reasonable worst-case conditions. As a result, the results of the HHRA will tend to over predict exposures, and therefore associated risks, to Project emissions. Some of the main sources of uncertainty in the HHRA, and their impact on the results of the HHRA, are presented in the following table. Section 7.1 presents a sensitivity analysis evaluating the impact of using the mean concentrations of the coal constituents (versus the 95 % upper confidence limit of the mean), as well as TRVs from other sources, on the results of the HHRA.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
Problem Formulation	The air dispersion model used by Levelton (2014) uses both conservative assumptions meteorological conditions to predict emissions, resulting in conservative air quality estimates. The conservatism in the air quality model is described in Levelton (2014). A summary of the main sources of conservatism in the model include: ◆ Use of reasonable worst-case meteorological conditions. ◆ Reporting of maximum concentrations over several different metrological/emissions scenarios. ◆ The modelling domain contains over ten thousand receptors where ambient air concentrations were predicted, which included numerous sensitive receptors for: hospitals; schools; senior care residences; and, day care centres. ◆ Although the reported emission rates of some of the PAH species were below detection limit, the detection limit values were adopted to give a conservative estimate for the FSD HHRA study. The maximum concentrations predicted by Levelton (2014) at the highest exposed receptors (e.g., maximum North Delta residential receptor, maximum rail corridor residential receptor and a fence-line [industrial] receptor) were reported by Levelton (2014) and used to predict exposure point concentrations in the HHRA. In the case of the maximum residential receptors, maximum predicted concentrations of the varying CACs were predict to occur at different locations; however, the HHRA assumed these maximum concentrations to be occurring in one 'maximum' location with a single receptor exposed to all maximum concentrations. The HHRA assumed exposure to maximum predicted concentrations on a continuous basis.	Use of the Levelton (2014) maximum predicted concentrations and the assumption that the predicted maximum concentrations occur at a single receptor location has resulted in an over-prediction of potential exposures. It is noted that the (2014) Air Quality Assessment is based on air dispersion modelling, and as with all models, there is inherent uncertainty. The assumptions and associated uncertainty in the model are detailed in the Levelton (2014) AQA report; SNC-Lavalin understands that appropriate and generally conservative assumptions were used in the modelling, with the model developed with input from Metro Vancouver. SNC-Lavalin also understands that an Air Quality Management Plan will be developed for the Project, and will include air monitoring to validate the model.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	Chemical laboratory analysis of coal was conducted by an independent Canadian Association for Laboratory Accreditation certified laboratory (ALS Environmental). The analytical results were used to determine the concentrations of constituents (metals, metalloids and PAHs) in coal proposed to be transported during the Project. Lab analysis was conducted on coal particles representative of coal dust (< 100 µm) in an effort not to underestimate concentrations that receptors have the potential to be exposed to. The laboratory analysis of the coal, and in particular the extractions performed as part of the analysis, will tend to overestimate the bioavailability of the coal constituents. As discussed throughout the report, metals, metalloids and PAHs in coal are generally not bio-available under typical environmental conditions. The availability of coal constituents is considered to be an important source of uncertainty.	The use of the coal constituent concentrations reported by the laboratory, as well as the assumption of 100% bioavailability in the estimation of oral and inhalation exposures for the majority of the coal constituents, is likely to have resulted in an over-prediction of exposures to the coal constituents in fugitive dust from the Project.
	To assess human bio-accessibility of select metals, (i.e., how much of the metal in coal is anticipated to be available for uptake in the human gastrointestinal tract), coal samples were submitted to Royal Road University for analysis using the Physiologically Based Extraction Test (PBET); PBET is an in vitro test system which simulates the human gastrointestinal tract conditions and is used estimate the bio-accessibility of metals from a solid matrix. Health Canada supports the use of PBET results in the estimation of oral/ingestion exposures to lead. The results of the PBET testing were used as a data source in the HHRA for determining lead bioavailability for oral/ingestion exposures.	The use of PBET results in the HHRA was done in accordance to Health Canada guidance to more accurately reflect human exposures. Therefore, the use of PBET results is anticipated to result in a more accurate prediction of exposures to lead via the oral pathways (e.g. soil ingestion)
	Baseline air data was not available for select COPCs including PAHs, select metals (boron, antimony, mercury, uranium, titanium) and select other COPCs (acrolein, acetaldehyde, formaldehyde, hexachlorobenzene, PCBs, epichlorohydrin). Based on the lack of available data, a literature search was conducted. Ambient air data for Canadian cities were available for PAHs, acetaldehyde, acrolein and formaldehyde. Although Canadian data were not available for mercury, the maximum of the range of typical ambient air concentrations reported by the European Commission (available at http://ec.europa.eu/environment/air/pdf/pp_mercury4.pdf) was used. As noted in text, where reported as a range, the maximum concentrations were conservatively selected in an effort to ensure that baseline exposures are not underestimated. No background air concentrations were available for select PAHs (2-methlynaphthalene), select metals (antimony, boron, uranium and titanium), hexachlorobenzene, PCBs and epichlorohydrin. Similarly, no background soil data were available for select COPCs (boron, PCBs and epichlorohydrin). Epichlorohydrin was retained as a COPC as it is a chemical constituent in the dust palliatives. No other appreciable sources of epichchlorohydrin are anticipated in the Study Area, as it is associated with the chemical manufacturing industry, and in particular is formed during from the manufacture of various chemicals (e.g. glycerin), plastics and rubbers. As discussed throughout the report, the approach used to estimate exposures and associated risks to dust suppressants is highly conservative; the HHRA assumes the entire dust suppressant is composed on the most potent ingredient (epichlorohydrin) and therefore, risks estimates associated with exposures to epichlorohydrin in Project emissions are highly conservative and are well below the Health Canada negligible risk levels. Given this, and as baseline concentrations are not expected to	As has been discussed in the report, although there is uncertainty in using literature values, this approach was preferred to not assessing the Baseline Scenario for these COPCs. Data for Canadian cities, and where available, for Vancouver, was used. For some parameters (e.g., PAHs) the data are older; however, given the technological advances in reducing combustion emissions, the use of this data is considered conservative. Given the conservative approach used in the selection of the literature based concentrations, it is likely that the data are overestimates of current ambient concentrations in residential locations near the FSD facility. As such, use of the literature values is likely to overpredict exposures and associated risks for the Baseline Scenario.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	contribute appreciably to exposures, the risks estimates for epichlorohydrin are considered to overestimate actual risks. As discussed in Section 4, no significant sources of PCBs are expected in the Study Area, and therefore, baseline exposures and not likely to contribute appreciably to overall exposures. The approach used to evaluate PCBs from Project emissions is highly conservative as it is assumed that PCBs are present as the most potent congener. As such, and given that baseline concentrations are not expected to contribute appreciably to exposures, the risks estimates for PBCs in Project emissions are considered to overestimate actual risks.	
	SNC-Lavalin conducted a review of the dust palliative, or dust suppressant, agents proposed for use at FSD, as well as those likely to be used on the coal transported to FSD by rail. Dust palliatives are applied to reduce potential exposures to coal. The HHRA evaluated the potential for exposures to both coal dust, and the constituents of the dust palliatives. These agents bind to coal to reduce fugitive dust. Therefore, dust palliatives are not anticipated to be present at significant levels in fugitive dust due to the efficacy of these agents.	The evaluation of potential health risks of dust palliative constituents likely results in an over prediction of human exposures to dust suppressants in fugitive dust from the Project as dust palliatives are likely less available than assumed in the HHRA.
Exposure Assessment	The HHRA used maximum predicted concentrations of air and dust fall predicted by Levelton (2014) at the Facility Area and the Rail Corridor to determine chronic exposures to receptors in the Study Area. In addition, the maximum constituent specific deposition rates from multiple potential combustion sources were used in the exposure assessment. These maximum assumed rates were used to estimate resulting soil and vegetation concentrations used to determine exposure point concentrations (EPCs) in the HHRA.	The use of maximum predicted dustfall, air concentrations and deposition rates has likely resulted in the over-prediction of risks.
	It was assumed that residents adjacent the Project Facility and along the Rail Corridor would remain at their residents and exposed to the Project COPCs for 24 hours per day for their entire lifetime. It was assumed that residents would be inhaling maximum Project air emissions assumed to occur at a single location for a period of 24 hours per day, 7 days per week, for the entire duration of the Project. Furthermore, it was assumed that soil and produce exposures would continue following the Project duration with average soil and produce concentrations predicted for the duration of the Project assumed to persist for a person's lifetime. This is an overestimate of soil and produce exposures as chemical loss process that would occur following the cessation of the Project (e.g., bio-degradation, volatilization) are not taken into account. Statistics Canada reports an average motility rate of 46% (movers) over a 5 year period (based on Statistics Canada - 2006 Census. Catalogue Number 97-556-XCB2006006. http://www12.statcan.ca/census-recensement/2006/dp-pd/tbt/Rp-); however, this information does not account for the potential of movement within the same area. Mobility information from the US Bureau of Census which considered zip codes (as presented in US EPA, 2005) indicates that typically, individuals do not reside at the same residence for the entirety of their lifetime. Further to the number of years at a particular location/residence, the amount of time spent at that location each day varies (e.g., people working away from home, children attending schools outside of areas where maximum concentrations were predicted). The US EPA (2005) recommends use of a	The receptor exposure duration assumptions used in the HHRA are highly conservative when determining chronic risks due to the Project. It is considered unlikely that a single receptor would be exposed to maximum predicted concentrations to the extent of that assumed in the HHRA. The use of the conservative assumptions will result in the overprediction of exposures and risks for receptors in the Study Area.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	30 year exposure time for combustion related emissions to account for mobility. For soil and vegetation exposures, the HHRA has conservatively assumed an 80 year exposure duration for residential receptors. Although motility was not taken into account, carcinogenic risks were not drivers of risks in the HHRA. Therefore, the conservatism in this parameter is not anticipated to significantly change the results of the HHRA. The HHRA assumed that industrial workers would be exposed to soil impacted by the Project emissions for 10 hours per day, 5 days per week for a period of 35 years based on Health Canada guidance. It was also assumed that industrial workers would be exposed to the Project COPCs in air for 10 hours a day, 5 days a week for the lifetime of the Project (10 years).	
	The HHRA considered inhalation of suspended soil dust. Concentrations of dust arising from soil were estimated using near surface soil concentrations (i.e., the top 2 cm) and the Health Canada recommended soil dust rates for typical environments. It was assumed that residential receptors would be exposed to soil particulate (i.e., dust from soil) 24 hours a day (both indoors and outdoors) to conservatively account for exposures to indoor dust sourced from Project emissions.	Project related soil dust concentrations in the outdoor environment are anticipated to be higher than that of Project sourced COPCs in the indoor environment. Therefore the assumption that receptors are exposed to outdoor soil dust 24 hours a day will tend to over predict exposures and associated risks. However, it is acknowledged that exposures to soil dusts were not considered a significant contributor to risks in the HHRA.
	It was assumed that 100% of the vegetation consumed by residents would be obtained from a home garden in the Project area (i.e., that no other sources would be used to supplement the home garden produce). Furthermore, when estimating COPC concentrations in produce, it was assumed that vegetation would not be washed/peeled prior to consumption. In addition, the US EPA (2005) recommended correction factor for below ground chemical uptake was not applied; the correction factor is typically applied to exposure estimates to account for the protective outer skin, size, and shape of bulky produce, and transfer of lipophilic COPCs to the center of the produce being lower than that accounted for in the US EPA (2005) exposure model (used to determine produce concentrations in the HHRA). As the correction factor is based on literature data for bulky produce, and does not take into account leafy vegetables/legumes which may be present in home gardens, the application of this factor was not considered appropriate as the HHRA was conducted to assume a wide array of produce. In the urban environment, it is very likely that a garden produce supply would be supplemented with other sources. Also, COPCs may be reduced from produce as a result of washing, peeling and/or cooking (US EPA, 2005). The outer layer of above ground produce, such as broccoli or lettuce is often removed prior to consumption, root vegetables are typically washed and some vegetables contain outer protective layers (e.g., melons, corn husks) that may not be commonly used for consumption (US EPA, 2005). However, as a conservative approach was used in the HHRA, correction or reduction factors taking the above into account	The assumption that garden produce represents 100% of the produce consumed and residents, as well as the lack of application of a wash/peel factor or empirical correction factor, will result in an over prediction of vegetable COPC concentrations and therefore an over-prediction of risks due to consumption.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	were not incorporated into produce exposure modelling. Although the above is considered to result in an over prediction of COPCs in produce, considering the relatively low contribution of produce consumption to overall risks estimates, these assumptions are not anticipated to significantly impact the results of the HHRA.	
	The US EPA (2005) recommends a soil mixing depth of 2 cm or 20 cm, when determining soil and vegetable root uptake exposures. It was assumed that Project scenario soil would have a 2 cm mixing depth and furthermore, soil run-off was not assumed to occur. As the shallower mixing depth was assumed, higher COPC predictions per unit soil mass was estimated than if the 20 cm mixing depth were used. The use of a 2 cm mixing depth was considered appropriate for determining soil contact exposures (e.g., dermal contact, dust generation and incidental ingestion). However, the use of this parameter for determining root uptake is conservative as the majority of a plant's root mass will be present beyond depths of 2 cm. Furthermore, the bio-concentration factors used in the HHRA are considered to be highly conservative, further resulting in an over prediction of root uptake exposures.	The assumption that chemical loss due to run-off does not occur results in the over-prediction of COPC concentrations in soil (and subsequently vegetation via the soil uptake pathway) and therefore will result in the over-prediction of COPC exposure and associated risks. The soil modelling depth is not anticipated to result in over or under prediction of exposures due to direct soil contact, but is anticipated to overestimate COPC concentrations in produce, resulting in the over-prediction of risks for the food consumption pathway.
	The HHRA evaluated exposures to dust palliatives. It was assumed that epichlorohydin, the dust palliative constituent of the highest known toxic potency, formed the entirety of the dust suppressant. The dust suppressant application rate (units of mass of suppressant per unit mass of coal) was used to determine the epichlorhydrin concentrations in fugitive dust from the Project. It is noted that according to manufacturer's MSDS, 30-60% of the product is an adipic acid, diethylenetriamine, epichlorohydrin polymer. To estimate the air concentration of epichlorohydrin for the Project Scenario, the manufacturer's (i.e., GE's) application rate was used along with the predicted coal dust concentration. It was conservatively assumed that 100% of the product is epichlorohydrin (vs. the 30-60% adipic acid, diethylenetriamine, epichlorohydrin polymer).	The assumption of the dust palliative being comprised 100% of the constituent of highest known toxic potency will result in an over prediction of exposures to epichlorohydin from the Project and thus the over-prediction of risks resulting from exposure to dust palliatives associated with the Project.
	Use of US EPA (2005) model for combustion sources and recommended input parameters, where appropriate, were used to determine EPCs of soil, soil dust, above ground produce and below ground produce in the HHRA. The US EPA (2005) risk assessment guidance is specific to combustion sources with modelling equations and input parameters taking into account other relevant EPA studies and the results of US EPA literature searches. Chronic daily intakes of COPCs for the above media were determined using uptake equations, receptor characteristics and receptor exposure durations recommended by Health Canada (2012). Given the scrutiny of review conducted by regulatory agencies and the supporting literature sources considered by the US EPA and Health Canada, the use of these models and guidance are considered appropriate for use in conducting HHRAs for regulatory compliance. Therefore, the approach used in this HHRA is similar to those conducted until similar scenarios for the purposes of regulatory compliance.	Exposure modelling of soil, soil dust, above ground produce and below ground produce in the HHRA was largely based on guidance provided by the US EPA (2005) and is therefore consistent with HHRAs conducted under similar scenarios. Human uptake of these EPCs was determined using Health Canada recommended equations and receptor assumptions. Although the use of the framework and models provided by these agencies is not anticipated

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
		to over or under predict risk, it results in an HHRA that is consistent/appropriate for the purposes of achieving regulatory compliance.
	The US EPA OSW (2005) recommends an Rp (intercept fraction of edible plant parts) value of 0.05 for fruits, vegetables and legumes; however, the later sections of the document detail the limitations/uncertainty in predicting vegetable COPC concentrations arising from the Rp parameter. The US EPA OSW (2005) document also recommends a more conservative Rp value of 0.5 for forage. Given the US EPA identified uncertainties in the Rp value, and as an effort was made to protect for a wide variety of produce that may be grown within the Study Area, the more conservative Rp value was used in the HHRA. As discussed in Appendix B of US EPA OSW (2005), when determining COPC concentrations in above ground produce, the uncertainties associated with Rp are introduced "when the calculated parameter values don't accurately represent aboveground produce-specific values" while "uncertainties associated with Yp are not expected to be significant". The use of the US EPA OSW (2005) Rp of 0.5 is considered to be conservative and is considered to address uncertainly associated with variable food type.	The use of the more conservative value could result in an approximate order of magnitude overprediction in exposures resulting from the ingestion of above ground produce.
	As noted, where background ambient air concentrations were not available, for select parameters (PAHs, acrolein, formaldehyde and mercury), literature values were used. As discussed above, an effort was made to identify data for Canadain cities, and a conservative approach was used in the selection of the values. The concentrations obtained from the literature ranged from one month to annual average concentrations. Adjustment factors for estimating a 24 hour or 1 hour concentration from an annual average concentration, for example, are available; however, given the conservative approach used in the selection of the ambient air concentrations for these parameters, and in an effort not to over-inflate baseline exposures and risks, these adjustment factors were not used in the HHRA.	The acute inhalation risks for these parameters from Project emissions were lower than those estimated for the Baseline Scenario using the literature derived background concentrations. On this basis, and as it was concluded that no unacceptable risks were predicted for Project emissions, use of a higher acute baseline concentration would not have changed the results of the HHRA.
Toxicity Assessment	Due to the absence of a threshold, $PM_{2.5}$ concentrations to protect all individuals from all possible health outcomes cannot be derived (WHO, 2006). A review of the regional health status with regards to $PM_{2.5}$ was conducted to address uncertainty (see Appendix IV). WHO (2006) indicates that epidemiological studies on large populations have not identified a threshold concentration for non-mortality endpoints below which ambient PM has no effect on health. As some health effects are expected with any increase of $PM_{2.5}$, $PM_{2.5}$ objectives have been set to consider the context of continually improving air quality and establishing guidelines at concentrations where significant adverse effects have not been demonstrated. Due to the absence of a threshold, $PM_{2.5}$ concentrations to protect all individuals from all possible health outcomes cannot be derived (WHO, 2006), however, WHO (2006) indicates that the measureable health effects (i.e., morbidity endpoints) at ambient concentrations of 11 µg/m³ are similar to those observed at background concentrations of 3 µg/m³ to 5 µg/m³, and therefore have recommended a ambient air guideline for $PM_{2.5}$ of 10 µg/m³.	As indicated in Section 6, no measureable health risks are anticipated for $PM_{2.5}$ concentrations below the WHO (2006) guideline of 10 µg/m³. Given that the maximum predicted annual average $PM_{2.5}$ concentrations + background for the Project are less than the Metro Vancouver and BC MoE planning goal of 6 µg/m³, no measureable health effects are predicted from $PM_{2.5}$ from Project emissions.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	Although diesel emissions are classified as a known carcinogen (IARC) or a likely human carcinogen (US EPA), few agencies, including the US EPA, have derived carcinogenic TRVs (e.g. inhalation unit risk, slope factor) for diesel emissions. The US EPA (http://www.epa.gov/iris/subst/0642.htm) indicates that a quantitative estimate of carcinogenic risk from inhalation exposure to diesel emissions has not been derived based on the absence of adequate data to develop a sufficiently confident dose-response relationship from the epidemiological studies. The inhalation UR included in-text for DPM of $3.0x10^{-4}$ $(\mu g/m^3)^{-1}$ is recommended by the OEHHA, and is based on human occupational exposure lung tumor incidence in studies of US railroad workers (OEHHA, 2009). It is noted that the unit risk value is considered to be conservative given that is based on occupational exposure data, and thus the incremental lifetime cancer risk estimate is inherently conservative and likely overestimates risks. In addition, personal communication with Health Canada suggests that the cancer risk value is not widely accepted within Canada and may overestimate the carcinogenic potency of diesel particulates. Based on a lack of a more defensible TRV, the OEHHA inhalation UR was used in the HHRA to characterize carcinogenic risks associated with DPM.	Given the conservatism in the TRV for diesel emissions, cancer risks estimated using the OEHHA (2009) inhalation unit risk estimate are considered to be overestimates of actual risks.
	Chronic inhalation TRVs for NO_2 vary from 40 ug/m^3 (WHO) to 60 ug/m^3 (CCME, maximum desirable) / 100 ug/m^3 (CCME, maximum acceptable). The HHRA selected the most conservative TRV of 40 ug/m^3 as WHO (2006) indicated that retaining a conservative annual NO_2 guideline is considered prudent and health-protective. During epidemiological studies NO_2 is often used as a marker for other combustion-generated pollutants and it is difficult to attribute health effects solely to NO_2 exposure when there are other correlated co-pollutants present. In the case of Facility Area emissions, Levelton (2014) predicted concentrations of NO_2 (maximum concentration of 48 $\mu g/m^3$) in excess of the AAQO at and adjacent to the FSD fenceline, in a region concentrated over the Fraser River. The exceedances were confined to an approximate 400 m^2 area over the river, and Levelton (2014) indicated that predicted annual average concentrations of NO_2 decrease with distance from the FSD fenceline, with concentrations below the AAQO of 40 $\mu g/m^3$ approximately 200 m from the fenceline, in an area over the Fraser River. Given that the exceedances are located at the fenceline and over the river, the potential for exposures to the elevated NO_2 are limited to receptors that would spend time in this area.	Based on the conservatism assumed in the exposure assessment and the use of the more conservative WHO TRV for predicting potential risks due to NO_2 exposures, it is considered unlikely that potential health risks have been underestimated.
	A suitable TRV was unavailable for 2,2,4-trimethylpentane. The maximum predicted annual average concentration of 2,2,4-trimethylpentane for all receptors is 6.2E-04 ug/m^3. Although not specific to 2,2,4-trimethylpentane, Health Canada recommends a TRV of 1,000 ug/m^3 for hydrocarbons in the C8-10 aliphatic hydrocarbon range. Predicted 2,2,4-trimethylpentane exposures are orders of magnitude lower (6.2E-04 ug/m^3 ÷ 1,000 ug/m^3 = 6.2E-07).	On this basis, it is unlikely that the lack of a TRV for this substance would change the results of the HHRA.
	The TRVs provided by regulatory health agencies (e.g., Health Canada, US EPA, WHO) are protective of critical sub-groups, or sensitive subpopulations (i.e., those with physical characteristics or conditions that may result in an increased likelihood of adverse effect to a given level of exposure, for example, the elderly or	The HHRA used TRVs recommended by regulatory health agencies to determine potential risks resulting from exposure to

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	persons suffering from existing medical conditions). These sensitive subpopulations are considered by the agencies in the derivation of TRVs. When deriving TRVs, health agencies apply safety or uncertainty factors (i.e., an intraspecies/human variability uncertainty factor) to protect for sensitive subpopulations. TRVs are determined from responses to exposures observed in toxicity (animal) studies and epidemiology (human) studies. For noncarcinogens, these responses were typically reported as an oral dose (i.e., mg chemical/kg body weight/day), or air concentration (i.e., mg/m3), associated with a No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL), which was then adjusted (i.e., reduced) by the application of uncertainty factors. Uncertainty factors are assigned to account for uncertainty of the response between species (e.g., 10-fold), the response within a species population (e.g., 10-fold), the difference in response to sub-chronic versus chronic exposures (e.g., 10 fold), the difference between a LOAEL and the NOAEL (e.g., 10-fold), and the quality of the database for observed effects (e.g., 3-fold). The overall uncertainty associated with an observed response is the product of the individual uncertainty factors and generally ranges from 10 to 1000.	COPCs. TRVs selected for use in the HHRA have incorporated an uncertainty factor to account for potential inter-individual differences in sensitivity. Uncertainty factors generally result in a 10 to 1000 fold adjustment/reduction to account for sensitivity. Therefore, the TRVs are considered to be health protective, and protective of sensitive sub-populations. It is considered unlikely that risks have been under-predicted based on the TRVs used in the HHRA.
	No TRVs are available for select COPCs including acenaphthylene, diethylaminoethanol, diethylenetriamine and succinic acid. Although no Worksafe BC limits were available for linear alkyl sulfonate and succinic acid, the limits available for epichlorohydrin, diethylaminoethanol and diethylaminotriamine support the assumption that epichlorohydrin has the highest relative toxicity for the dust palliative constituents. Despite the lack of TRVs and occupational exposure limits for linear alkyl sulfonate and succinic acid, the toxicity of these COPCs is unlikely to exceed that of epichlorohydrin based on how they are used. Succinic acid is a food additive (as an acidity regulator), is used in pharmaceuticals, and is on the US Food and Drug Administration's (FDAs) Generally Recognized as Safe (GRAS) list. Linear alkyl sulfonate is a surfactant that is widely used in household and personal care products.	Due to the assumption that epichlorohydrin, the constituent of highest known toxic potency comprises 100% of the dust palliative, the lack of TRVs for these COPCs is not anticipated to change the results of the HHRA. As discussed above, the assumption that the dust suppressants are comprised 100% of the constituent with the highest known potency will tend to overestimate risks.
	Health Canada (2012) guidance indicates that total Hazard Quotients are interpreted according to the following general guidelines: < 0.2 = negligible human health risks; and > 0.2 = potential unacceptable risks which may require mitigation or more detailed assessment. Health Canada's negligible risk level of 0.2 (or 20% of the TRV) for non-carcinogens allows for 80% of the acceptable exposure level (i.e., as defined by the TRV) to come from other sources; this approach is based on the potential for exposures to a chemical in air, soil, water, food and consumer products (i.e., 20% of the acceptable exposure is allocated to each of these 5 media/sources). However, Health Canada considered a HQ of 1.0 acceptable when background/multimedia exposures are taken into account. Where available, the HHRA used background exposure data for determining cumulative risks.	The risk estimates in the HHRA assumed air, soil and food exposures and considered background concentrations where available. The Health Canada (2012) essentially negligible human health risks HQ of 0.2 is derived to take into account additional background and multimedia sources of COPCs. Therefore, use of a HQ of 0.2 as a target risk level in the HHRA where background and multiple exposure media have been considered is highly conservative.

HHRA Section	Source of Uncertainty	Impact on Results of the HHRA
	The report evaluated potential health risks resulting from the Project using a risk assessment framework. The HHRA was completed using Health Canada risk assessment guidance and is generally consistent with risk assessment methodology used by the US EPA and WHO.	The use of Health Canada guidance and supporting regulatory guidance indicates that the HHRA results are consistent with HHRA's conducted under similar assumptions and scenarios for Projects those seeking regulatory compliance. It is considered unlikely that risks have been under predicted in the context of the regulatory framework.
	As discussed through out the report, the mean concentrations of majority of the metals (antimony, arsenic, bismuth, cadmium, chromium (+3), chromium (+6), cobalt, copper, lead, lithium, manganese, molybdenum, nickel, silver, tin, uranium, vanadium, zinc, aluminum, iron, thallium and titanium) and select PAHs (benzo(a)pyrene, benzo(g,h,i)perylene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3-c,d)pyrene and phenanthrene) in the background soil samples were higher than those measured in the coal samples, and therefore, deposition of coal dust containing these constituents on soil in the Study Area, would not result in an increase in soil concentrations, and therefore would not result in increased exposures and associated risks to receptors in the Study Area. The practice of summing risks for the Project and Baseline Scenarios is used to evaluate increased environmental concentrations resulting from the project. As has been explained in the HHRA report, deposition of coal dust deposited onto soil or vegetation is not expected to result in increased soil/vegetation concentrations for the coal constituents that had higher concentrations in the background soil than in the coal. In fact, for these parameters, it is impossible that the concentrations of these constituents could slightly decrease in soil/vegetation from this pathway. As an example, the mean concentrations of lead in the coal and in background soil are 2 mg/kg and 22 mg/kg, respectively. The deposition of 2 mg/kg lead in coal dust onto 22 mg/kg lead in background soil does not result in 24 mg/kg lead in soil. In fact, if anything, the soil concentration of lead would slightly decrease following deposition. Despite this, Cumulative Scenario risk estimates have been estimated and presented in the HHRA for COPCs with higher background soil concentrations than those measured in the coal.	Based on the rationale provided, the Cumulative Scenario HQs for these COPCs, which have been estimated by adding the Baseline Scenario HQs to the Project Scenario HQs, are considered to be an overestimate of actual risks.
	Risk characterization uses the results of the previous assessments and assumptions considered in the HHRA (e.g., exposure assessment, toxicity assessment). Therefore the uncertainties of the previous sections are compounded in the risk characterization stage of the HHRA.	Due to the conservatism in the previous sections, it is considered likely that potential risks due to the Project have been over-estimated.

As summarized in the above table, the conservative approach used to estimate exposures and associated risks to the receptors of concern will, overall, tend to overestimate exposures and risks.

7.1 Sensitivity Analysis

7.1.1 Use of Mean Coal Concentrations

As discussed in earlier sections of the report, exposure modeling to determine COPC concentrations in coal resulting from the Project was conducted using the concentrations of the individual coal constituents determined through laboratory analysis. The arithmetic mean concentrations of the coal constituents were calculated and used to represent COPC concentrations in coal. It is recognized that other statistics may be used, however, in most cases there was not an appreciable difference between mean and 95% UCLM (the 95 percentile upper bound of the mean) concentrations (see Appendix VI of HHRA).

To investigate the sensitivity of using mean concentrations, a comparison was conducted to determine the overall impact on the results of the HHRA. With the exception of barium, sodium, phosphorus and acenaphthalene, all parameters had less than a 20% relative percent difference (RPD) between mean and 95% UCLM concentrations with most substances having less than a 10% RPD. For the purposes of sensitivity analysis, the parameter with the highest RPD (i.e., barium) was selected for sensitivity analysis along with the COPCs that have a greater than 10% RPD that may be of the highest toxic potency (the carcinogenic PAHs benzo(a)anthrance and benzo(b)fluoranthene, and the metals mercury and uranium).

The maximum North Delta residential receptor was assessed for the Project Scenario using a toddler for the evaluation of non-carcinogenic risks and an adult for the evaluation of carcinogenic risks. The differences in the mean and 95% UCLM concentrations for the COPCs selected for evaluation in the sensitivity analysis are:

- The arithmetic mean of barium was 351 µg/g and the 95% UCLM was 516.9 µg/g;
- benzo(a)anthracene had an arithmetic mean of 0.58 µg/g compared to a 95% UCLM of 0.65 µg/g;
- benzo(b)fluoranthene had an arithmetic mean of 0.85 µg/g compared to a 95% UCLM of 0.97 µg/g;
- uranium had an arithmetic mean of 0.35 µg/g and a 95% UCLM of 0.39 µg/g; and,
- mercury had an arithmetic mean of 0.08 µg/g and 95% UCLM of 0.086 µg/g.

Multimedia concentrations of these COPCs were determined using 95% UCLM coal concentrations and associated risks were compared to those estimated in the Section 6 using arithmetic mean concentrations.

The results of the sensitivity analysis indicated that both carcinogenic and non-carcinogenic risks remained acceptable (i.e., below the Health Canada negligible risk levels of 0.2 and 1E-05, respectively) when the 95%

UCLM coal concentrations were used. For non-carcinogenic risks, the overall hazard quotient (HQ) increased by 3% for barium (HQ= 0.118 compared to 0.115), by 10% for mercury (HQ=0.0063 compare to 0.0057), by 10% for uranium (HQ=0.00025 compared to 0.00022), by 1% for benzo(a)anthracene (HQ=0.000007) and by 1% for benzo(b)fluoranthene (HQ=0.0000047). Use of 95% UCLM coal concentrations also resulted in increases (over the ILCRs predicted based on mean coal concentrations) in the overall incremental lifetime cancer risk (ILCR) of 10% for benzo(a)anthracene (ILCR= 1.5E-09 compared to 1.5E-10), 12% for benzo(b)fluoranthene (ILCR=7.4E-10 compared to 1.0E-10) for an overall increase of 1% to the additive carcinogenic PAH mixture (ILCR=1.9E-08 compared to 1.9E-08).

As demonstrated, the use of arithmetic mean concentrations of the COPCs measured in the coal samples over 95% UCLMs does not change the results of the HHRA.

7.1.2 TRVs from Other Sources

The TRVs selected for use in the HHRA are presented in Section 5 of the report, with details on the TRVs reviewed as part of the Toxicity Assessment, as well as rationale for the TRVs selected for use, provided in Appendix V. For select COPCs, including arsenic, mercury, benzene and PCBs, the sensitivity of using TRVs from other sources, was investigated to determine the overall impact on the results of the HHRA. The impact of using the TRVs from other sources on the results of the HHRA is summarized as follows:

- For arsenic, the OEHHA chronic oral REL of 0.0035 µg/kg bw/day was considered and compared to the US EPA oral RfD of 0.3 µg/kg bw/day used in the HHRA. Use of the OEHHA REL would result in an approximate 100 fold increase in the HQs estimated for arsenic for oral exposures (i.e., for the soil and vegetation pathways). The maximum HQ estimated for arsenic for oral exposures is 0.03, and was estimated for the toddler receptor, maximum rail corridor residential receptor. Use of the OEHHA chronic REL would therefore result in an approximate HQ of 3, which exceeds the Health Canada negligible risk level of 0.2. It is noted that the Baseline Scenario HQ estimated for arsenic for oral exposures to the maximum rail corridor residential receptor toddler, is 1.4, and thus, use of the OEHHA REL would result in a Baseline Scenario HQ of approximately 140.

- For mercury, a TC of 0.06 µg/m3, as previously recommended by Health Canada, was considered and compared to the US EPA RfC of 0.3 µg/m3 used in the HHRA. Use of a TC of 0.06 µg/m3 would result in an approximate order of magnitude increase in the HQs estimated for mercury for inhalation exposures. The maximum HQ estimated for mercury for inhalation exposure is 3.4E-05 for the toddler receptor, maximum rail corridor residential receptor. If this HQ was to increase by an order of magnitude (i.e., 3.4E-04), it will remain well below the Health Canada negligible risk level of 0.2.
- For benzene, both the ATSDR chronic inhalation MRL of 9.6 µg/m3 and the OEHHA chronic inhalation REL of 3 µg/m3 were considered and compared to the US EPA RfC of 30 µg/m3 used in the HHRA. Use of the ATSDR and OEHHA TRVs would result in up to an order of magnitude increase in the HQs estimated for the chronic inhalation of benzene. The maximum HQ of 6.5E-05 estimated for the chronic inhalation of benzene was for the industrial receptor. If this HQ was to increase by an order of magnitude (i.e., 6.5E-04), it would remain well below the Health Canada negligible risk level of 0.2.
- For dioxin-like PCBs, the US EPA RfD of 7 x 10-7 µg/kg bw/day for 2,3,7,8 - tetrachlorodibenzo-p-dioxin was considered and compared to the Health Canada TDI of 2.3 x 10-6 µg/kg bw/day used in the HHRA. Use of the US EPA RfD would result in less than order of magnitude increase in the HQs estimated for PCBs for oral exposures (i.e., for the soil and vegetation pathways). The maximum HQ estimated for PCBs for oral exposures is 3.8E-04, and was estimated for the toddler receptor, maximum rail corridor residential receptor. Use of the US EPA RfD would therefore result in an HQ of less than 3.8E-03, which is well below the Health Canada negligible risk level of 0.2.

As demonstrated, the use of the TRVs for these COPCs from other sources would not result in unacceptable risks for the COPCs from Project emissions, except in the case of arsenic. As noted above, if the OEHHA oral REL was used for arsenic, the maximum Project Scenario HQ for oral exposures would be approximately 3; however, the maximum Baseline Scenario HQ for oral exposures would be 140, resulting in a Cumulative Scenario HQ of 143. Regardless of the TRV that is used, the Project Scenario contributes negligibly to the overall oral exposures and risks, with approximately 98% of the HQ attributable to the Baseline Scenario (i.e., background concentrations). As such, use of the OEHHA oral REL for arsenic would not change the concentrations of the HHRA.

8 SUMMARY AND CONCLUSIONS OF THE HHRA

The HHRA has been conducted using methods and guidance recommended by Health Canada, and using a series of conservative assumptions that will tend to overpredict exposures, and therefore risks, to the identified receptors of concern. Despite the conservative approach, no unacceptable risks have been predicted for the receptors identified to have the potential to be exposed to the maximum Project emissions, including the maximum North Delta residential receptor, the maximum rail corridor residential receptor and the industrial receptor. Characterization of the exposures and risks for these receptors is protective of lesser exposed individuals, including commercial workers, urban park users, agricultural receptors and people involved in fishing activities in the Study Area. On this basis, no unacceptable risks have been predicted for these receptor groups.

In summary, no unacceptable health risks are predicted for exposures to the Project emissions in the Study Area.

9 NOTICE TO READER

This report has been prepared and the work referred to in this report have been undertaken by SNC-Lavalin Inc. (SNC-Lavalin) for the exclusive use of Fraser Surrey Docks LP (FSD), who has been party to the development of the scope of work and understands its limitations. The methodology, findings, conclusions and recommendations in this report are based solely upon the scope of work and subject to the time and budgetary considerations described in the proposal and/or contract pursuant to which this report was issued. Any use, reliance on, or decision made by a third party based on this report is the sole responsibility of such third party. SNC-Lavalin accepts no liability or responsibility for any damages that may be suffered or incurred by any third party as a result of the use of, reliance on, or any decision made based on this report.

The findings, conclusions and recommendations in this report (i) have been developed in a manner consistent with the level of skill normally exercised by professionals currently practicing under similar conditions in the area, and (ii) reflect SNC-Lavalin's best judgment based on information available at the time of preparation of this report. No other warranties, either expressed or implied, are made as to the professional services provided under the terms of our original contract and included in this report. The findings and conclusions contained in this report are valid only as of the date of this report and may be based, in part, upon information provided by others. If any of the information is inaccurate, new information is discovered, site conditions change or applicable standards are amended, modifications to this report may be necessary. The results of this assessment should in no way be construed as a warranty that the subject site is free from any and all contamination.

Any soil and rock descriptions in this report and associated logs have been made with the intent of providing general information on the subsurface conditions of the site. This information should not be used as geotechnical data for any purpose unless specifically addressed in the text of this report. Groundwater conditions described in this report refer only to those observed at the location and time of observation noted in the report.

This report must be read as a whole, as sections taken out of context may be misleading. If discrepancies occur between the preliminary (draft) and final version of this report, it is the final version that takes precedence. Nothing in this report is intended to constitute or provide a legal opinion.

The contents of this report are confidential and proprietary. Other than by FSD, copying or distribution of this report or use of or reliance on the information contained herein, in whole or in part, is not permitted without the express written permission of FSD and SNC-Lavalin.

10 REFERENCES

Ahrens and Morrisey, 2005. Biological effects of unburnt coal in the marine environment. Oceanography and Marine Biology: An Annual Review. 2005, 43, 69-122.

ATSDR, 2004. Toxicological Profile for Cobalt. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

ATSDR, 2007. Toxicological Profile for Xylenes. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

ATSDR, 2010a. Toxicological Profile for Boron. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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

ATSDR, 2012a. Toxicological Profile for Cadmium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

ATSDR, 2012b. Toxicological Profile for Chromium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

ATSDR, 2012c. Toxicological Profile for Vanadium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

ATSDR, 2013. Toxicological Profile for Uranium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

BC Ministry of Environment (MoE). 2013. Provincial Air Quality Objective Information Sheet. British Columbia Ambient Air Quality Objectives. Environmental Standards Branch.

BC Ministry of Environment (MoE). 2014a. *Contaminated Sites Regulation* (CSR), B.C. Reg. 375/96, including amendments up to B.C. Reg. 4/2014, January 31, 2014.

BC Ministry of Environment (MoE). 2014b. Procedure 8: Definitions and Acronyms for Contaminated Sites. Environmental Protection Division. January 14, 2014.

Canadian Council of Ministers of the Environment (CCME). 1999. National Ambient Air Quality Objectives: Process and Status. In: Canadian Environmental Quality Guidelines. 1999. CCME, Winnipeg, MB.

Canadian Council of Ministers of the Environment (CCME). 2000. Canada-Wide Standards for Particulate Matter and Ozone. 2000. Quebec City, QC

Canadian Council of Ministers of the Environment (CCME). 2012. Canadian Ambient Air Quality Standards (CAAQS) for Fine Particulate Matter (PM2.5) and Ozone. 2012. Quebec City, QC

Canadian Environmental Protection Agency (CEPA), 1999a. Priority Substances List Report, Acetaldehyde. May 2000.

Canadian Environmental Protection Agency (CEPA), 1999b. Priority Substances List Report, 1,3-Butadiene. Revised August 2000.

Health Canada. 2006. Residential Indoor Air Quality Guideline: Formaldehyde. Health Canada, Ottawa, ON. Available at: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/air/formaldehyde-eng.pdf.

Health Canada, 2010a. Federal Contaminated Site Risk Assessment in Canada – Part V: Guidance on Complex Human Health Detailed Quantitative Risk Assessment. Version 1.0. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

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

Health Canada, 2011. Residential Indoor Air Quality Guideline: Toluene. Health Canada, Ottawa, ON. Available on-line at http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/air/toluene/toluene-eng.pdf.

Health Canada, 2012. Federal Contaminated Site Risk Assessment in Canada – Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). Version 2.0. September 2010, revised 2012. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013a. Residential Indoor Air Quality Guideline: Naphthalene. Health Canada, Ottawa, ON. Available on-line at: http://www.hc-sc.gc.ca/ewh-semt/pubs/air/naphthalene/index-eng.php

Health Canada, 2013b. Toxicological Reference Doses obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

Jaffe et al., 2014. Jaffe D, Hof G, Malashanka S, Putz J, Thayer J, Fry J, Ayres B, Pierce JR. Diesel particulate matter emission factors and air quality implications from in–service rail in Washington State, USA. Atmospheric Pollution Research, 5 (2014) 344-351.

Jia and Batterman, 2010. Chungrong Jia, Stuart Batterman. A Critical Review of Naphthalene Sources and Exposures in Indoor and Outdoor Air. Int J Environ Res Public Health. Jul 2010; 7(7): 2903–2939.

Levelton Consultants Ltd. (Levelton). 2014. Fraser Surrey Docks Direct Coal Transfer Facility Draft Air Quality Assessment.

National Sanitation Foundation (NSF) International. 2006. NSF Adipic Acid Standard. Published November 1, 2006. Available on-line at: http://www.techstreet.com/nsf/products/1287485#jumps.

National Sanitation Foundation (NSF) International. 2010. NSF Lanthanum Carbonate – 2010. Oral Risk Assessment Document. Published November 1, 2010. Available on-line at: http://www.techstreet.com/nsf/products/1754626?product_id=1754626#jumps

OEHHA, 2001. California Office of Environmental Health Hazard Assessment. Chronic Toxicity Summary, Selenium and Selenium Compounds (other than hydrogen selenide). December 2001. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=476.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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, 2009. California Office of Environmental Health Hazard Assessment. 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, 2014. California Office of Environmental Health Hazard Assessment. Technical Support Document for Non-Cancer RELs. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. June 2014. Available at http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=139.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

SNC-Lavalin Inc., Triton Environmental Consultants Ltd., Levelton Consultants Inc., 2013. Draft Environmental Impact Assessment for the Direct Coal Transfer Facility. Fraser Surrey Docks. Submitted to Port Metro Vancouver. October 17, 2013.

Statistics Canada. 2014. Census Database. Statistics Canada. Government of Canada. Available at http://www12.statcan.gc.ca/census-recensement/index-eng.cfm?tz=120308.

Texas Commission on Environmental Quality (TCEQ). 2014. Acrolein. CAS Registry Number: 107-02-8. Final Development Support Document. March 2014. Available at: http://www.tceq.com/assets/public/implementation/tox/dsd/final/mar2014/acrolein_107-02-8_revised.pdf

Triton Environmental Consultants (Triton). 2013a. Overview discussion of potential toxicity of unburnt coal. Prepared for Fraser Surrey Docks. Triton, January 2013.

Triton Environmental Consultants (Triton). 2013b. Evaluation of Bekaert Access Road Option 2 at Fraser Surrey Docks (FSD). Triton, June 5, 2013.

United States Environmental Protection Agency (US EPA). 1986. Procedures for Estimating Probability of a Nonattainment of PM_{10} NAAQS Using Total Suspended Particulate Matter or PM10 Data. EPA450/4-86-017. December 1986.

United States Environmental Protection Agency (US EPA). 1990 and 1994a. IRIS Summary of Propionaldehyde (CASRN 75-56-9). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0403.htm.

United States Environmental Protection Agency (US EPA), 1992 and 1994b. IRIS Summary of Epichlorohydrin (CASRN 106-89-8). US EPA, Washington, DC Available at: http://www.epa.gov/iris/subst/0050.htm.

United States Environmental Protection Agency (US EPA). 1993a. IRIS Summary of Manganese (CASRN 7439-96-5), US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0144.htm.

United States Environmental Protection Agency (US EPA). 1993b and 1998. IRIS Summary of Arsenic (CASRN 7440-38-2). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0278.htm.

United States Environmental Protection Agency (US EPA). 1995. IRIS Summary of Mercury, elemental (CASRN 7439-97-6). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0370.htm .

United States Environmental Protection Agency (US EPA). 1991 and 1996a. IRIS Summary of Hexachlorobenzene (CASRN 118-74-1). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0374.htm.

United States Environmental Protection Agency (US EPA).1996b. IRIS Summary of Strontium (CASRN: 7440-24-6). US EPA, Washington, DC. Available at http://www.epa.gov/iris/subst/0550.htm.

United States Environmental Protection Agency (US EPA). 1998a. IRIS Summary of Beryllium (CASRN 7440-41-7). USE EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0012.htm.

United States Environmental Protection Agency (US EPA). 1998b. IRIS Summary of Chromium VI (CASRN 18540-29-9). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0144.htm.

United States Environmental Protection Agency (US EPA). 2003a. IRIS Summary of Diesel Engine Exhaust (CASRN N.A.). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0642.htm.

United States Environmental Protection Agency (US EPA), 2003b. IRIS Summary of Benzene (CASRN 71-43-2). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0276.htm.

United States Environmental Protection Agency (US EPA). 2005. Human Health Risk Assessment Protocol for Hazardous Waste and Combustion Facilities. Office of Solid Waste and Emergency Response (5305W).EPA530-R-05-006, September 2005. Available at: www.epa.gov/osw.

United States Environmental Protection Agency (US EPA). 2006. Provisional Peer Reviewed Toxicity Values for Epichlorohydrin. National Center for Environmental Assessment. Office of Research and Development, US EPA, Cincinnati, OH.

United States Environmental Protection Agency (US EPA). 2008a. National Ambient Air Quality Standards for Lead. Available at http://www.epa.gov/air/criteria.html.

United States Environmental Protection Agency (US EPA). 2008b. IRIS Summary of Propionaldehyde (CASRN 123-38-6). Available at: http://www.epa.gov/iris/subst/1011.htm.

United States Environmental Protection Agency (US EPA). 2009a. Provisional Peer Reviewed Toxicity Values for Benzene (CASRN 71-43-2).

United States Environmental Protection Agency (US EPA). 2009b. Provisional Peer Reviewed Toxicity Values for Vanadium and Its Soluble Inorganic Compounds Other Than Vanadium Pentoxide. National Center for Environmental Assessment. Office of Research and Development, US EPA, Cincinnati, OH.

Wilson, R. and Richardson, G.M. 2013. Lead (Pb) is now a non-threshold substance: how does this affect soil quality guidelines? Human and Ecological Risk Assessment: An International Journal. 19(5):1152-1171.

World Health Organization (WHO), 2000. Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91, Copenhagen.

World Health Organization (WHO), 2006. WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulphur Dioxide. Global Update 2005. Summary of Risk Assessment. WHO Press, World Health Organization, Geneva.

World Health Organization (WHO), 2010. WHO Guidelines for Indoor Air Quality, Selected Pollutants. The WHO European Centre for Environment and Health, Bonn Office, ISBN 978 92 890 0213 4. 2010. Available at http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf.

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

APPENDIX I

SNC·LAVALIN

Risk Estimates

Table I-1A: Acute Inhalation Risk Estimates for All Receptors **Scenario: Baseline**

Chemical	Acute TRV ($\mu g/m^3$)		Exposure Concentration ($\mu g/m^3$)[a,b]	Averaging Time	Hazard Quotient
Carcinogenic PAHs					
Benzo(a)pyrene	--		--	--	--
Benzo(a)anthracene	--		--	--	--
Benzo(b)fluoranthene	--		--	--	--
Benzo(g,h,i)perylene	--		--	--	--
Benzo(k)fluoranthene	--		--	--	--
Chrysene	--		--	--	--
Dibenzo(a,h)anthracene	--		--	--	--
Fluoranthene	--		--	--	--
Indeno(1,2,3-cd)pyrene	--		--	--	--
Phenanthrene	--		--	--	--
Non-Carcinogenic PAHs[c]					
Acenaphthene	--		--	--	--
Acenaphthylene	--		--	--	--
Anthracene	--		--	--	--
Fluorene	--		--	--	--
Fluoranthene	--		--	--	--
Naphthalene	--		--	--	--
2-Methylnaphthalene	--		--	--	--
Pyrene	--		--	--	--
Metals & Metalloids[d]					
Aluminum	--		--	--	--
Antimony	--		--	--	--
Arsenic	1 hour REL:	0.2	7.3E-03	1 hour	3.6E-02
Barium	--		--	--	--
Beryllium	--		--	--	--
Boron	Acute MRL:	300	NA	--	--
Cadmium	Acute MRL:	0.03	1.9E-02	24 hour	6.3E-01
Total Chromium	--		--	--	--
Cobalt	--		--	--	--
Copper	1 hour REL:	100	1.1E-01	1 hour	1.1E-03
Indium	--		--	--	--
Iron	--		--	--	--
Lanthanum	--		--	--	--
Lead	--		--	--	--
Manganese	8 hour REL:	0.17	3.2E-02	1 hour	1.9E-01
Mercury	1 hour REL:	0.6	NA	--	--
Molybdenum	--		--	--	--
Nickel	1 hour REL:	0.2	1.4E-02	1 hour	6.9E-02
Selenium	--		--	--	--
Strontium	--		--	--	--
Tin	--		--	--	--
Titanium	--		--	--	--
Uranium	--		--	--	--
Vanadium	1 hour REL:	30	2.7E-02	1 hour	8.9E-04
Zinc	--		--	--	--
VOCs[c]					
Acetaldehyde	1 hour REL:	470	3.35	1 hour	7.1E-03
Acrolein	1 hour ReV	11	0.2	1 hour	1.8E-02
	1 hour REL	2.5	0.2	1 hour	8.0E-02
Benzene	Subchronic PPRTV:	80	2.4E+00	24 hour	3.0E-02
1,3-Butadiene	1 hour REL:	660	1.0E+00	1 hour	1.6E-03
Ethylbenzene	Acute MRL:	21700	2.0E+00	24 hour	9.1E-05
Ethylene	--		--	--	--
Formaldehyde	30 min exposure limit:	100	8.76	30 min	8.8E-02
Hexachlorobenzene	--		--	--	--
n-Hexane	--		--	--	--
Propionaldehyde	--		--	--	--
Propylene (1-Propene)	--		--	--	--
Toluene	8 hour exposure limit:	15000	2.7E+01	1 hour	1.8E-03
2,2,4-Trimethylpentane	--		--	--	--
Styrene	1 hour REL:	21000	2.4E+01	1 hour	1.1E-03
Xylenes	Acute (2 hour) MRL:	8700	1.8E+01	1 hour	2.1E-03
Dust Palliatives Chemical Constituents					
Epichlorohydrin[a]	Acute REL:	1300	NA	--	--
Others[c]					
Polychlorinated Biphenyls (PCB)	--		--	--	--
Sulfate	1 hour REL	120	7.3E+00	1 hour	6.1E-02
Criteria Air Contaminants					
CO	1 hour AQO:	14300	6.2E+02	1 hour	4.3E-02
	8 hour AQO:	5500	5.6E+02	8 hour	1.0E-01
NO_2	1 hour AQO:	200	6.6E+01	1 hour	3.3E-01
SO_2	1 hour AQO:	450	2.8E+01	1 hour	6.2E-02
	24 hour AQO:	20	1.8E+01	24 hour	9.0E-01
$PM_{2.5}$	24 hour AQO:	25	1.2E+01	24 hour	4.8E-01
PM_{10}	24 hour AQO:	50	2.7E+01	24 hour	5.4E-01
DPM	--		2.3E+00	24 hour	--
TPM	--		5.6E+01	24 hour	--
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					1.1E-01 – 1.7E-01
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					1.1E-01 – 1.7E-01
Respiratory irritants (acetaldehyde, cadmium, *epichlorohydrin,* formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**					**1.2E+00**

[a] Baseline concentrations based on data from Metro Vancouver's Burnaby South NAPs Super Site.

[b] 1 hour maximum baseline concentrations calculated from 24 hour maximum concentrations using a 2.4 conversion recommended by the Ontario MoE; available at http://allianceforrisk.org/Workshop/CaseStudies/24-hour_sampling/MOE_24_hour_case_study.pdf.

[c] Baseline concentrations calcuated based on data collected over 5 years (2008-2012) (n=294 for VOCs and Naphthalene, n=563 for Sulfate)

[d] Baseline concentrations calculated based on data collected over 2 years (2008-2009 [only data available]) (n=171)

--' not evaluated; no acute TRVs available for parameter

NA - no baseline data available for parameter

** - no baseline data available for italicized COPCs

BOLD: indicates acute inhalation risks > 1.0

Table I-1B: Acute Inhalation Risk Estimates for the Maximum North Delta Residential Receptor **Scenario: Project**

Chemical	Acute TRV ($\mu g/m^3$)		Exposure Concentration ($\mu g/m^3$)	Averaging Time	Hazard Quotient
Carcinogenic PAHs					
Benzo(a)pyrene	--		--	--	--
Benzo(a)anthracene	--		--	--	--
Benzo(b)fluoranthene	--		--	--	--
Benzo(g,h,i)perylene	--		--	--	--
Benzo(k)fluoranthene	--		--	--	--
Chrysene	--		--	--	--
Dibenzo(a,h)anthracene	--		--	--	--
Fluoranthene	--		--	--	--
Indeno(1,2,3-cd)pyrene	--		--	--	--
Phenanthrene	--		--	--	--
Non-Carcinogenic PAHs[b]					
Acenaphthene	--		--	--	--
Acenaphthylene	--		--	--	--
Anthracene	--		--	--	--
Fluorene	--		--	--	--
Fluoranthene	--		--	--	--
Naphthalene	--		--	--	--
2-Methylnaphthalene	--		--	--	--
Pyrene	--		--	--	--
Metals & Metalloids[c]					
Aluminum	--		--	--	--
Antimony	--		--	--	--
Arsenic	1 hour REL:	0.2	3.3E-03	1 hour	1.6E-02
Barium	--		--	--	--
Beryllium	--		--	--	--
Boron	Acute MRL:	300	1.6E-02	24 hour	5.5E-05
Cadmium	Acute MRL:	0.03	2.1E-04	24 hour	7.0E-03
Total Chromium	--		--	--	--
Cobalt	--		--	--	--
Copper	1 hour REL:	100	1.3E-02	1 hour	1.3E-04
Indium	--		--	--	--
Iron	--		--	--	--
Lanthanum	--		--	--	--
Lead	--		--	--	--
Manganese	8 hour REL:	0.17	4.4E-02	1 hour	2.6E-01
Mercury	1 hour REL:	0.6	2.7E-04	1 hour	4.5E-04
Molybdenum	--		--	--	--
Nickel	1 hour REL:	0.2	9.1E-03	1 hour	4.5E-02
Selenium	--		--	--	--
Strontium	--		--	--	--
Tin	--		--	--	--
Titanium	--		--	--	--
Uranium	--		--	--	--
Vanadium	1 hour REL:	30	3.1E-02	1 hour	1.0E-03
Zinc	--		--	--	--
VOCs[b]					
Acetaldehyde	1 hour REL:	470	9.2E-01	1 hour	2.0E-03
Acrolein	1 hour ReV:	11	1.5E-01	1 hour	1.4E-02
	1 hour REL	2.5	1.5E-01	1 hour	6.0E-02
Benzene	Subchronic PPRTV	80	1.9E-02	24 hour	2.3E-04
1,3-Butadiene	1 hour REL:	660	7.3E-02	1 hour	1.1E-04
Ethylbenzene	Acute MRL:	21700	6.0E-02	1 hour	2.8E-06
Ethylene	--		--	--	--
Formaldehyde	30 min exposure limit:	100	2.3E+00	30 min	2.3E-02
Hexachlorobenzene	--		--	--	--
n-Hexane	--		--	--	--
Propionaldehyde	--		--	--	--
Propylene (1-Propene)	--		--	--	--
Toluene	8 hour exposure limit:	15000	2.9E-01	1 hour	1.9E-05
2,2,4-Trimethylpentane	--		--	--	--
Styrene	1 hour REL:	21000	1.8E-02	1 hour	8.4E-07
Xylenes	Acute (2 hour) MRL:	8700	6.4E-02	1 hour	7.3E-06
Dust Palliatives Chemical Constituents					
Epichlorohydrin[a]	Acute REL:	1300	2.4E-05	1 hour	1.8E-08
Others[b]					
Polychlorinated Biphenyls (PCB)	--		--	--	--
Sulfate	1 hour REL	120	1.1E+00	1 hour	9.0E-03
Criteria Air Contaminants					
CO	1 hour AQO:	14300	7.6E+01	1 hour	5.3E-03
	8 hour AQO:	5500	4.8E+01	8 hour	8.7E-03
NO_{2**}	1 hour AQO:	200	1.0E+02	1 hour	5.0E-01
SO_2	1 hour AQO:	450	3.6E-01	1 hour	8.0E-04
	24 hour AQO:	20	7.9E-02	24 hour	4.0E-03
$PM_{2.5}$	24 hour AQO:	25	4.9E+00	24 hour	2.0E-01
PM_{10}	24 hour AQO:	50	3.9E+00	24 hour	7.8E-02
DPM	--		3.9E+00	24 hour	--
TPM	--		9.5E+00	24 hour	--
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					3.7E-02 8.4E-02
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					3.7E-02 8.3E-02
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)					5.4E-01

--' Not evaluated; no acute TRVs available for parameter

** - NO2 exposure point concentration includes background based on the method of calculation (ARM method) (Levelton, 2014) and therefore the HQ for the Project+Background (Cumulative)

Table I-1C: Acute Inhalation Risk Estimates for the Maximum Rail Corridor Residential Receptor **Scenario: Project**

Chemical	Acute TRV ($\mu g/m^3$)		Exposure Concentration ($\mu g/m^3$)	Averaging Time	Hazard Quotient
Carcinogenic PAHs					
Benzo(a)pyrene	--		--	--	--
Benzo(a)anthracene	--		--	--	--
Benzo(b)fluoranthene	--		--	--	--
Benzo(g,h,i)perylene	--		--	--	--
Benzo(k)fluoranthene	--		--	--	--
Chrysene	--		--	--	--
Dibenzo(a,h)anthracene	--		--	--	--
Fluoranthene	--		--	--	--
Indeno(1,2,3-cd)pyrene	--		--	--	--
Phenanthrene	--		--	--	--
Non-Carcinogenic PAHs[b]					
Acenaphthene	--		--	--	--
Acenaphthylene	--		--	--	--
Anthracene	--		--	--	--
Fluorene	--		--	--	--
Fluoranthene	--		--	--	--
Naphthalene	--		--	--	--
2-Methylnaphthalene	--		--	--	--
Pyrene	--		--	--	--
Metals & Metalloids[c]					
Aluminum	--		--	--	--
Antimony	--		--	--	--
Arsenic	1 hour REL:	0.2	1.3E-03	1 hour	6.7E-03
Barium	--		--	--	--
Beryllium	--		--	--	--
Boron	Acute MRL:	300	7.4E-03	24 hour	2.5E-05
Cadmium	Acute MRL:	0.03	4.5E-04	24 hour	1.5E-02
Total Chromium	--		--	--	--
Cobalt	--		--	--	--
Copper	1 hour REL:	100	1.3E-02	1 hour	1.3E-04
Indium	--		--	--	--
Iron	--		--	--	--
Lanthanum	--		--	--	--
Lead	--		--	--	--
Manganese	8 hour REL:	0.17	2.0E-02	1 hour	1.2E-01
Mercury	1 hour REL:	0.6	1.4E-04	1 hour	2.3E-04
Molybdenum	--		--	--	--
Nickel	1 hour REL:	0.2	4.1E-03	1 hour	2.0E-02
Selenium	--		--	--	--
Strontium	--		--	--	--
Tin	--		--	--	--
Titanium	--		--	--	--
Uranium	--		--	--	--
Vanadium	1 hour REL:	30	1.1E-02	1 hour	3.6E-04
Zinc	--		--	--	--
VOCs[b]					
Acetaldehyde	1 hour REL:	470	5.0E-01	1 hour	1.1E-03
Acrolein	1 hour ReV:	11	NA	--	--
	1 hour REL	2.5	NA	--	--
Benzene	Subchronic PPRTV:	80	2.6E-03	24 hour	3.2E-05
1,3-Butadiene	1 hour REL:	660	NA	--	--
Ethylbenzene	Acute MRL:	21700	8.4E-02	24 hour	3.9E-06
Ethylene	--		--	--	--
Formaldehyde	30 min exposure limit:	100	1.4E+00	30 min (0.5 hour)	1.4E-02
Hexachlorobenzene	--		--	--	--
n-Hexane	--		--	--	--
Propionaldehyde	--		--	--	--
Propylene (1-Propene)	--		--	--	--
Toluene	8 hour exposure limit:	15000	4.0E-01	1 hour	2.7E-05
2,2,4-Trimethylpentane	--		--	--	--
Styrene	1 hour REL:	21000	1.6E-02	1 hour	7.6E-07
Xylenes	Acute (2 hour) MRL:	8700	NA	--	--
Dust Palliatives Chemical Constituents					
Epichlorohydrin[a]	Acute REL:	1300	1.1E-05	1 hour	8.1E-09
Others[b]					
Polychlorinated Biphenyls (PCB)	--		--	--	--
Sulfate	1 hour REL	120	NA	--	--
Criteria Air Contaminants					
CO	1 hour AQO:	14300	8.3E+01	1 hour	5.8E-03
	8 hour AQO:	5500	5.5E+01	8 hour	1.0E-02
NO_2	1 hour AQO:	200	9.7E+01	1 hour	4.9E-01
SO_2	1 hour AQO:	450	2.9E-01	1 hour	6.4E-04
	24 hour AQO:	20	1.0E-02	24 hour	5.0E-04
$PM_{2.5}$	24 hour AQO:	25	1.5E+00	24 hour	6.0E-02
PM_{10}	24 hour AQO:	50	3.0E+00	24 hour	6.0E-02
DPM	--		5.3E-01	24 hour	--
TPM	--		4.7E+00	24 hour	--
Nasal irritants (acrolein, boron, formaldehyde, toluene, styrene)					1.4E-02
Ocular irritants (acrolein, epichlorohydrin, formaldehyde, toluene)					1.4E-02
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)					5.2E-01

NA - parameter was not a COPC for this receptor (COPC is present in emissions associated with transportion equipment used at FSD's facility)
--' Not evaluated; no acute TRVs available for parameter

Table I-1D: Acute Inhalation Risk Estimates for the Industrial Receptor **Scenario: Project**

Chemical	Acute TRV ($\mu g/m^3$)		Exposure Concentration ($\mu g/m^3$)	Averaging Time	Hazard Quotient
Carcinogenic PAHs					
Benzo(a)pyrene	--		--	--	--
Benzo(a)anthracene	--		--	--	--
Benzo(b)fluoranthene	--		--	--	--
Benzo(g,h,i)perylene	--		--	--	--
Benzo(k)fluoranthene	--		--	--	--
Chrysene	--		--	--	--
Dibenzo(a,h)anthracene	--		--	--	--
Fluoranthene	--		--	--	--
Indeno(1,2,3-cd)pyrene	--		--	--	--
Phenanthrene	--		--	--	--
Non-Carcinogenic PAHs[b]					
Acenaphthene	--		--	--	--
Acenaphthylene	--		--	--	--
Acridine	--		--	--	--
Anthracene	--		--	--	--
Fluorene	--		--	--	--
Fluoranthene	--		--	--	--
Naphthalene	--		--	--	--
2-Methylnaphthalene	--		--	--	--
Pyrene	--		--	--	--
Quinoline	--		--	--	--
Metals & Metalloids[c]					
Aluminum	--		--	--	--
Antimony	--		--	--	--
Arsenic	1 hour REL:	0.2	8.6E-03	1 hour	4.3E-02
Barium	--		--	--	--
Beryllium	--		--	--	--
Boron	Acute MRL:	300	4.2E-02	24 hour	1.4E-04
Cadmium	Acute MRL:	0.03	5.2E-04	24 hour	1.7E-02
Total Chromium	--		--	--	--
Cobalt	--		--	--	--
Copper	1 hour REL:	100	7.8E-02	1 hour	7.8E-04
Indium	--		--	--	--
Iron	--		--	--	--
Lanthanum	--		--	--	--
Lead	--		--	--	--
Manganese	8 hour REL:	0.17	1.1E-01	1 hour	6.5E-01
Mercury	1 hour REL:	0.6	6.0E-04	1 hour	1.0E-03
Molybdenum	--		--	--	--
Nickel	1 hour REL:	0.2	2.3E-02	1 hour	1.2E-01
Selenium	--		--	--	--
Strontium	--		--	--	--
Tin	--		--	--	--
Titanium	--		--	--	--
Uranium	--		--	--	--
Vanadium	1 hour REL:	30	8.5E-02	1 hour	2.8E-03
Zinc	--		--	--	--
VOCs[b]					
Acetaldehyde	1 hour REL:	470	2.5E+00	1 hour	5.3E-03
Acrolein	1 hour ReV:	11	2.1E+00	1 hour	1.9E-01
	1 hour REL:	2.5	2.1E+00	1 hour	8.2E-01
Benzene	Subchronic PPRTV:	80	9.9E-02	24 hour	1.2E-03
1,3-Butadiene	1 hour REL:	660	9.4E-01	1 hour	1.4E-03
Ethylbenzene	Acute MRL:	21700	1.1E-01	1 hour	5.2E-06
Ethylene	--		--	--	--
Formaldehyde	30 min exposure limit:	100	6.2E+00	30 min	6.2E-02
Hexachlorobenzene	--		--	--	--
n-Hexane	--		--	--	--
Propionaldehyde	--		--	--	--
Propylene (1-Propene)	--		--	--	--
Toluene	8 hour exposure limit:	15000	5.5E-01	1 hour	3.6E-05
2,2,4-Trimethylpentane	--		--	--	--
Styrene	1 hour REL:	21000	2.2E-02	1 hour	1.0E-06
Xylenes	Acute (2 hour) MRL:	8700	3.2E-01	1 hour	3.7E-05
Dust Palliatives Chemical Constituents					
Epichlorohydrin[a]	Acute REL:	1300	6.0E-05	1 hour	4.6E-08
Others[b]					
Polychlorinated Biphenyls (PCB)	--		--	--	--
Sulfate	1 hour REL	120	3.5E+00	1 hour	3.0E-02
Criteria Air Contaminants					
CO	1 hour AQO:	14300	2.4E+02	1 hour	1.7E-02
	8 hour AQO:	5500	1.0E+02	8 hour	1.8E-02
NO_{2**}	1 hour AQO:	200	1.1E+02	1 hour	5.6E-01
SO_2	1 hour AQO:	450	9.7E-01	1 hour	2.2E-03
	24 hour AQO:	20	8.9E-02	24 hour	4.4E-03
$PM_{2.5}$	24 hour AQO:	25	5.0E+00	24 hour	2.0E-01
PM_{10}	24 hour AQO:	50	1.8E+01	24 hour	3.7E-01
DPM	--		4.3E+00	24 hour	--
TPM	--		4.7E+01	24 hour	--
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					2.5E-01 8.9E-01
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)** (presented as a range using based on the use of both the TCEQ acute ReV and the OEHHA acute REL for acolein)					2.5E-01 8.9E-01
Respiratory irritants (acetaldehyde, cadmium, epichlorohydrin, formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)					6.7E-01

--' Not evaluated; no acute TRVs available for parameter

** - NO2 exposure point concentration includes background based on the method of calculation (ARM method) (Levelton, 2014) and therefore the HQ for the Project+Background (Cumulative)

Table I-1E: Cumulative Acute Inhalation Risk Estimates for the Maximum North Delta Residential Receptor Scenario: Cumulative

Chemical	Baseline Hazard Quotient[a]	Project Hazard Quotient	Cumulative Hazard Quotient[b]
Carcinogenic PAHs			
Benzo(a)pyrene	--	--	--
Benzo(a)anthracene	--	--	--
Benzo(b)fluoranthene	--	--	--
Benzo(g,h,i)perylene	--	--	--
Benzo(k)fluoranthene	--	--	--
Chrysene	--	--	--
Dibenzo(a,h)anthracene	--	--	--
Fluoranthene	--	--	--
Indeno(1,2,3-cd)pyrene	--	--	--
Phenanthrene	--	--	--
Non-Carcinogenic PAHs			
Acenaphthene	--	--	--
Acenaphthylene	--	--	--
Anthracene	--	--	--
Fluorene	--	--	--
Fluoranthene	--	--	--
Naphthalene	--	--	--
2-Methylnaphthalene	--	--	--
Pyrene	--	--	--
Metals & Metalloids			
Aluminum	--	--	--
Antimony	--	--	--
Arsenic	3.6E-02	1.6E-02	5.3E-02
Barium	--	--	--
Beryllium	--	--	--
Boron	--	5.5E-05	NC
Cadmium	6.3E-01	7.0E-03	6.4E-01
Total Chromium	--	--	--
Cobalt	--	--	--
Copper	1.1E-03	1.3E-04	1.2E-03
Indium	--	--	--
Iron	--	--	--
Lanthanum	--	--	--
Lead	--	--	--
Manganese	1.9E-01	2.6E-01	4.4E-01
Mercury	ND	4.5E-04	NC
Molybdenum	--	--	--
Nickel	6.9E-02	4.5E-02	1.1E-01
Selenium	--	--	--
Strontium	--	--	--
Tin	--	--	--
Titanium	--	--	--
Uranium	--	--	--
Vanadium	8.9E-04	1.0E-03	1.9E-03
Zinc	--	--	--
VOCs			
Acetaldehyde	7.1E-03	2.0E-03	9.1E-03
Acrolein[c]	1.8E-02	1.4E-02	3.2E-02
Acrolein[d]	8.0E-02	6.0E-02	1.4E-01
Benzene	3.0E-02	2.3E-04	3.0E-02
1,3-Butadiene	1.6E-03	1.1E-04	1.7E-03
Ethylbenzene	2.2E-04	2.8E-06	2.2E-04
Ethylene	--	--	--
Formaldehyde	8.8E-02	2.3E-02	1.1E-01
Hexachlorobenzene	--	--	--
n-Hexane	--	--	--
Propionaldehyde	--	--	--
Propylene (1-Propene)	--	--	--
Toluene	1.8E-03	1.9E-05	1.8E-03
2,2,4-Trimethylpentane	--	--	--
Styrene	1.1E-03	8.4E-07	1.1E-03
Xylenes	2.1E-03	7.3E-06	2.1E-03
Dust Palliatives Chemical Constituents			
Epichlorohydrin[a]	--	1.8E-08	NC
Others			
Polychlorinated Biphenyls (PCB)	--	--	--
Sulfate	6.1E-02	9.0E-03	7.0E-02
Criteria Air Contaminants			
CO	4.3E-02	5.3E-03	4.8E-02
	1.0E-01	8.7E-03	1.1E-01
NO_2****	3.3E-01	****	5.0E-01
SO_2	6.2E-02	8.0E-04	6.3E-02
	9.0E-01	4.0E-03	9.0E-01
$PM_{2.5}$	4.8E-01	2.0E-01	6.8E-01
PM_{10}	5.4E-01	7.8E-02	6.2E-01
DPM	--	--	--
TPM	--	--	--
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[c]	1.1E-01	3.7E-02	1.5E-01
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[d]	1.7E-01	8.4E-02	2.5E-01
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)**[c]	1.1E-01	3.7E-02	1.4E-01
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)**[d]	1.7E-01	8.3E-02	2.5E-01
Respiratory irritants *(acetaldehyde,* cadmium, *epichlorohydrin,* formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**	**1.2E+00**	4.3E-02	**1.2E+00**

[a] Based on baseline air data from Metro Vancouver's Burnaby South NAPs Super Site.

[b] Cumulative Hazard Quotient (HQ) calculated as Baseline HQ + Project HQ

[c] Based on TCEQ acute ReV (1 hour) for acrolein of 11 ug/m3

[d] Based on OEHHA acute REL (1 hour) for acrolein of 2.5 ug/m3

--' not evaluated; no acute TRVs available for parameter

NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

** - no baseline data available for italicized COPCs

**** - HQ for the Project Scenario could not be estimated for NO_2 based on the method of calculation (ARM method) (Levelton, 2014) for 1 hour NO_2 which includes background concentraitons

Table I-1F: Cumulative Acute Inhalation Risk Estimates for the Maximum Rail Corridor Residential Receptor **Scenario: Cumulative**

Chemical	Baseline Hazard Quotient[a]	Project Hazard Quotient	Cumulative Hazard Quotient[b]
Carcinogenic PAHs			
Benzo(a)pyrene	--	--	--
Benzo(a)anthracene	--	--	--
Benzo(b)fluoranthene	--	--	--
Benzo(g,h,i)perylene	--	--	--
Benzo(k)fluoranthene	--	--	--
Chrysene	--	--	--
Dibenzo(a,h)anthracene	--	--	--
Fluoranthene	--	--	--
Indeno(1,2,3-cd)pyrene	--	--	--
Phenanthrene	--	--	--
Non-Carcinogenic PAHs			
Acenaphthene	--	--	--
Acenaphthylene	--	--	--
Anthracene	--	--	--
Fluorene	--	--	--
Fluoranthene	--	--	--
Naphthalene	--	--	--
2-Methylnaphthalene	--	--	--
Pyrene	--	--	--
Metals & Metalloids			
Aluminum	--	--	--
Antimony	--	--	--
Arsenic	3.6E-02	6.7E-03	4.3E-02
Barium	--	--	--
Beryllium	--	--	--
Boron	--	2.5E-05	NC
Cadmium	6.3E-01	1.5E-02	6.5E-01
Total Chromium	--	--	--
Cobalt	--	--	--
Copper	1.1E-03	1.3E-04	1.2E-03
Indium	--	--	--
Iron	--	--	--
Lanthanum	--	--	--
Lead	--	--	--
Manganese	1.9E-01	1.2E-01	3.0E-01
Mercury	ND	2.3E-04	NC
Molybdenum	--	--	--
Nickel	6.9E-02	2.0E-02	9.0E-02
Selenium	--	--	--
Strontium	--	--	--
Tin	--	--	--
Titanium	--	--	--
Uranium	--	--	--
Vanadium	8.9E-04	3.6E-04	1.2E-03
Zinc	--	--	--
VOCs			
Acetaldehyde	7.1E-03	1.1E-03	8.2E-03
Acrolein	--	--	--
Benzene	3.0E-02	3.2E-05	3.0E-02
1,3-Butadiene	--	--	--
Ethylbenzene	2.2E-04	3.9E-06	2.3E-04
Ethylene	--	--	--
Formaldehyde	8.8E-02	1.4E-02	1.0E-01
Hexachlorobenzene	--	--	--
n-Hexane	--	--	--
Propionaldehyde	--	--	--
Propylene (1-Propene)	--	--	--
Toluene	1.8E-03	2.7E-05	1.8E-03
2,2,4-Trimethylpentane	--	--	--
Styrene	1.1E-03	7.6E-07	1.1E-03
Xylenes	--	--	--
Dust Palliatives Chemical Constituents			
Epichlorohydrin[a]	--	8.1E-09	NC
Others			
Polychlorinated Biphenyls (PCB)	--	--	--
Sulfate	--	--	--
Criteria Air Contaminants			
CO	4.3E-02	5.8E-03	4.9E-02
	1.0E-01	1.0E-02	1.1E-01
NO_2	3.3E-01	****	4.9E-01
SO_2	6.2E-02	6.4E-04	6.3E-02
	9.0E-01	5.0E-04	9.0E-01
$PM_{2.5}$	4.8E-01	6.0E-02	5.4E-01
PM_{10}	5.4E-01	6.0E-02	6.0E-01
DPM	--	--	--
TPM	--	--	--
Nasal irritants (*boron,* formaldehyde, toluene, styrene)**	9.1E-02	1.4E-02	1.0E-01
Ocular irritants (*epichlorohydrin,* formaldehyde, toluene)**	8.9E-02	1.4E-02	1.0E-01
Respiratory irritants *(acetaldehyde,* cadmium, *epichlorohydrin,* formaldehyde, NO_2, SO_2, vanadium, sulfate)**	**1.1E+00**	3.1E-02	**1.2E+00**

[a] Based on baseline air data from Metro Vancouver's Burnaby South NAPs Super Site.

[b] Cumulative Hazard Quotient (HQ) calculated as Baseline HQ + Project HQ

[c] Based on TCEQ acute ReV (1 hour) for acrolein of 11 ug/m3

[d] Based on OEHHA acute REL (1 hour) for acrolein of 2.5 ug/m3

--' not evaluated; no acute TRVs available for parameter

NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

** - no baseline data available for italicized COPCs

**** - HQ for the Project Scenario could not be estimated for NO_2 based on the method of calculation (ARM method) (Levelton, 2014) for 1 hour NO_2 which includes background concentraitons

Table I-1G: Cumulative Acute Inhalation Risk Estimates for the Industrial Receptor **Scenario: Cumulative**

Chemical	Baseline	Project	Cumulative
	Hazard Quotient[a]	Hazard Quotient	Hazard Quotient[b]
Carcinogenic PAHs			
Benzo(a)pyrene	--	--	--
Benzo(a)anthracene	--	--	--
Benzo(b)fluoranthene	--	--	--
Benzo(g,h,i)perylene	--	--	--
Benzo(k)fluoranthene	--	--	--
Chrysene	--	--	--
Dibenzo(a,h)anthracene	--	--	--
Fluoranthene	--	--	--
Indeno(1,2,3-cd)pyrene	--	--	--
Phenanthrene	--	--	--
Non-Carcinogenic PAHs			
Acenaphthene	--	--	--
Acenaphthylene	--	--	--
Anthracene	--	--	--
Fluorene	--	--	--
Fluoranthene	--	--	--
Naphthalene	--	--	--
2-Methylnaphthalene	--	--	--
Pyrene	--	--	--
Metals & Metalloids			
Aluminum	--	--	--
Antimony	--	--	--
Arsenic	3.6E-02	4.3E-02	7.9E-02
Barium	--	--	--
Beryllium	--	--	--
Boron	--	1.4E-04	NC
Cadmium	6.3E-01	1.7E-02	6.5E-01
Total Chromium	--	--	--
Cobalt	--	--	--
Copper	1.1E-03	7.8E-04	1.9E-03
Indium	--	--	--
Iron	--	--	--
Lanthanum	--	--	--
Lead	--	--	--
Manganese	1.9E-01	6.5E-01	8.4E-01
Mercury	--	1.0E-03	NC
Molybdenum	--	--	--
Nickel	6.9E-02	1.2E-01	1.9E-01
Selenium	--	--	--
Strontium	--	--	--
Tin	--	--	--
Titanium	--	--	--
Uranium	--	--	--
Vanadium	8.9E-04	2.8E-03	3.7E-03
Zinc	--	--	--
VOCs			
Acetaldehyde	7.1E-03	5.3E-03	1.2E-02
Acrolein[c]	1.8E-02	1.9E-01	2.1E-01
Acrolein[d]	8.0E-02	8.2E-01	9.0E-01
Benzene	3.0E-02	1.2E-03	3.1E-02
1,3-Butadiene	1.6E-03	1.4E-03	3.0E-03
Ethylbenzene	2.2E-04	5.2E-06	2.3E-04
Ethylene	--	--	--
Formaldehyde	8.8E-02	6.2E-02	1.5E-01
Hexachlorobenzene	--	--	--
n-Hexane	--	--	--
Propionaldehyde	--	--	--
Propylene (1-Propene)	--	--	--
Toluene	1.8E-03	3.6E-05	1.9E-03
2,2,4-Trimethylpentane	--	--	--
Styrene	1.1E-03	1.0E-06	1.1E-03
Xylenes	2.1E-03	3.7E-05	2.1E-03
Dust Palliatives Chemical Constituents			
Epichlorohydrin[a]	--	4.6E-08	NC
Others			
Polychlorinated Biphenyls (PCB)	--	--	--
Sulfate	6.1E-02	3.0E-02	9.0E-02
Criteria Air Contaminants			
CO	4.3E-02	1.7E-02	6.0E-02
	1.0E-01	1.8E-02	1.2E-01
NO_2	3.3E-01	****	5.6E-01
SO_2	6.2E-02	2.2E-03	6.4E-02
	9.0E-01	4.4E-03	9.0E-01
$PM_{2.5}$	4.8E-01	2.0E-01	6.8E-01
PM_{10}	5.4E-01	3.7E-01	9.1E-01
DPM	--	--	--
TPM	--	--	--
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[c]	1.1E-01	2.5E-01	3.6E-01
Nasal irritants (acrolein, *boron,* formaldehyde, toluene, styrene)**[d]	1.7E-01	8.9E-01	1.1E+00
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)**[c]	1.1E-01	2.5E-01	3.6E-01
Ocular irritants (acrolein, *epichlorohydrin,* formaldehyde, toluene)**[d]	1.7E-01	8.9E-01	1.1E+00
Respiratory irritants *(acetaldehyde,* cadmium, *epichlorohydrin,* formaldehyde, NO_2, SO_2, vanadium, xylenes, sulfate)**	**1.2E+00**	1.2E-01	**1.3E+00**

[a] Based on baseline air data from Metro Vancouver's Burnaby South NAPs Super Site.

[b] Cumulative Hazard Quotient (HQ) calculated as Baseline HQ + Project HQ

[c] Based on TCEQ acute ReV (1 hour) for acrolein of 11 ug/m3

[d] Based on OEHHA acute REL (1 hour) for acrolein of 2.5 ug/m3

--' not evaluated; no acute TRVs available for parameter

NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

** - no baseline data available for italicized COPCs

**** - HQ for the Project Scenario could not be estimated for NO_2 based on the method of calculation (ARM method) (Levelton, 2014) for 1 hour NO_2 which includes background concentraitons

Table I-2A: Chronic Baseline Inhalation Risk Estimates for All Receptors, Gaseous COPCs **Scenario: Baseline**

Chemical	Chronic Inhalation TRV		Baseline Concentration	Baseline	Non-Cancer Baseline Concentration	Baseline
	RfC/TC	Unit Risk	($\mu g/m^3$)[a,b,c]	Hazard Quotient	($\mu g/m^3$)[a,b,d]	Hazard Quotient
	($\mu g/m^3$)	($\mu g/m^3$)$^{-1}$	(Residential)	(Residential)	(Amortized: Industrial)	(Industrial)
VOCs[d]						
Acetaldehyde	390	5.80E-07	3.35	8.6E-03	0.9045	2.3E-03
Acrolein	2.7	--	0.2	7.4E-02	0.054	2.0E-02
	0.35	--	0.2	5.7E-01	0.054	1.5E-01
Benzene	30	3.30E-06	0.56	1.9E-02	0.1512	5.0E-03
1,3-Butadiene	2	5.90E-06	0.64	3.2E-01	0.1728	8.6E-02
Ethylbenzene	260	--	0.37	1.4E-03	0.0999	3.8E-04
Ethylene	--	--	1.23	--	0.3321	--
Formaldehyde	50	--	8.76	1.8E-01	8.76	1.8E-01
Hexachlorobenzene	--	4.60E-04	NA	NA	NA	NA
n-Hexane	700	--	0.52	7.4E-04	0.1404	2.0E-04
Propionaldehyde	8	--	NA	NA	NA	NA
Propylene (1-Propene)	3000	--	0.42	1.4E-04	0.1134	3.8E-05
Toluene	2300	--	2.4	1.0E-03	0.648	2.8E-04
2,2,4-Trimethylpentane	--	--	0.75	--	0.2025	--
Styrene	92	--	0.14	1.5E-03	0.0378	4.1E-04
Xylenes	180	--	1.2	6.7E-03	0.324	1.8E-03
Criteria Air Contaminants						
CAC	**AAQO (Annual Average) ($\mu g/m^3$)**		**Baseline Concentration ($\mu g/m^3$)[a,b,c]**	**Baseline Hazard Quotient (Residential)**	**Non-Cancer Baseline Concentration ($\mu g/m^3$)[a,b,c (d - for DPM only)]**	**Baseline Hazard Quotient (Industrial)**
CO	--		--	--	--	--
NO_2	40		27	6.8E-01	27	6.8E-01
SO_2	25		4	1.6E-01	4	1.6E-01
$PM_{2.5}$	8	[e]	4.4	5.5E-01	4.4	5.5E-01
	6	[e]	4.4	7.3E-01	4.4	7.3E-01
PM_{10}	20		12	6.0E-01	12	6.0E-01
DPM	5	[f]	0.8	1.6E-01	0.216	4.3E-02

[a] Baseline concentrations based on data from Metro Vancouver's Burnaby South NAPs Super Site.

[b] Annual average baseline concentrations

[c] No amortization conducted for baseline exposures; receptors assumed to be exposed for 24 hours a day, 7 days a week, 52 weeks a year for 80 years

[d] Amortization conducted for exposures for industrial receptor: receptors assumed to be exposed for 10 hours a day, 5 days a week for 48 weeks a year (ET = 2.7)

[e] Hazard quotients estimates using Metro Vancouver's AAQO of 8 ug/m^3, as well as their planning goal of 6 ug/m^3

[f] AAQO are not available for diesel particulate matter (DPM); the US EPA RfC of 5 ug/m^3 was used to estimate non-cancer risks

[g] AAQO are not available for DPM; the California OEHHA (2009) UR of 3E-04 was used to estimate carcinogenic risks

--' no TRV available/risks could not be estimated

NA - no baseline data available for parameter

AAQO - ambient air quality objective

Table I-2B: Chronic Inhalation Risk Estimates for the Maximum North Delta Residential Receptor, Gaseous COPCs **Scenario: Project**

Chemical	Chronic Inhalation TRV		Exposure Concentration	Amortized Exposure Concentration	Project	Project
	RfC/TC	Unit Risk	Non-Cancer	Cancer	Hazard Quotient	ILCR
	($\mu g/m^3$)	$(\mu g/m^3)^{-1}$	$(\mu g/m^3)^{a,b,c}$	$(\mu g/m^3)^{a,b,d}$		
VOCs						
Acetaldehyde	390	5.80E-07	4.7E-03	5.9E-04	1.2E-05	3.4E-10
Acrolein	2.7	--	3.4E-04	--	1.3E-04	--
	0.35	--	3.4E-04	--	9.8E-04	--
Benzene	30	3.30E-06	8.3E-04	1.0E-04	2.8E-05	3.4E-10
1,3-Butadiene	2	5.90E-06	3.1E-04	3.8E-05	1.5E-04	2.3E-10
Ethylbenzene	260	--	7.4E-04	--	2.8E-06	--
Ethylene	--	--	3.5E-04	--	--	--
Formaldehyde	50	--	1.1E-02	--	2.1E-04	--
Hexachlorobenzene	--	4.60E-04	6.8E-10	8.4E-11	--	3.9E-14
n-Hexane	700	--	4.1E-04	--	5.9E-07	--
Propionaldehyde	8	--	2.1E-03	--	2.7E-04	--
Propylene (1-Propene)	3000	--	2.1E-04	--	7.0E-08	--
Toluene	2300	--	3.5E-03	--	1.5E-06	--
2,2,4-Trimethylpentane	--	--	4.5E-04	--	--	--
Styrene	92	--	3.4E-04	--	3.7E-06	--
Xylenes	180	--	7.5E-04	--	4.2E-06	--
Criteria Air Contaminants						
CAC	**AAQO (Annual Average) ($\mu g/m^3$)**		**Exposure Concentration $(\mu g/m^3)^{a,b,c}$**	**Amortized Exposure Concentration Cancer $(\mu g/m^3)^{d}$**	**Project Hazard Quotient**	**Project ILCR**
CO	--		--	--	--	--
NO_2	40		5.0E+00	--	1.3E-01	--
SO_2	25		3.0E-03	--	1.2E-04	--
$PM_{2.5}$	8	[e]	2.0E-01	--	2.5E-02	--
	6	[e]	2.0E-01	--	3.3E-02	--
PM_{10}	20		4.0E-01	--	2.0E-02	--
DPM	5	[f]	1.3E-01	--	2.6E-02	--
	3.0E-04	[g]	--	1.6E-02	--	4.9E-06

[a] Exposure concentrations based on the results of the Levelton (2014) AQA; predicted concentrations from the Project from all sources (coal, agricultural emissions and combustion emissions from transporation equipment, as applicable)

[b] Predicted annual average concentrations (Levelton, 2014)

[c] No amortization conducted for exposures to non-carcinogenic COPCs for a residential receptor; receptors assumed to be exposed for 24 hours a day, 7 days a week for 52 weeks a year

[d] Amortization conducted for exposures to carcinogenic COPCs from Project emissions for a residential receptor based on the lifetime of the Project (10 years) (i.e., amortization for 10 years/80 years) (ET = 0.125)

[e] Hazard quotients estimates using Metro Vancouver's AAQO of 8 ug/m^3, as well as their planning goal of 6 ug/m^3

[f] AAQO are not available for diesel particulate matter (DPM); the US EPA RfC of 5 ug/m^3 was used to estimate non-cancer risks

[g] AAQO are not available for DPM; the California OEHHA (2009) UR of 3E-04 was used to estimate carcinogenic risks

--' no TRV available/risks could not be estimated

AAQO - ambient air quality objective

Table I-2C: Chronic Inhalation Risk Estimates for the Maximum Rail Corridor Residential Receptor, Gaseous COPCs **Scenario: Project**

Chemical	Chronic Inhalation TRV		Exposure Concentration	Amortized Exposure Concentration	Project	Project
	RfC/TC	Unit Risk	Non-Cancer	Cancer	Hazard Quotient	ILCR
	($\mu g/m^3$)	($\mu g/m^3$)$^{-1}$	($\mu g/m^3$)[a,b,c]	($\mu g/m^3$)[a,b,d]		
VOCs						
Acetaldehyde	390	5.80E-07	5.1E-03	6.3E-04	1.3E-05	3.7E-10
Acrolein	2.7	--	Not a COPC	--	--	--
	0.35	--	Not a COPC	--	--	--
Benzene	30	3.30E-06	7.0E-04	8.7E-05	2.3E-05	2.9E-10
1,3-Butadiene	2	5.90E-06	Not a COPC	--	--	--
Ethylbenzene	260	--	8.4E-04	--	3.2E-06	--
Ethylene	--	--	Not a COPC	--	--	--
Formaldehyde	50	--	1.2E-02	--	2.3E-04	--
Hexachlorobenzene	--	4.60E-04	Not a COPC	--	--	--
n-Hexane	700	--	4.3E-04	--	6.2E-07	--
Propionaldehyde	8	--	2.4E-03	--	3.0E-04	--
Propylene (1-Propene)	3000	--	Not a COPC	--	--	--
Toluene	2300	--	4.1E-03	--	1.8E-06	--
2,2,4-Trimethylpentane	--	--	4.0E-04	--	--	--
Styrene	92	--	1.6E-04	--	1.8E-06	--
Xylenes	180	--	7.5E-04	--	4.2E-06	--
Criteria Air Contaminants						
CAC	**AAQO (Annual Average) ($\mu g/m^3$)**		**Exposure Concentration Non-Cancer ($\mu g/m^3$)[a,b,c]**	**Amortized Exposure Concentration Cancer ($\mu g/m^3$)[a,b,c]**	**Project Hazard Quotient**	**Project ILCR**
CO	--		--	--	--	--
NO_2	40		5.9E+00	--	1.5E-01	--
SO_2	25		3.0E-03	--	1.2E-04	--
$PM_{2.5}$	8	[e]	4.1E-01	--	5.1E-02	--
	6	[e]	4.1E-01	--	6.8E-02	--
PM_{10}	20		8.1E-01	--	4.1E-02	--
DPM	5	[f]	1.4E-01	--	2.8E-02	--
	3.0E-04	[g]	--	1.8E-02	--	5.3E-06

[a] Exposure concentrations based on the results of the Levelton (2014) AQA; predicted concentrations from the Project from all sources (coal, agricultural emissions and combustion emissions from transporation equipment, as applicable)

[b] Predicted annual average concentrations (Levelton, 2014)

[c] No amortization conducted for exposures to non-carcinogenic COPCs for a residential receptor; receptors assumed to be exposed for 24 hours a day, 7 days a week for 52 weeks a year

[d] Amortization conducted for exposures to carcinogenic COPCs from Project emissions for a residential receptor based on the lifetime of the Project (10 years) (i.e., amortization for 10 years/80 years)

[e] Hazard quotients estimates using Metro Vancouver's AAQO of 8 ug/m^3, as well as their planning goal of 6 ug/m^3

[f] AAQO are not available for diesel particulate matter (DPM); the US EPA RfC of 5 ug/m^3 was used to estimate non-cancer risks

[g] AAQO are not available for DPM; the California OEHHA (2009) UR of 3E-04 was used to estimate carcinogenic risks

--' no TRV available/risks could not be estimated

AAQO - ambient air quality objective

Table I-2D: Chronic Inhalation Risk Estimates for the Industrial Receptor, Gaseous COPCs **Scenario: Project**

Chemical	Chronic Inhalation TRV		Amortized Exposure Concentration	Amortized Exposure Concentration	Project	Project
	RfC/TC	Unit Risk	Non-Cancer	Cancer	Hazard Quotient	ILCR
	($\mu g/m^3$)	$(\mu g/m^3)^{-1}$	$(\mu g/m^3)^{a,b,c}$	$(\mu g/m^3)^{a,b,c}$		
VOCs						
Acetaldehyde	390	5.80E-07	7.3E-03	2.4E-04	1.9E-05	1.4E-10
Acrolein	2.7	--	3.4E-04	--	1.2E-04	--
	0.35	--	3.4E-04	--	9.6E-04	--
Benzene	30	3.30E-06	2.0E-03	6.6E-05	6.7E-05	2.2E-10
1,3-Butadiene	2	5.90E-06	1.7E-04	5.5E-06	8.5E-05	3.3E-11
Ethylbenzene	260	--	2.8E-04	--	1.1E-06	--
Ethylene	--	--	1.1E-03	--	--	--
Formaldehyde	50	--	5.5E-02	--	1.1E-03	--
Hexachlorobenzene	--	4.60E-04	3.6E-09	1.2E-10	--	5.4E-14
n-Hexane	700	--	7.0E-04	--	1.0E-06	--
Propionaldehyde	8	--	8.2E-04	--	1.0E-04	--
Propylene (1-Propene)	3000	--	6.6E-04	--	2.2E-07	--
Toluene	2300	--	1.4E-03	--	5.9E-07	--
2,2,4-Trimethylpentane	--	--	1.7E-04	--	--	--
Styrene	92	--	1.4E-04	--	1.5E-06	--
Xylenes	180	--	5.2E-04	--	2.9E-06	--
Criteria Air Contaminants						
CAC	**AAQO (Annual Average) ($\mu g/m^3$)**		**Exposure Concentration Non-Cancer $(\mu g/m^3)^{a,b,d}$**	**Amortized Exposure Concentration Cancer $(\mu g/m^3)^{c}$**	**Project Hazard Quotient**	**Project ILCR**
CO	--		--	--	--	--
NO_2	40		2.1E+01	--	5.3E-01	--
SO_2	25		9.5E-03	--	3.8E-04	--
$PM_{2.5}$	8	[d]	7.0E-01	--	8.8E-02	--
	6	[d]	7.0E-01	--	1.2E-01	--
PM_{10}	20		1.8E+00	--	9.0E-02	--
DPM	5	[e]	9.2E-02	--	1.8E-02	--
	3.0E-04	[f]	--	1.1E-02	--	3.3E-06

[a] Exposure concentrations based on the results of the Levelton (2014) AQA; predicted concentrations from the Project from all sources (coal, agricultural emissions and combustion emissions from transporation equipment, as applicable)

[b] Predicted annual average concentrations (Levelton, 2014)

[c] Amortization conducted for exposures for industrial receptor; receptors assumed to be exposed for 10 hours a day, 5 days a week for 48 weeks a year for the lifetime of the Project (10 years)

[d] Hazard quotients estimates using Metro Vancouver's AAQO of 8 ug/m^3, as well as their planning goal of 6 ug/m^3

[e] AAQO are not available for diesel particulate matter (DPM); the US EPA RfC of 5 ug/m^3 was used to estimate non-cancer risks

[f] AAQO are not available for DPM; the California OEHHA (2009) UR of 3E-04 was used to estimate carcinogenic risks

--' no TRV available/risks could not be estimated

AAQO - ambient air quality objective

Table I-2E: Cumulative Chronic Inhalation Risk Estimates for the Maximum North Delta Residential Receptor, Gaseous COPCs **Scenario: Cumulative**

Chemical	Baseline	Project	Cumulative
	Hazard Quotient	Hazard Quotient	Hazard Quotient
VOCs			
Acetaldehyde	8.6E-03	1.2E-05	8.6E-03
Acrolein[a]	7.4E-02	1.3E-04	7.4E-02
Acrolein[b]	5.7E-01	9.8E-04	5.7E-01
Benzene	1.9E-02	2.8E-05	1.9E-02
1,3-Butadiene	3.2E-01	1.5E-04	3.2E-01
Ethylbenzene	1.4E-03	2.8E-06	1.4E-03
Ethylene	--	--	
Formaldehyde	1.8E-01	2.1E-04	1.8E-01
Hexachlorobenzene	NA	8.4E-11	--
n-Hexane	7.4E-04	5.9E-07	7.4E-04
Propionaldehyde	NA	2.7E-04	NC
Propylene (1-Propene)	1.4E-04	7.0E-08	1.4E-04
Toluene	1.0E-03	1.5E-06	1.0E-03
2,2,4-Trimethylpentane	--	--	--
Styrene	1.5E-03	3.7E-06	1.5E-03
Xylenes	6.7E-03	4.2E-06	6.7E-03
Criteria Air Contaminants			
CAC	**Baseline Hazard Quotient**	**Project Hazard Quotient**	**Cumulative Hazard Quotient**
CO	--	--	
NO_2	6.8E-01	1.3E-01	8.0E-01
SO_2	1.6E-01	1.2E-04	1.6E-01
$PM_{2.5}$	5.5E-01	2.5E-02	5.8E-01
	7.3E-01	3.3E-02	7.7E-01
PM_{10}	6.0E-01	2.0E-02	6.2E-01
DPM	1.6E-01	2.6E-02	1.9E-01

--' no TRV available/risks could not be estimated
NA - no baseline data available for parameter
NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

[a] Based on TCEQ chronic ReV for acrolein of 2.7 ug/m3
[b] Based on OEHHA chronic REL for acrolein of 0.35 ug/m3

Table I-2F: Cumulative Chronic Inhalation Risk Estimates for the Maximum Rail Corridor Residential Receptor, Gaseous COPCs **Scenario: Cumulative**

Chemical	Baseline	Project	Cumulative
	Hazard Quotient	Hazard Quotient	Hazard Quotient
VOCs			
Acetaldehyde	8.6E-03	1.3E-05	8.6E-03
Acrolein	--	--	--
Benzene	1.9E-02	2.3E-05	1.9E-02
1,3-Butadiene	3.2E-01	--	--
Ethylbenzene	1.4E-03	3.2E-06	1.4E-03
Ethylene	--	--	--
Formaldehyde	1.8E-01	2.3E-04	1.8E-01
Hexachlorobenzene	--	--	--
n-Hexane	7.4E-04	6.2E-07	7.4E-04
Propionaldehyde	NA	3.0E-04	NC
Propylene (1-Propene)	--	--	--
Toluene	1.0E-03	1.8E-06	1.0E-03
2,2,4-Trimethylpentane	--	--	--
Styrene	1.5E-03	1.8E-06	1.5E-03
Xylenes	6.7E-03	4.2E-06	6.7E-03
Criteria Air Contaminants			
CAC	**Baseline Hazard Quotient**	**Project Hazard Quotient**	**Cumulative Hazard Quotient**
CO	--	--	
NO_2	6.8E-01	1.5E-01	8.2E-01
SO_2	1.6E-01	1.2E-04	1.6E-01
$PM_{2.5}$	5.5E-01	5.1E-02	6.0E-01
	7.3E-01	6.8E-02	8.0E-01
PM_{10}	6.0E-01	4.1E-02	6.4E-01
DPM	1.6E-01	2.8E-02	1.9E-01

--' no TRV available/risks could not be estimated
NA - no baseline data available for parameter
NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

Table I-2G: Cumulative Chronic Inhalation Risk Estimates for the Industrial Receptor, Gaseous COPCs **Scenario: Cumulative**

Chemical	Baseline	Project	Cumulative
	Hazard Quotient	Hazard Quotient	Hazard Quotient
VOCs			
Acetaldehyde	2.2E-03	1.9E-05	2.3E-03
Acrolein[a]	2.0E-02	1.2E-04	2.0E-02
Acrolein[b]	1.5E-01	9.6E-04	1.6E-01
Benzene	4.9E-03	6.7E-05	4.9E-03
1,3-Butadiene	8.3E-02	8.5E-05	8.3E-02
Ethylbenzene	3.7E-04	1.1E-06	3.7E-04
Ethylene	--	--	
Formaldehyde	1.8E-01	1.1E-03	1.8E-01
Hexachlorobenzene	NA	--	--
n-Hexane	1.9E-04	1.0E-06	1.9E-04
Propionaldehyde	NA	1.0E-04	NC
Propylene (1-Propene)	3.6E-05	2.2E-07	3.7E-05
Toluene	2.7E-04	5.9E-07	2.7E-04
2,2,4-Trimethylpentane	--	--	--
Styrene	4.0E-04	1.5E-06	4.0E-04
Xylenes	1.7E-03	2.9E-06	1.7E-03
Criteria Air Contaminants			
CAC	**Baseline Hazard Quotient**	**Project Hazard Quotient**	**Cumulative Hazard Quotient**
CO	--	--	
NO_2	6.8E-01	5.3E-01	**1.2E+00**
SO_2	1.6E-01	3.8E-04	1.6E-01
$PM_{2.5}$	5.5E-01	8.8E-02	6.4E-01
	7.3E-01	1.2E-01	8.5E-01
PM_{10}	6.0E-01	9.0E-02	6.9E-01
DPM	4.2E-02	1.8E-02	5.9E-02

--' no TRV available/risks could not be estimated

NA - no baseline data available for parameter

NC - not calculated; cumulative risks could not be calculated as baseline data was not available for the COPC

BOLD: Indicates HQ > target HQ of 1.0 for CACs

[a] Based on TCEQ chronic ReV for acrolein of 2.7 ug/m3

[b] Based on OEHHA chronic REL for acrolein of 0.35 ug/m3

TABLE I-3A Baseline Scenario Risk Estimates for Maximum North Delta Residential Receptor (Toddler)
(Based on Background Multi-Media Concentrations)

Scenario: Baseline

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04	5.5E-05	7.0E-06	4.33E-09	1.25E-04	1.96E-04	1.34E-05	3.83E-04	4.0E-04
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04	6.5E-05	8.2E-06	5.10E-09	9.00E-05	1.41E-04	6.71E-06	3.04E-04	3.1E-04
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04	9.7E-05	1.2E-05	7.65E-09	2.15E-04	3.37E-04	1.17E-05	6.62E-04	6.7E-04
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA	4.0E-05	5.1E-06	3.19E-09	2.89E-05	4.53E-05	NA	1.20E-04	1.2E-04
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04	3.9E-05	4.9E-06	3.06E-09	5.55E-05	8.70E-05	5.03E-06	1.86E-04	1.9E-04
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA	6.5E-05	8.2E-06	5.10E-09	1.38E-04	2.16E-04	NA	4.27E-04	4.3E-04
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA	9.7E-06	1.2E-06	7.65E-10	5.92E-06	9.28E-06	NA	2.61E-05	2.6E-05
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	1.0E-04	1.3E-05	8.22E-09	5.28E-04	8.27E-04	8.55E-05	1.47E-03	1.6E-03
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03	4.4E-05	5.6E-06	3.44E-09	2.81E-05	4.40E-05	3.19E-05	1.21E-04	1.5E-04
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02	8.4E-05	1.1E-05	6.63E-09	1.08E-03	1.69E-03	2.68E-04	2.87E-03	3.1E-03
Carcinogenic PAH Mixture											4.23E-04	6.57E-03	3.9E-03
Non-carcinogenic PAHs													
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03	1.6E-06	2.1E-07	1.27E-10	4.26E-05	6.68E-05	1.84E-05	1.11E-04	1.3E-04
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03	1.3E-06	1.6E-07	1.02E-10	1.68E-05	2.64E-05	2.68E-06	4.47E-05	4.7E-05
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03	4.8E-06	6.2E-07	3.82E-10	9.05E-05	1.42E-04	6.41E-05	2.38E-04	3.0E-04
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	1.0E-04	1.3E-05	8.22E-09	5.28E-04	8.27E-04	8.55E-05	1.47E-03	1.6E-03
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01	4.8E-06	6.2E-07	1.52E-09	2.92E-04	4.58E-04	1.50E-02	7.55E-04	1.6E-02
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA	2.4E-05	3.1E-06	1.91E-09	6.93E-04	1.09E-03	NA	1.81E-03	1.8E-03
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03	1.1E-04	1.4E-05	8.92E-09	8.32E-04	1.30E-03	8.22E-05	2.26E-03	2.3E-03
Non-carcinogenic PAH Mixture											1.53E-02	6.69E-03	2.2E-02
Metals and Metalloids													
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA	8.2E-04	7.1E-05	6.50E-08	2.07E-02	3.25E-02	NA	5.41E-02	5.4E-02
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04	9.8E-02	2.5E-03	4.61E-06	**4.93E-01**	**7.73E-01**	4.90E-04	**1.37E+00**	**1.4E+00**
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03	1.6E-03	1.4E-04	5.06E-05	3.04E-02	4.76E-02	3.60E-03	7.98E-02	8.3E-02
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07	5.8E-04	5.0E-05	9.12E-06	7.31E-04	1.15E-03	4.30E-05	2.52E-03	2.6E-03
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04	1.3E-03	1.1E-05	1.67E-05	8.07E-02	1.27E-01	1.70E-02	**2.09E-01**	**2.3E-01**
Chromium(III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	6.3E-04	8.3E-05	7.2E-06	3.92E-06	7.86E-05	1.23E-04	1.26E-04	2.96E-04	4.2E-04
Chromium(VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	6.3E-04	8.6E-04	7.4E-05	1.22E-06	8.12E-04	1.27E-03	6.30E-03	3.02E-03	9.3E-03
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05	3.6E-03	3.1E-04	5.63E-05	9.03E-03	1.41E-02	2.60E-04	2.71E-02	2.7E-02
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03	1.3E-03	6.8E-05	1.87E-05	6.59E-02	1.03E-01	3.40E-03	1.71E-01	1.7E-01
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03	1.8E-01	9.1E-04	1.11E-04	1.90E-01	**2.98E-01**	2.13E-02	**6.67E-01**	**6.9E-01**
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03	1.3E-02	1.1E-03	5.39E-03	**3.97E-01**	**6.22E-01**	5.40E-02	**1.04E+00**	**1.1E+00**
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04	2.3E-04	2.0E-06	6.84E-08	7.15E-03	1.12E-02	1.92E-05	1.86E-02	1.9E-02
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03	8.1E-04	6.9E-05	1.27E-07	9.14E-02	1.43E-01	1.23E-02	**2.35E-01**	**2.5E-01**
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03	9.2E-03	7.2E-04	4.54E-03	6.95E-02	1.09E-01	**4.00E-01**	1.93E-01	**5.9E-01**
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04	2.3E-04	2.0E-06	1.14E-08	7.37E-04	1.15E-03	9.00E-06	2.13E-03	2.1E-03
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04	5.5E-04	4.7E-05	4.32E-08	1.72E-01	**2.70E-01**	5.53E-07	**4.42E-01**	**4.4E-01**
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04	5.0E-06	4.3E-07	3.96E-10	1.89E-05	2.96E-05	1.01E-07	5.40E-05	5.4E-05
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA	6.1E-03	5.2E-04	1.43E-05	6.47E-03	1.01E-02	NA	2.32E-02	2.3E-02
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03	1.4E-02	1.2E-03	3.40E-04	9.98E-03	1.56E-02	2.60E-02	4.17E-02	6.8E-02
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02	8.1E-04	7.0E-05	6.37E-08	1.01E-01	1.58E-01	1.21E-05	**2.59E-01**	**2.6E-01**
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04	**2.2E-01**	1.9E-02	1.73E-05	1.10E-01	1.72E-01	2.52E-07	**5.20E-01**	**5.2E-01**
Boron	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02	1.1E-01	9.8E-03	8.94E-06	5.70E-02	8.93E-02	3.40E-05	**2.70E-01**	**2.7E-01**
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA	1.2E-03	1.0E-04	9.49E-08	8.31E-04	1.30E-03	NA	3.44E-03	3.4E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Others													
Hexachlorobenzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PCBs	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sulphate	NA	NA	NA	NA	9.0E-01	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-Methylnaphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives													
Epichlorohydrin	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-3B Project Scneario Risk Estimates for the Maximum North Delta Residential Receptor (Toddler)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.7E-04	2.8E-10	1.3E-06	1.0E-06	3.8E-04	5.9E-08	7.5E-09	4.66E-12	1.7E-07	2.1E-07	6.41E-06	4.49E-07	6.9E-06
Benzo[a]pyrene	6.7E-04	5.1E-10	1.2E-06	1.1E-06	1.2E-05	1.1E-07	1.4E-08	8.60E-12	1.7E-07	2.4E-07	2.05E-07	5.26E-07	7.3E-07
Benzo[b]fluoranthene	1.4E-04	1.1E-10	7.1E-07	3.7E-07	2.6E-04	2.2E-08	2.9E-09	1.77E-12	9.6E-08	7.8E-08	4.42E-06	2.00E-07	4.6E-06
Benzo[g,h,i]perylene	2.2E-04	1.6E-10	2.2E-07	1.9E-07	2.0E-05	3.5E-08	4.5E-09	2.76E-12	2.9E-08	3.9E-08	3.39E-07	1.08E-07	4.5E-07
Benzo[k]fluoranthene	5.7E-04	4.3E-10	1.1E-06	9.7E-07	5.6E-05	9.2E-08	1.2E-08	7.22E-12	1.4E-07	2.1E-07	9.36E-07	4.51E-07	1.4E-06
Chrysene	7.8E-04	5.9E-10	2.1E-06	2.0E-06	1.1E-04	1.3E-07	1.6E-08	9.91E-12	2.9E-07	4.2E-07	1.86E-06	8.53E-07	2.7E-06
Dibenzo[a,h]anthracene	1.7E-04	1.3E-10	1.4E-07	1.2E-07	1.6E-05	2.7E-08	3.5E-09	2.16E-12	1.9E-08	2.6E-08	2.60E-07	7.63E-08	3.4E-07
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04	2.9E-08	3.6E-09	2.25E-12	2.2E-07	2.3E-07	6.51E-06	4.77E-07	7.0E-06
Indeno[1,2,3-c,d]pyrene	5.6E-04	4.3E-10	5.1E-07	4.3E-07	5.4E-05	9.1E-08	1.2E-08	7.14E-12	6.9E-08	9.1E-08	9.09E-07	2.62E-07	1.2E-06
Phenanthrene	4.2E-06	3.2E-12	3.3E-07	6.5E-08	5.2E-04	6.8E-10	8.7E-11	5.39E-14	4.4E-08	1.4E-08	8.72E-06	5.89E-08	8.8E-06
Carcinogenic PAH Mixture											3.06E-05	3.46E-06	2.5E-05
Non-carcinogenic PAHs													
Acenaphthene	3.1E-07	2.4E-13	1.7E-07	9.9E-09	1.2E-04	2.5E-11	3.2E-12	1.99E-15	1.2E-08	1.0E-09	9.71E-07	1.29E-08	9.8E-07
Acenaphthylene	1.1E-07	8.5E-14	4.6E-08	3.4E-09	9.8E-05	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.0E-06	1.5E-12	1.3E-07	3.2E-08	4.2E-04	3.3E-11	4.2E-12	2.58E-15	1.7E-09	6.7E-10	7.04E-07	2.45E-09	7.1E-07
Fluorene	4.3E-07	3.2E-13	8.4E-08	9.5E-09	7.7E-05	5.2E-11	6.6E-12	4.08E-15	8.5E-09	1.5E-09	9.64E-07	1.01E-08	9.7E-07
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04	2.9E-08	3.6E-09	2.25E-12	2.2E-07	2.3E-07	6.51E-06	4.77E-07	7.0E-06
Naphthalene	4.0E-09	3.0E-15	2.7E-07	2.9E-10	1.8E-04	9.7E-13	1.2E-13	3.03E-16	5.5E-08	9.1E-11	1.83E-05	5.55E-08	1.8E-05
2-Methylnaphthalene	7.1E-09	5.4E-15	2.6E-08	2.4E-10	2.1E-05	8.6E-12	1.1E-12	6.77E-16	2.6E-08	3.8E-10	2.61E-06	2.67E-08	2.6E-06
Pyrene	3.1E-04	2.4E-10	3.7E-06	2.7E-06	6.6E-04	5.0E-08	6.4E-09	3.96E-12	5.0E-07	5.8E-07	1.11E-05	1.14E-06	1.2E-05
Non-carcinogenic PAH Mixture											4.11E-05	1.72E-06	4.3E-05
Metals and Metalloids													
Antimony	1.6E-03	1.2E-09	4.7E-05	4.7E-05	4.1E-05	2.5E-06	2.2E-07	1.98E-10	6.3E-05	9.9E-05	6.80E-06	1.65E-04	1.7E-04
Arsenic	2.0E-02	1.5E-08	1.2E-04	1.2E-04	4.1E-04	3.3E-04	8.4E-06	1.54E-08	1.7E-03	2.6E-03	4.11E-04	4.56E-03	5.0E-03
Barium	5.4E+00	4.1E-06	1.2E-01	1.2E-01	1.1E-01	1.3E-04	1.1E-05	4.08E-06	2.5E-03	3.8E-03	1.09E-01	6.45E-03	1.2E-01
Beryllium	3.3E-03	2.5E-09	5.1E-06	4.9E-06	6.9E-05	7.9E-06	6.8E-07	1.24E-07	1.0E-05	1.6E-05	3.47E-03	3.46E-05	3.5E-03
Cadmium	1.7E-03	1.3E-09	1.3E-04	1.3E-04	1.2E-05	1.0E-05	8.6E-08	1.30E-07	6.3E-04	9.8E-04	1.18E-03	1.62E-03	2.8E-03
Chromium(III)	1.5E-03	1.2E-09	1.8E-06	1.7E-06	1.5E-04	5.0E-09	4.3E-10	2.33E-10	4.8E-09	7.3E-09	3.03E-05	1.78E-08	3.0E-05
Chromium(VI)	4.6E-02	3.5E-08	5.3E-05	5.2E-05	4.7E-07	2.5E-04	2.1E-05	3.50E-07	2.4E-04	3.7E-04	4.74E-06	8.75E-04	8.8E-04
Cobalt	1.9E-02	1.5E-08	5.8E-05	5.8E-05	6.3E-05	9.4E-06	8.1E-07	1.47E-07	2.4E-05	3.7E-05	6.32E-04	7.10E-05	7.0E-04
Copper	2.0E-01	1.5E-07	1.2E-02	1.2E-02	6.5E-04	1.1E-05	5.4E-07	1.50E-07	5.3E-04	8.3E-04	6.47E-04	1.37E-03	2.0E-03
Lead	3.4E-02	2.6E-08	2.3E-04	2.3E-04	1.2E-04	2.7E-04	1.4E-06	1.72E-07	3.0E-04	4.6E-04	8.15E-04	1.04E-03	1.8E-03
Manganese	3.0E-01	2.2E-07	1.1E-02	1.1E-02	9.6E-04	1.1E-05	9.1E-07	4.49E-06	3.3E-04	5.2E-04	1.93E-02	8.65E-04	2.0E-02
Molybdenum	7.7E-03	5.9E-09	2.9E-04	2.9E-04	2.7E-05	1.6E-06	1.4E-08	4.89E-10	5.1E-05	8.0E-05	2.28E-06	1.33E-04	1.4E-04
Mercury	1.2E-03	9.1E-10	1.6E-04	1.6E-04	5.8E-06	1.9E-05	1.7E-06	3.04E-09	2.2E-03	3.4E-03	1.93E-05	5.65E-03	5.7E-03
Nickel	6.2E-02	4.7E-08	5.6E-04	5.5E-04	2.0E-04	2.7E-05	2.1E-06	1.34E-05	2.0E-04	3.2E-04	5.74E-02	5.68E-04	5.8E-02
Selenium	9.9E-03	7.5E-09	3.7E-05	3.7E-05	3.2E-05	7.8E-06	6.7E-08	3.77E-10	2.5E-05	3.8E-05	1.62E-06	7.05E-05	7.2E-05
Strontium	3.4E+00	2.6E-06	1.3E+00	1.3E+00	1.1E-02	2.7E-05	2.4E-06	2.16E-09	8.6E-03	1.3E-02	9.28E-06	2.21E-02	2.2E-02
Tin	3.5E-03	2.6E-09	1.6E-05	1.6E-05	1.7E-05	8.4E-09	7.3E-10	6.66E-13	3.2E-08	5.0E-08	4.23E-09	9.12E-08	9.5E-08
Uranium	5.4E-03	4.1E-09	7.0E-06	6.9E-06	1.8E-05	4.4E-05	3.8E-06	1.03E-07	4.8E-05	7.3E-05	4.41E-04	1.68E-04	6.1E-04
Vanadium	1.6E-01	1.2E-07	1.4E-04	1.3E-04	5.4E-04	5.2E-05	4.5E-06	1.22E-06	3.7E-05	5.6E-05	5.39E-03	1.51E-04	5.5E-03
Zinc	2.1E-01	1.6E-07	3.1E-02	3.1E-02	7.1E-04	2.0E-06	1.7E-07	1.59E-10	2.5E-04	3.9E-04	7.14E-07	6.45E-04	6.5E-04
Aluminum	3.5E+01	2.6E-05	2.2E-02	2.1E-02	1.1E-01	5.6E-04	4.8E-05	4.42E-08	2.9E-04	4.4E-04	1.90E-04	1.34E-03	1.5E-03
Boron	2.7E-01	2.0E-07	1.6E-01	1.6E-01	8.8E-04	7.4E-05	6.4E-07	6.79E-10	3.7E-02	5.8E-02	2.92E-06	9.58E-02	9.6E-02
Iron	3.9E+01	2.9E-05	2.4E-02	2.3E-02	1.3E-01	2.3E-04	2.0E-05	1.85E-08	1.2E-04	1.8E-04	7.95E-05	5.62E-04	6.4E-04
Titanium	2.4E+00	1.8E-06	2.0E-03	2.0E-03	7.9E-03	3.9E-06	3.3E-07	3.07E-10	2.8E-06	4.2E-06	1.32E-06	1.12E-05	1.3E-05
Indium	1.4E-09	1.1E-15	6.8E-08	8.7E-11	5.9E-06	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	3.7E-09	2.8E-15	1.7E-07	2.2E-10	1.5E-05	3.6E-14	3.1E-15	2.80E-18	1.4E-09	2.8E-12	1.49E-08	1.40E-09	1.6E-08
Others													
Hexachlorobenzene	8.2E-14	6.2E-20	1.7E-11	0.0E+00	6.8E-10	1.5E-15	1.3E-16	1.16E-19	2.6E-10	0.0E+00	1.26E-09	2.58E-10	1.5E-09
PCBs	3.0E-12	2.3E-18	2.2E-10	1.3E-15	8.4E-09	6.4E-09	5.5E-10	5.04E-13	3.8E-04	3.7E-09	1.84E-03	3.80E-04	2.2E-03
Sulphate	4.6E-07	3.5E-13	2.2E-05	2.8E-08	1.9E-03	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (gas phase) (above ground plant only)	0.0E+00	0.0E+00	1.7E-13	0.0E+00	6.8E-10	0.0E+00	0.0E+00	0.00E+00	2.5E-12	0.0E+00	1.26E-09	2.54E-12	1.3E-09
Hexachlorobenzene (total)	NA	NA	NA	NA	NA	1.5E-15	1.3E-16	1.16E-19	2.6E-10	0.0E+00	2.52E-09	2.61E-10	3.0E-09
Dust Pallatives													
Epichlorohydrin	3.8E-09	2.9E-15	4.7E-08	1.2E-08	1.3E-06	3.1E-12	2.6E-12	2.89E-15	3.2E-08	1.3E-08	1.25E-06	4.48E-08	1.3E-06

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-3C: Cumulative (Background+Project) Scenario Risk Estimates for a Maximum North Delta Residential Receptor, Toddler
(Based on Maximum Multi-Media Concentrations)

Scenario: Cumulative

	BASELINE HQ Air Inhalation	BASELINE HQ Soil/Vegetation	BASELINE HQ All Routes	PROJECT HQ Air Inhalation	PROJECT HQ Soil/Vegetation	PROJECT HQ All Routes	CUMULATIVE HQ Air Inhalation	CUMULATIVE HQ Soil/Vegetation	CUMULATIVE HQ All Routes
Polycyclic Aromatic Hydrocarbons									
Carcinogenic PAHs									
Benzo[a]anthracene	1.3E-05	3.8E-04	4.0E-04	6.4E-06	4.5E-07	6.9E-06	2.0E-05	3.8E-04	4.0E-04
Benzo[a]pyrene	6.7E-06	3.0E-04	3.1E-04	2.1E-07	5.3E-07	7.3E-07	6.9E-06	3.0E-04	3.1E-04
Benzo[b]fluoranthene	1.2E-05	6.6E-04	6.7E-04	4.4E-06	2.0E-07	4.6E-06	1.6E-05	6.6E-04	6.8E-04
Benzo[g,h,i]perylene	NA	1.2E-04	1.2E-04	3.4E-07	1.1E-07	4.5E-07	NA	1.2E-04	1.2E-04
Benzo[k]fluoranthene	5.0E-06	1.9E-04	1.9E-04	9.4E-07	4.5E-07	1.4E-06	6.0E-06	1.9E-04	1.9E-04
Chrysene	NA	4.3E-04	4.3E-04	1.9E-06	8.5E-07	2.7E-06	NA	4.3E-04	4.3E-04
Dibenzo[a,h]anthracene	NA	2.6E-05	2.6E-05	2.6E-07	7.6E-08	3.4E-07	NA	2.6E-05	2.6E-05
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	6.5E-06	4.8E-07	7.0E-06	9.2E-05	1.5E-03	1.6E-03
Indeno[1,2,3-c,d]pyrene	3.2E-05	1.2E-04	1.5E-04	9.1E-07	2.6E-07	1.2E-06	3.3E-05	1.2E-04	1.5E-04
Phenanthrene	2.7E-04	2.9E-03	3.1E-03	8.7E-06	5.9E-08	8.8E-06	2.8E-04	2.9E-03	3.1E-03
Carcinogenic PAH Mixture	4.2E-04	6.6E-03	3.9E-03	3.1E-05	3.5E-06	2.5E-05	4.5E-04	6.6E-03	7.0E-03
Non-carcinogenic PAHs									
Acenaphthene	1.8E-05	1.1E-04	1.3E-04	9.7E-07	1.3E-08	9.8E-07	1.9E-05	1.1E-04	1.3E-04
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.7E-06	4.5E-05	4.7E-05	7.0E-07	2.5E-09	7.1E-07	3.4E-06	4.5E-05	4.8E-05
Fluorene	6.4E-05	2.4E-04	3.0E-04	9.6E-07	1.0E-08	9.7E-07	6.5E-05	2.4E-04	3.0E-04
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	6.5E-06	4.8E-07	7.0E-06	9.2E-05	1.5E-03	1.6E-03
Naphthalene	1.5E-02	7.5E-04	1.6E-02	1.8E-05	5.5E-08	1.8E-05	1.5E-02	7.5E-04	1.6E-02
2-Methylnaphthalene	NA	1.8E-03	1.8E-03	2.6E-06	2.7E-08	2.6E-06	NC	1.8E-03	1.8E-03
Pyrene	8.2E-05	2.3E-03	2.3E-03	1.1E-05	1.1E-06	1.2E-05	9.3E-05	2.3E-03	2.4E-03
Non-carcinogenic PAH Mixture	1.5E-02	6.7E-03	2.2E-02	4.1E-05	1.7E-06	4.3E-05	1.5E-02	6.7E-03	2.2E-02
Metals and Metalloids									
Antimony	NA	5.4E-02	5.4E-02	6.8E-06	1.6E-04	1.7E-04	NC	5.4E-02	5.4E-02
Arsenic	4.9E-04	**1.4E+00**	**1.4E+00**	4.1E-04	4.6E-03	5.0E-03	9.0E-04	**1.4E+00**	**1.4E+00**
Barium	3.6E-03	8.0E-02	8.3E-02	1.1E-01	6.5E-03	1.2E-01	1.1E-01	8.6E-02	2.0E-01
Beryllium	4.3E-05	2.5E-03	2.6E-03	3.5E-03	3.5E-05	3.5E-03	3.5E-03	2.6E-03	6.1E-03
Cadmium	1.7E-02	**2.1E-01**	**2.3E-01**	1.2E-03	1.6E-03	2.8E-03	1.8E-02	**2.1E-01**	**2.3E-01**
Chromium(III)	1.3E-04	3.0E-04	4.2E-04	3.0E-05	1.8E-08	3.0E-05	1.6E-04	3.0E-04	4.5E-04
Chromium(VI)	6.3E-03	3.0E-03	9.3E-03	4.7E-06	8.8E-04	8.8E-04	6.3E-03	3.9E-03	1.0E-02
Cobalt	2.6E-04	2.7E-02	2.7E-02	6.3E-04	7.1E-05	7.0E-04	8.9E-04	2.7E-02	2.8E-02
Copper	3.4E-03	1.7E-01	1.7E-01	6.5E-04	1.4E-03	2.0E-03	4.0E-03	1.7E-01	1.8E-01
Lead	2.1E-02	**6.7E-01**	**6.9E-01**	8.1E-04	1.0E-03	1.8E-03	2.2E-02	**6.7E-01**	**6.9E-01**
Manganese	5.4E-02	**1.0E+00**	**1.1E+00**	1.9E-02	8.6E-04	2.0E-02	7.3E-02	**1.0E+00**	**1.1E+00**
Molybdenum	1.9E-05	1.9E-02	1.9E-02	2.3E-06	1.3E-04	1.4E-04	2.1E-05	1.9E-02	1.9E-02
Mercury	1.2E-02	**2.4E-01**	**2.5E-01**	1.9E-05	5.7E-03	5.7E-03	1.2E-02	**2.4E-01**	**2.5E-01**
Nickel	**4.0E-01**	1.9E-01	**5.9E-01**	5.7E-02	5.7E-04	5.8E-02	**4.6E-01**	1.9E-01	**6.5E-01**
Selenium	9.0E-06	2.1E-03	2.1E-03	1.6E-06	7.0E-05	7.2E-05	1.1E-05	2.2E-03	2.2E-03
Strontium	5.5E-07	**4.4E-01**	**4.4E-01**	9.3E-06	2.2E-02	2.2E-02	9.8E-06	**4.6E-01**	**4.6E-01**
Tin	1.0E-07	5.4E-05	5.4E-05	4.2E-09	9.1E-08	9.5E-08	1.0E-07	5.4E-05	5.4E-05
Uranium	NA	2.3E-02	2.3E-02	4.4E-04	1.7E-04	6.1E-04	NC	2.3E-02	2.4E-02
Vanadium	2.6E-02	4.2E-02	6.8E-02	5.4E-03	1.5E-04	5.5E-03	3.1E-02	4.2E-02	7.3E-02
Zinc	1.2E-05	**2.6E-01**	**2.6E-01**	7.1E-07	6.5E-04	6.5E-04	1.3E-05	**2.6E-01**	**2.6E-01**
Aluminum	2.5E-07	**5.2E-01**	**5.2E-01**	1.9E-04	1.3E-03	1.5E-03	1.9E-04	**5.2E-01**	**5.2E-01**
Boron	NA	NA	NA	2.9E-06	9.6E-02	9.6E-02	NC	NC	NC
Iron	3.4E-05	**2.7E-01**	**2.7E-01**	7.9E-05	5.6E-04	6.4E-04	1.1E-04	**2.7E-01**	**2.7E-01**
Titanium	NA	3.4E-03	3.4E-03	1.3E-06	1.1E-05	1.3E-05	NC	3.5E-03	3.5E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	1.5E-08	1.4E-09	1.6E-08	NC	NC	NC
Others									
PCBs	NA	NA	NA	1.8E-03	3.8E-04	2.2E-03	NC	NC	NC
Sulphate	NA	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (total)	NA	NA	NA	2.5E-09	2.6E-10	3.0E-09	NC	NC	NC
Dust Pallatives									
Epichlorohydrin	NA	NA	NA	1.25E-06	4.48E-08	1.3E-06	NC	NC	NC

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-4A Baseline Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Adult)
(Based on Background Multi-Media Concentrations)

Scenario: Baseline

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04	3.2E-06	4.1E-06	2.02E-09	5.98E-05	8.20E-05	6.26E-06	1.49E-04	1.6E-04
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04	3.8E-06	4.8E-06	2.38E-09	4.30E-05	5.90E-05	3.13E-06	1.10E-04	1.1E-04
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04	5.7E-06	7.2E-06	3.57E-09	1.03E-04	1.41E-04	5.48E-06	2.56E-04	2.6E-04
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA	2.4E-06	3.0E-06	1.49E-09	1.38E-05	1.89E-05	**NA**	3.81E-05	3.8E-05
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04	2.3E-06	2.9E-06	1.43E-09	2.65E-05	3.63E-05	2.35E-06	6.79E-05	7.0E-05
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA	3.8E-06	4.8E-06	2.38E-09	6.58E-05	9.03E-05	**NA**	1.65E-04	1.6E-04
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA	5.7E-07	7.2E-07	3.57E-10	2.82E-06	3.88E-06	**NA**	7.98E-06	8.0E-06
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	6.1E-06	7.7E-06	3.84E-09	2.52E-04	3.46E-04	3.99E-05	6.12E-04	6.5E-04
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03	2.5E-06	3.2E-06	1.61E-09	1.34E-05	1.84E-05	1.49E-05	3.76E-05	5.2E-05
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02	4.9E-06	6.2E-06	3.09E-09	5.16E-04	7.08E-04	1.25E-04	1.23E-03	1.4E-03
Carcinogenic PAH Mixture											1.97E-04	2.68E-03	1.5E-03
Non-carcinogenic PAHs													
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03	9.4E-08	1.2E-07	5.95E-11	2.03E-05	2.79E-05	8.61E-06	4.85E-05	5.7E-05
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03	7.5E-08	9.6E-08	4.76E-11	8.04E-06	1.10E-05	1.25E-06	1.92E-05	2.0E-05
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03	2.8E-07	3.6E-07	1.78E-10	4.32E-05	5.93E-05	2.99E-05	1.03E-04	1.3E-04
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	6.1E-06	7.7E-06	3.84E-09	2.52E-04	3.46E-04	3.99E-05	6.12E-04	6.5E-04
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01	2.8E-07	3.6E-07	1.52E-09	1.39E-04	1.91E-04	1.50E-02	3.31E-04	1.5E-02
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA	1.4E-06	1.8E-06	8.92E-10	3.31E-04	4.54E-04	**NA**	7.87E-04	7.9E-04
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03	6.6E-06	8.4E-06	4.16E-09	3.97E-04	5.45E-04	3.83E-05	9.57E-04	9.9E-04
Non-carcinogenic PAH Mixture											1.51E-02	2.86E-03	1.8E-02
Metals and Metalloids													
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA	4.8E-05	4.1E-05	3.03E-08	9.88E-03	1.36E-02	**NA**	2.35E-02	2.4E-02
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04	5.7E-03	1.5E-03	4.61E-06	2.35E-01	3.23E-01	4.90E-04	**5.65E-01**	**5.7E-01**
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03	9.4E-05	8.1E-05	5.06E-05	1.45E-02	1.99E-02	3.60E-03	3.46E-02	3.8E-02
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07	3.4E-05	2.9E-05	9.12E-06	3.49E-04	4.79E-04	4.30E-05	9.00E-04	9.4E-04
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04	7.5E-05	6.4E-06	1.67E-05	3.85E-02	5.29E-02	1.70E-02	9.15E-02	1.1E-01
Chromium(III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	6.3E-04	4.9E-06	4.2E-06	3.92E-06	3.75E-05	5.15E-05	1.26E-04	1.02E-04	2.3E-04
Chromium(VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	6.3E-04	5.0E-05	4.3E-05	1.22E-06	3.88E-04	5.32E-04	6.30E-03	1.01E-03	7.3E-03
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05	2.1E-04	1.8E-04	5.63E-05	4.31E-03	5.91E-03	2.60E-04	1.07E-02	1.1E-02
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03	4.9E-05	2.5E-05	1.87E-05	2.03E-02	2.79E-02	3.40E-03	4.83E-02	5.2E-02
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03	3.9E-03	2.0E-04	1.11E-04	3.41E-02	4.68E-02	2.13E-02	8.50E-02	1.1E-01
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03	6.4E-04	5.5E-04	5.39E-03	1.65E-01	2.27E-01	5.40E-02	**3.99E-01**	**4.5E-01**
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04	1.1E-05	9.3E-07	6.84E-08	2.80E-03	3.85E-03	1.92E-05	6.66E-03	6.7E-03
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03	4.7E-05	4.0E-05	1.27E-07	4.36E-02	5.98E-02	1.23E-02	1.04E-01	1.2E-01
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03	5.4E-04	4.2E-04	4.54E-03	3.32E-02	4.55E-02	**4.00E-01**	8.42E-02	**4.8E-01**
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04	1.5E-05	1.3E-06	1.14E-08	3.82E-04	5.25E-04	9.00E-06	9.23E-04	9.3E-04
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04	3.2E-05	2.7E-05	2.02E-08	8.21E-02	1.13E-01	2.58E-07	1.95E-01	1.9E-01
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04	2.9E-07	2.5E-07	1.85E-10	9.03E-06	1.24E-05	4.70E-08	2.20E-05	2.2E-05
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA	3.5E-04	3.0E-04	1.43E-05	3.09E-03	4.24E-03	**NA**	8.00E-03	8.0E-03
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03	8.4E-04	7.2E-04	3.40E-04	4.76E-03	6.54E-03	2.60E-02	1.32E-02	3.9E-02
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02	3.9E-05	3.4E-05	2.48E-08	4.00E-02	5.49E-02	4.70E-06	9.49E-02	9.5E-02
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04	1.3E-02	1.1E-02	8.06E-06	5.25E-02	7.21E-02	1.17E-07	1.48E-01	1.5E-01
Boron	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	0.00E+00	**NA**
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02	6.6E-03	5.7E-03	4.17E-06	2.72E-02	3.73E-02	1.58E-05	7.68E-02	7.7E-02
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA	7.0E-05	6.0E-05	4.43E-08	3.97E-04	5.44E-04	**NA**	1.07E-03	1.1E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Lanthanum	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Others													
Hexachlorobenzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
PCBs	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Sulphate	NA	NA	NA	NA	9.0E-01	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Hexachlorobenzene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Naphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
2-Methylnaphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Dust Pallatives													
Epichlorohydrin	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

TABLE I-4B: Project Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Adult)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes	ILCR Soil Ingestion	ILCR Soil Dermal	ILCR Soil Dust Inhalation	ILCR Plant (Aboveground)	ILCR Plant (Belowground)	ILCR Air Inhalation	ILCR All Routes
Polycyclic Aromatic Hydrocarbons																				
Carcinogenic PAHs																				
Benzo[a]anthracene	3.7E-04	2.8E-10	1.3E-06	1.0E-06	3.8E-04	3.5E-09	4.4E-09	2.18E-12	8.1E-08	8.8E-08	2.99E-06	1.78E-07	3.2E-06	2.4E-11	3.0E-11	8.6E-16	5.6E-10	6.1E-10	1.5E-10	1.4E-09
Benzo[a]pyrene	6.7E-04	5.1E-10	1.2E-06	1.1E-06	1.2E-05	6.4E-09	8.1E-09	4.01E-12	7.9E-08	9.9E-08	9.58E-08	1.93E-07	2.9E-07	4.4E-10	5.6E-10	1.6E-14	5.4E-09	6.9E-09	4.7E-11	1.3E-08
Benzo[b]fluoranthene	1.4E-04	1.1E-10	7.1E-07	3.7E-07	2.6E-04	1.3E-09	1.7E-09	8.26E-13	4.6E-08	3.3E-08	2.07E-06	8.16E-08	2.1E-06	9.0E-12	1.1E-11	3.3E-16	3.2E-10	2.3E-10	1.0E-10	6.7E-10
Benzo[g,h,i]perylene	2.2E-04	1.6E-10	2.2E-07	1.9E-07	2.0E-05	2.0E-09	2.6E-09	1.29E-12	1.4E-08	1.6E-08	1.58E-07	3.51E-08	1.9E-07	1.4E-12	1.8E-12	5.1E-17	9.7E-12	1.1E-11	7.8E-13	2.5E-11
Benzo[k]fluoranthene	5.7E-04	4.3E-10	1.1E-06	9.7E-07	5.6E-05	5.3E-09	6.8E-09	3.37E-12	6.8E-08	8.6E-08	4.37E-07	1.66E-07	6.0E-07	3.7E-11	4.7E-11	1.3E-15	4.7E-10	5.9E-10	2.2E-11	1.2E-09
Chrysene	7.8E-04	5.9E-10	2.1E-06	2.0E-06	1.1E-04	7.3E-09	9.3E-09	4.63E-12	1.4E-07	1.8E-07	8.68E-07	3.31E-07	1.2E-06	5.1E-12	6.4E-12	1.8E-16	9.6E-11	1.2E-10	4.3E-12	2.3E-10
Dibenzo[a,h]anthracene	1.7E-04	1.3E-10	1.4E-07	1.2E-07	1.6E-05	1.6E-09	2.0E-09	1.01E-12	9.2E-09	1.1E-08	1.21E-07	2.38E-08	1.5E-07	1.1E-10	1.4E-10	4.0E-15	6.4E-10	7.5E-10	6.0E-11	1.7E-09
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04	1.7E-09	2.1E-09	1.05E-12	1.0E-07	9.5E-08	3.04E-06	2.03E-07	3.2E-06	1.5E-13	1.9E-13	5.5E-18	9.6E-12	8.7E-12	2.0E-12	2.1E-11
Indeno[1,2,3-cd]pyrene	5.6E-04	4.3E-10	5.1E-07	4.3E-07	5.4E-05	5.3E-09	6.7E-09	3.33E-12	3.3E-08	3.8E-08	4.24E-07	8.29E-08	5.1E-07	3.6E-11	4.6E-11	1.3E-15	2.3E-10	2.6E-10	2.1E-11	5.9E-10
Phenanthrene	4.2E-06	3.2E-12	3.3E-07	6.5E-08	5.2E-04	4.0E-11	5.1E-11	2.52E-14	2.1E-08	5.8E-09	4.07E-06	2.70E-08	4.1E-06	2.8E-15	3.5E-15	1.0E-19	1.5E-12	4.0E-13	2.0E-12	3.9E-12
Carcinogenic PAH Mixture											1.43E-05	1.32E-06	1.1E-05							1.9E-08
Non-carcinogenic PAHs																				
Acenaphthene	3.1E-07	2.4E-13	1.7E-07	9.9E-09	1.2E-04	1.5E-12	1.9E-12	9.30E-16	5.6E-09	4.4E-10	4.53E-07	6.07E-09	4.6E-07	NA	NA	NA	NA	NA	NA	NA
Acenaphthylene	1.1E-07	8.5E-14	4.6E-08	3.4E-09	9.8E-05	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.0E-06	1.5E-12	1.3E-07	3.2E-08	4.2E-04	1.9E-12	2.4E-12	1.21E-15	8.3E-10	2.8E-10	3.28E-07	1.12E-09	3.3E-07	NA	NA	NA	NA	NA	NA	NA
Fluorene	4.3E-07	3.2E-13	8.4E-08	9.5E-09	7.7E-05	3.0E-12	3.8E-12	1.90E-15	4.1E-09	6.3E-10	4.50E-07	4.71E-09	4.5E-07	NA	NA	NA	NA	NA	NA	NA
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04	1.7E-09	2.1E-09	1.05E-12	1.0E-07	9.5E-08	3.04E-06	2.03E-07	3.2E-06	1.5E-13	1.9E-13	5.5E-18	9.6E-12	8.7E-12	2.0E-12	2.1E-11
Naphthalene	4.0E-09	3.0E-15	2.7E-07	2.9E-10	1.8E-04	5.6E-14	7.1E-14	3.03E-16	2.6E-08	3.8E-11	1.83E-05	2.65E-08	1.8E-05	NA	NA	NA	NA	NA	NA	NA
2-Methylnaphthalene	7.1E-09	5.4E-15	2.6E-08	2.4E-10	2.1E-05	5.0E-13	6.3E-13	3.16E-16	1.3E-08	1.6E-10	1.22E-06	1.27E-08	1.2E-06	NA	NA	NA	NA	NA	NA	NA
Pyrene	3.1E-04	2.4E-10	3.7E-06	2.7E-06	6.6E-04	2.9E-09	3.7E-09	1.85E-12	2.4E-07	2.4E-07	5.16E-06	4.87E-07	5.6E-06	NA	NA	NA	NA	NA	NA	NA
Non-carcinogenic PAH Mixture											2.89E-05	7.41E-07	3.0E-05							
Metals and Metalloids																				
Antimony	1.6E-03	1.2E-09	4.7E-05	4.7E-05	4.1E-05	1.5E-07	1.3E-07	9.24E-11	3.0E-05	4.1E-05	3.17E-06	7.18E-05	7.5E-05	NA	NA	NA	NA	NA	NA	NA
Arsenic	2.0E-02	1.5E-08	1.2E-04	1.2E-04	4.1E-04	1.9E-05	4.9E-06	1.54E-08	7.9E-04	1.1E-03	4.11E-04	1.89E-03	2.3E-03	1.0E-08	2.6E-09	9.8E-11	4.3E-07	5.8E-07	3.3E-07	1.3E-06
Barium	5.4E+00	4.1E-06	1.2E-01	1.2E-01	1.1E-01	7.6E-06	6.5E-06	4.08E-06	1.2E-03	1.6E-03	1.09E-01	2.80E-03	1.1E-01	NA	NA	NA	NA	NA	NA	NA
Beryllium	3.3E-03	2.5E-09	5.1E-06	4.9E-06	6.9E-05	4.6E-07	4.0E-07	1.24E-07	4.9E-06	6.5E-06	3.47E-03	1.24E-05	3.5E-03	NA	NA	6.0E-12	NA	NA	2.1E-08	2.1E-08
Cadmium	1.7E-03	1.3E-09	1.3E-04	1.3E-04	1.2E-05	5.8E-07	5.0E-08	1.30E-07	3.0E-04	4.1E-04	1.18E-03	7.11E-04	1.9E-03	NA	NA	1.3E-11	NA	NA	1.4E-08	1.4E-08
Chromium(III)	1.5E-03	1.2E-09	1.8E-06	1.7E-06	1.5E-04	2.9E-10	2.5E-10	2.33E-10	2.3E-09	3.1E-09	3.03E-05	6.14E-09	3.0E-05	NA	NA	NA	NA	NA	NA	NA
Chromium(VI)	4.6E-02	3.5E-08	5.3E-05	5.2E-05	4.7E-07	1.4E-05	1.2E-05	3.50E-07	1.1E-04	1.5E-04	4.74E-06	2.94E-04	3.0E-04	5.5E-09	4.7E-09	2.7E-09	4.3E-08	5.8E-08	4.5E-09	1.2E-07
Cobalt	1.9E-02	1.5E-08	5.8E-05	5.8E-05	6.3E-05	5.5E-07	4.7E-07	1.47E-07	1.1E-05	1.5E-05	6.32E-04	2.79E-05	6.6E-04	NA	NA	NA	NA	NA	NA	NA
Copper	2.0E-01	1.5E-07	1.2E-02	1.2E-02	6.5E-04	4.0E-07	2.0E-07	1.50E-07	1.6E-04	2.2E-04	6.47E-04	3.87E-04	1.0E-03	NA	NA	NA	NA	NA	NA	NA
Lead	3.4E-02	2.6E-08	2.3E-04	2.3E-04	1.2E-04	6.0E-06	3.1E-07	1.72E-07	5.3E-05	7.2E-05	8.15E-04	1.32E-04	9.5E-04	NA	NA	NA	NA	NA	NA	NA
Manganese	3.0E-01	2.2E-07	1.1E-02	1.1E-02	9.6E-04	5.4E-07	4.6E-07	4.49E-06	1.4E-04	1.9E-04	1.93E-02	3.32E-04	2.0E-02	NA	NA	NA	NA	NA	NA	NA
Molybdenum	7.7E-03	5.9E-09	2.9E-04	2.9E-04	2.7E-05	7.8E-08	6.7E-09	4.89E-10	2.0E-05	2.8E-05	2.28E-06	4.76E-05	5.0E-05	NA	NA	NA	NA	NA	NA	NA
Mercury	1.2E-03	9.1E-10	1.6E-04	1.6E-04	5.8E-06	1.1E-06	9.7E-07	3.04E-09	1.0E-03	1.4E-03	1.93E-05	2.49E-03	2.5E-03	NA	NA	NA	NA	NA	NA	NA
Nickel	6.2E-02	4.7E-08	5.6E-04	5.5E-04	2.0E-04	1.6E-06	1.2E-06	1.34E-05	9.8E-05	1.3E-04	5.74E-02	2.48E-04	5.8E-02	NA	NA	6.1E-11	NA	NA	3.3E-08	3.3E-08
Selenium	9.9E-03	7.5E-09	3.7E-05	3.7E-05	3.2E-05	4.9E-07	4.2E-08	3.77E-10	1.3E-05	1.7E-05	1.62E-06	3.06E-05	3.2E-05	NA	NA	NA	NA	NA	NA	NA
Strontium	3.4E+00	2.6E-06	1.3E+00	1.3E+00	1.1E-02	1.6E-06	1.4E-06	1.01E-09	4.1E-03	5.6E-03	4.33E-06	9.74E-03	9.7E-03	NA	NA	NA	NA	NA	NA	NA
Tin	3.5E-03	2.6E-09	1.6E-05	1.6E-05	1.7E-05	4.9E-10	4.2E-10	3.11E-13	1.5E-08	2.1E-08	1.98E-09	3.71E-08	3.9E-08	NA	NA	NA	NA	NA	NA	NA
Uranium	5.4E-03	4.1E-09	7.0E-06	6.9E-06	1.8E-05	2.5E-06	2.2E-06	1.03E-07	2.3E-05	3.1E-05	4.41E-04	5.81E-05	5.0E-04	NA	NA	NA	NA	NA	NA	NA
Vanadium	1.6E-01	1.2E-07	1.4E-04	1.3E-04	5.4E-04	3.0E-06	2.6E-06	1.22E-06	1.8E-05	2.4E-05	5.39E-03	4.81E-05	5.4E-03	NA	NA	NA	NA	NA	NA	NA
Zinc	2.1E-01	1.6E-07	3.1E-02	3.1E-02	7.1E-04	9.8E-08	8.4E-08	6.18E-11	1.0E-04	1.4E-04	2.78E-07	2.37E-04	2.4E-04	NA	NA	NA	NA	NA	NA	NA
Aluminum	3.5E+01	2.6E-05	2.2E-02	2.1E-02	1.1E-01	3.3E-05	2.8E-05	2.06E-08	1.4E-04	1.8E-04	8.87E-05	3.85E-04	4.7E-04	NA	NA	NA	NA	NA	NA	NA
Boron	2.7E-01	2.0E-07	1.6E-01	1.6E-01	8.8E-04	4.3E-06	3.7E-07	6.79E-10	1.8E-02	2.4E-02	2.92E-06	4.22E-02	4.2E-02	NA	NA	NA	NA	NA	NA	NA
Iron	3.9E+01	2.9E-05	2.4E-02	2.3E-02	1.3E-01	1.4E-05	1.2E-05	8.63E-09	5.9E-05	7.7E-05	3.71E-05	1.61E-04	2.0E-04	NA	NA	NA	NA	NA	NA	NA
Titanium	2.4E+00	1.8E-06	2.0E-03	2.0E-03	7.9E-03	2.3E-07	1.9E-07	1.43E-10	1.3E-06	1.8E-06	6.16E-07	3.50E-06	4.1E-06	NA	NA	NA	NA	NA	NA	NA
Indium	1.4E-09	1.1E-15	6.8E-08	8.7E-11	5.9E-06	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	3.7E-09	2.8E-15	1.7E-07	2.2E-10	1.5E-05	2.1E-15	1.8E-15	1.31E-18	6.7E-10	1.2E-12	6.97E-09	6.66E-10	7.6E-09	NA	NA	NA	NA	NA	NA	NA
Others																				
Hexachlorobenzene	8.2E-14	6.2E-20	1.7E-11	0.0E+00	6.8E-10	8.5E-17	7.3E-17	5.39E-20	1.2E-10	0.0E+00	5.88E-10	1.23E-10	7.1E-10	3.7E-20	3.2E-20	2.9E-23	5.3E-14	0.0E+00	3.9E-14	9.2E-14
PCBs	3.0E-12	2.3E-18	2.2E-10	1.3E-15	8.4E-09	3.7E-10	3.2E-10	2.35E-13	1.8E-04	1.5E-09	8.59E-04	1.81E-04	1.0E-03	NA	NA	NA	NA	NA	NA	NA
Sulphate	4.6E-07	3.5E-13	2.2E-05	2.8E-08	1.9E-03	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (gas phase) (above ground plant only)	0.0E+00	0.0E+00	1.7E-13	0.0E+00	See above	0.0E+00	0.0E+00	0.00E+00	1.2E-12	0.0E+00	See above	1.21E-12	1.2E-12	0.0E+00	0.0E+00	0.0E+00	5.2E-16	0.0E+00	See above	5.2E-16
Hexachlorobenzene (total)	0.0E+00	0.0E+00	0.0E+00	0.0E+00	See above	8.5E-17	7.3E-17	5.39E-20	1.2E-10	0.0E+00	5.88E-10	1.24E-10	7.1E-10	3.7E-20	3.2E-20	2.9E-23	5.4E-14	0.0E+00	3.9E-14	9.3E-14
Dust Pallatives																				
Epichlorohydrin	3.8E-09	2.9E-15	4.7E-08	1.2E-08	1.3E-06	1.8E-13	1.5E-12	2.89E-15	1.5E-08	5.4E-09	1.25E-06	2.06E-08	1.3E-06	1.1E-17	9.1E-17	3.5E-21	9.0E-13	3.2E-13	1.9E-13	1.4E-12

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

[1] maximum outdoor air concentration assumed equal to the analytical detection limit

TABLE I-4C: Cumulative (Background+Project) Scenario Risk Estimates for a Maximum North Delta Residential Receptor, Adult
(Based on Maximum Multi-Media Concentrations)

Scenario: Cumulative

	BASELINE HQ Air Inhalation	BASELINE HQ Soil/Vegetation	BASELINE HQ All Routes	PROJECT HQ Air Inhalation	PROJECT HQ Soil/Vegetation	PROJECT HQ All Routes	CUMULATIVE HQ Air Inhalation	CUMULATIVE HQ Soil/Vegetation	CUMULATIVE HQ All Routes
Polycyclic Aromatic Hydrocarbons									
Carcinogenic PAHs									
Benzo[a]anthracene	6.3E-06	1.5E-04	1.6E-04	3.0E-06	1.8E-07	3.2E-06	9.3E-06	1.5E-04	1.6E-04
Benzo[a]pyrene	3.1E-06	1.1E-04	1.1E-04	9.6E-08	1.9E-07	2.9E-07	3.2E-06	1.1E-04	1.1E-04
Benzo[b]fluoranthene	5.5E-06	2.6E-04	2.6E-04	2.1E-06	8.2E-08	2.1E-06	7.5E-06	2.6E-04	2.6E-04
Benzo[g,h,i]perylene	NA	3.8E-05	3.8E-05	1.6E-07	3.5E-08	1.9E-07	NA	3.8E-05	3.8E-05
Benzo[k]fluoranthene	2.3E-06	6.8E-05	7.0E-05	4.4E-07	1.7E-07	6.0E-07	2.8E-06	6.8E-05	7.1E-05
Chrysene	NA	1.6E-04	1.6E-04	8.7E-07	3.3E-07	1.2E-06	NA	1.6E-04	1.6E-04
Dibenzo[a,h]anthracene	NA	8.0E-06	8.0E-06	1.2E-07	2.4E-08	1.5E-07	NA	8.0E-06	8.0E-06
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	3.0E-06	2.0E-07	3.2E-06	4.3E-05	6.1E-04	6.5E-04
Indeno[1,2,3-c,d]pyrene	1.5E-05	3.8E-05	5.2E-05	4.2E-07	8.3E-08	5.1E-07	1.5E-05	3.8E-05	5.3E-05
Phenanthrene	1.3E-04	1.2E-03	1.4E-03	4.1E-06	2.7E-08	4.1E-06	1.3E-04	1.2E-03	1.4E-03
Carcinogenic PAH Mixture	2.0E-04	2.7E-03	1.5E-03	1.4E-05	1.3E-06	1.1E-05	2.1E-04	2.7E-03	2.9E-03
Non-carcinogenic PAHs									
Acenaphthene	8.6E-06	4.8E-05	5.7E-05	4.5E-07	6.1E-09	4.6E-07	9.1E-06	4.8E-05	5.8E-05
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	1.3E-06	1.9E-05	2.0E-05	3.3E-07	1.1E-09	3.3E-07	1.6E-06	1.9E-05	2.1E-05
Fluorene	3.0E-05	1.0E-04	1.3E-04	4.5E-07	4.7E-09	4.5E-07	3.0E-05	1.0E-04	1.3E-04
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	3.0E-06	2.0E-07	3.2E-06	4.3E-05	6.1E-04	6.5E-04
Naphthalene	1.5E-02	3.3E-04	1.5E-02	1.8E-05	2.6E-08	1.8E-05	1.5E-02	3.3E-04	1.5E-02
2-Methylnaphthalene	NA	7.9E-04	7.9E-04	1.2E-06	1.3E-08	1.2E-06	NC	7.9E-04	7.9E-04
Pyrene	3.8E-05	9.6E-04	9.9E-04	5.2E-06	4.9E-07	5.6E-06	4.4E-05	9.6E-04	1.0E-03
Non-carcinogenic PAH Mixture	1.5E-02	2.9E-03	1.8E-02	2.9E-05	7.4E-07	3.0E-05	1.5E-02	2.9E-03	1.8E-02
Metals and Metalloids									
Antimony	NA	2.4E-02	2.4E-02	3.2E-06	7.2E-05	7.5E-05	NC	2.4E-02	2.4E-02
Arsenic	4.9E-04	**5.7E-01**	**5.7E-01**	4.1E-04	1.9E-03	2.3E-03	9.0E-04	**5.7E-01**	**5.7E-01**
Barium	3.6E-03	3.5E-02	3.8E-02	1.1E-01	2.8E-03	1.1E-01	1.1E-01	3.7E-02	1.5E-01
Beryllium	4.3E-05	9.0E-04	9.4E-04	3.5E-03	1.2E-05	3.5E-03	3.5E-03	9.1E-04	4.4E-03
Cadmium	1.7E-02	9.1E-02	1.1E-01	1.2E-03	7.1E-04	1.9E-03	1.8E-02	9.2E-02	1.1E-01
Chromium(III)	1.3E-04	1.0E-04	2.3E-04	3.0E-05	6.1E-09	3.0E-05	1.6E-04	1.0E-04	2.6E-04
Chromium(VI)	6.3E-03	1.0E-03	7.3E-03	4.7E-06	2.9E-04	3.0E-04	6.3E-03	1.3E-03	7.6E-03
Cobalt	2.6E-04	1.1E-02	1.1E-02	6.3E-04	2.8E-05	6.6E-04	8.9E-04	1.1E-02	1.2E-02
Copper	3.4E-03	4.8E-02	5.2E-02	6.5E-04	3.9E-04	1.0E-03	4.0E-03	4.9E-02	5.3E-02
Lead	2.1E-02	8.5E-02	1.1E-01	8.1E-04	1.3E-04	9.5E-04	2.2E-02	8.5E-02	1.1E-01
Manganese	5.4E-02	**4.0E-01**	**4.5E-01**	1.9E-02	3.3E-04	2.0E-02	7.3E-02	**4.0E-01**	**4.7E-01**
Molybdenum	1.9E-05	6.7E-03	6.7E-03	2.3E-06	4.8E-05	5.0E-05	2.1E-05	6.7E-03	6.7E-03
Mercury	1.2E-02	1.0E-01	1.2E-01	1.9E-05	2.5E-03	2.5E-03	1.2E-02	1.1E-01	1.2E-01
Nickel	**4.0E-01**	8.4E-02	**4.8E-01**	5.7E-02	2.5E-04	5.8E-02	**4.6E-01**	8.4E-02	**5.4E-01**
Selenium	9.0E-06	9.2E-04	9.3E-04	1.6E-06	3.1E-05	3.2E-05	1.1E-05	9.5E-04	9.6E-04
Strontium	2.6E-07	1.9E-01	1.9E-01	4.3E-06	9.7E-03	9.7E-03	4.6E-06	2.0E-01	2.0E-01
Tin	4.7E-08	2.2E-05	2.2E-05	2.0E-09	3.7E-08	3.9E-08	4.9E-08	2.2E-05	2.2E-05
Uranium	NA	8.0E-03	8.0E-03	4.4E-04	5.8E-05	5.0E-04	NC	8.1E-03	8.5E-03
Vanadium	2.6E-02	1.3E-02	3.9E-02	5.4E-03	4.8E-05	5.4E-03	3.1E-02	1.3E-02	4.5E-02
Zinc	4.7E-06	9.5E-02	9.5E-02	2.8E-07	2.4E-04	2.4E-04	5.0E-06	9.5E-02	9.5E-02
Aluminum	1.2E-07	**1.5E-01**	**1.5E-01**	8.9E-05	3.9E-04	4.7E-04	8.9E-05	**1.5E-01**	**1.5E-01**
Boron	NA	NA	NA	2.9E-06	4.2E-02	4.2E-02	NC	NC	NC
Iron	1.6E-05	7.7E-02	7.7E-02	3.7E-05	1.6E-04	2.0E-04	5.3E-05	7.7E-02	7.7E-02
Titanium	NA	1.1E-03	1.1E-03	6.2E-07	3.5E-06	4.1E-06	NC	1.1E-03	1.1E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	7.0E-09	6.7E-10	7.6E-09	NC	NC	NC
Others									
PCBs	NA	NA	NA	8.6E-04	1.8E-04	1.0E-03	NC	NC	NC
Sulphate	NA	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (total)	NA	NA	NA	5.9E-10	1.2E-10	7.1E-10	NC	NC	NC
Dust Pallatives									
Epichlorohydrin	NA	NA	NA	1.25E-06	2.06E-08	1.3E-06	NC	NC	NC

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-5A Baseline Scenario Risk Estimates for Maximum North Delta Residential Receptor (Toddler)
(Based on Background Multi-Media Concentrations)

Scenario: Baseline

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04	5.5E-05	7.0E-06	4.33E-09	1.25E-04	1.96E-04	1.34E-05	3.83E-04	4.0E-04
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04	6.5E-05	8.2E-06	5.10E-09	9.00E-05	1.41E-04	6.71E-06	3.04E-04	3.1E-04
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04	9.7E-05	1.2E-05	7.65E-09	2.15E-04	3.37E-04	1.17E-05	6.62E-04	6.7E-04
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA	4.0E-05	5.1E-06	3.19E-09	2.89E-05	4.53E-05	NA	1.20E-04	1.2E-04
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04	3.9E-05	4.9E-06	3.06E-09	5.55E-05	8.70E-05	5.03E-06	1.86E-04	1.9E-04
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA	6.5E-05	8.2E-06	5.10E-09	1.38E-04	2.16E-04	NA	4.27E-04	4.3E-04
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA	9.7E-06	1.2E-06	7.65E-10	5.92E-06	9.28E-06	NA	2.61E-05	2.6E-05
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	1.0E-04	1.3E-05	8.22E-09	5.28E-04	8.27E-04	8.55E-05	1.47E-03	1.6E-03
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03	4.4E-05	5.6E-06	3.44E-09	2.81E-05	4.40E-05	3.19E-05	1.21E-04	1.5E-04
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02	8.4E-05	1.1E-05	6.63E-09	1.08E-03	1.69E-03	2.68E-04	2.87E-03	3.1E-03
Carcinogenic PAH Mixture											4.23E-04	6.57E-03	3.9E-03
Non-carcinogenic PAHs													
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03	1.6E-06	2.1E-07	1.27E-10	4.26E-05	6.68E-05	1.84E-05	1.11E-04	1.3E-04
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03	1.3E-06	1.6E-07	1.02E-10	1.68E-05	2.64E-05	2.68E-06	4.47E-05	4.7E-05
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03	4.8E-06	6.2E-07	3.82E-10	9.05E-05	1.42E-04	6.41E-05	2.38E-04	3.0E-04
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	1.0E-04	1.3E-05	8.22E-09	5.28E-04	8.27E-04	8.55E-05	1.47E-03	1.6E-03
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01	4.8E-06	6.2E-07	1.52E-09	2.92E-04	4.58E-04	1.50E-02	7.55E-04	1.6E-02
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA	2.4E-05	3.1E-06	1.91E-09	6.93E-04	1.09E-03	NA	1.81E-03	1.8E-03
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03	1.1E-04	1.4E-05	8.92E-09	8.32E-04	1.30E-03	8.22E-05	2.26E-03	2.3E-03
Non-carcinogenic PAH Mixture											1.53E-02	6.69E-03	2.2E-02
Metals and Metalloids													
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA	8.2E-04	7.1E-05	6.50E-08	2.07E-02	3.25E-02	NA	5.41E-02	5.4E-02
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04	9.8E-02	2.5E-03	4.61E-06	**4.93E-01**	**7.73E-01**	4.90E-04	**1.37E+00**	**1.4E+00**
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03	1.6E-03	1.4E-04	5.06E-05	3.04E-02	4.76E-02	3.60E-03	7.98E-02	8.3E-02
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07	5.8E-04	5.0E-05	9.12E-06	7.31E-04	1.15E-03	4.30E-05	2.52E-03	2.6E-03
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04	1.3E-03	1.1E-05	1.67E-05	8.07E-02	1.27E-01	1.70E-02	**2.09E-01**	**2.3E-01**
Chromium(III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	6.3E-04	8.3E-05	7.2E-06	3.92E-06	7.86E-05	1.23E-04	1.26E-04	2.96E-04	4.2E-04
Chromium(VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	6.3E-04	8.6E-04	7.4E-05	1.22E-06	8.12E-04	1.27E-03	6.30E-03	3.02E-03	9.3E-03
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05	3.6E-03	3.1E-04	5.63E-05	9.03E-03	1.41E-02	2.60E-04	2.71E-02	2.7E-02
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03	1.3E-03	6.8E-05	1.87E-05	6.59E-02	1.03E-01	3.40E-03	1.71E-01	1.7E-01
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03	1.8E-01	9.1E-04	1.11E-04	1.90E-01	**2.98E-01**	2.13E-02	**6.67E-01**	**6.9E-01**
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03	1.3E-02	1.1E-03	5.39E-03	**3.97E-01**	**6.22E-01**	5.40E-02	**1.04E+00**	**1.1E+00**
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04	2.3E-04	2.0E-06	6.84E-08	7.15E-03	1.12E-02	1.92E-05	1.86E-02	1.9E-02
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03	8.1E-04	6.9E-05	1.27E-07	9.14E-02	1.43E-01	1.23E-02	**2.35E-01**	**2.5E-01**
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03	9.2E-03	7.2E-04	4.54E-03	6.95E-02	1.09E-01	**4.00E-01**	1.93E-01	**5.9E-01**
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04	2.3E-04	2.0E-06	1.14E-08	7.37E-04	1.15E-03	9.00E-06	2.13E-03	2.1E-03
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04	5.5E-04	4.7E-05	4.32E-08	1.72E-01	**2.70E-01**	5.53E-07	**4.42E-01**	**4.4E-01**
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04	5.0E-06	4.3E-07	3.96E-10	1.89E-05	2.96E-05	1.01E-07	5.40E-05	5.4E-05
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA	6.1E-03	5.2E-04	1.43E-05	6.47E-03	1.01E-02	NA	2.32E-02	2.3E-02
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03	1.4E-02	1.2E-03	3.40E-04	9.98E-03	1.56E-02	2.60E-02	4.17E-02	6.8E-02
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02	8.1E-04	7.0E-05	6.37E-08	1.01E-01	1.58E-01	1.21E-05	**2.59E-01**	**2.6E-01**
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04	**2.2E-01**	1.9E-02	1.73E-05	1.10E-01	1.72E-01	2.52E-07	**5.20E-01**	**5.2E-01**
Boron	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02	1.1E-01	9.8E-03	8.94E-06	5.70E-02	8.93E-02	3.40E-05	**2.70E-01**	**2.7E-01**
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA	1.2E-03	1.0E-04	9.49E-08	8.31E-04	1.30E-03	NA	3.44E-03	3.4E-03
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Others													
Hexachlorobenzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PCBs	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sulphate	NA	NA	NA	NA	9.0E-01	NA	NA	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
Naphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
2-Methylnaphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives													
Epichlorohydrin	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

[1] maximum outdoor air concentration assumed equal to the analytical detection limit

TABLE I-5B: Project Scenario Risk Estimates for the Maximum Rail Corridor Residential Scenario (Toddler)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	5.5E-04	4.2E-10	6.4E-04	1.5E-06	7.4E-05	8.8E-08	1.1E-08	6.98E-12	8.7E-05	3.2E-07	1.2E-06	8.8E-05	8.9E-05
Benzo[a]pyrene	1.0E-03	7.7E-10	2.6E-04	1.7E-06	3.0E-05	1.6E-07	2.1E-08	1.29E-11	3.6E-05	3.6E-07	5.0E-07	3.6E-05	3.7E-05
Benzo[b]fluoranthene	2.1E-04	1.6E-10	9.4E-04	5.5E-07	1.1E-04	3.4E-08	4.3E-09	2.65E-12	1.3E-04	1.2E-07	1.8E-06	1.3E-04	1.3E-04
Benzo[g,h,i]perylene	3.2E-04	2.5E-10	7.1E-05	2.8E-07	8.3E-06	5.2E-08	6.7E-09	4.13E-12	9.6E-06	5.9E-08	1.4E-07	9.7E-06	9.8E-06
Benzo[k]fluoranthene	8.5E-04	6.4E-10	2.0E-04	1.4E-06	2.3E-05	1.4E-07	1.7E-08	1.08E-11	2.7E-05	3.1E-07	3.8E-07	2.7E-05	2.8E-05
Chrysene	1.2E-03	8.8E-10	3.9E-04	3.0E-06	4.5E-05	1.9E-07	2.4E-08	1.48E-11	5.3E-05	6.3E-07	7.6E-07	5.4E-05	5.5E-05
Dibenzo[a,h]anthracene	2.5E-04	1.9E-10	5.5E-05	1.8E-07	6.2E-06	4.1E-08	5.2E-09	3.23E-12	7.5E-06	3.9E-08	1.0E-07	7.6E-06	7.7E-06
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04	4.3E-08	5.4E-09	3.36E-12	1.8E-04	3.4E-07	2.7E-06	1.8E-04	1.9E-04
Indeno[1,2,3-c,d]pyrene	8.4E-04	6.4E-10	1.9E-04	6.4E-07	2.2E-05	1.4E-07	1.7E-08	1.07E-11	2.6E-05	1.4E-07	3.6E-07	2.6E-05	2.7E-05
Phenanthrene	6.3E-06	4.8E-12	4.4E-04	9.7E-08	5.2E-04	1.0E-09	1.3E-10	8.07E-14	6.0E-05	2.1E-08	8.7E-06	6.0E-05	6.8E-05
Carcinogenic PAH Mixture											1.7E-05	6.2E-04	5.7E-04
Non-carcinogenic PAHs													
Acenaphthene	4.7E-07	3.6E-13	4.0E-04	1.5E-08	4.9E-05	3.8E-11	4.8E-12	2.98E-15	2.7E-05	1.6E-09	4.1E-07	2.7E-05	2.7E-05
Acenaphthylene	1.7E-07	1.3E-13	1.1E-04	5.1E-09	9.0E-05	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	3.0E-06	2.3E-12	2.2E-04	4.7E-08	4.2E-05	4.9E-11	6.2E-12	3.87E-15	3.0E-06	1.0E-09	7.1E-08	3.0E-06	3.0E-06
Fluorene	6.4E-07	4.8E-13	1.9E-04	1.4E-08	4.7E-05	7.7E-11	9.8E-12	6.10E-15	1.9E-05	2.3E-09	5.9E-07	1.9E-05	2.0E-05
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04	4.3E-08	5.4E-09	3.36E-12	1.8E-04	3.4E-07	2.7E-06	1.8E-04	1.9E-04
Naphthalene	5.9E-09	4.5E-15	7.9E-05	4.2E-10	1.7E-05	1.4E-12	1.8E-13	4.48E-16	1.6E-05	1.3E-10	1.7E-06	1.6E-05	1.8E-05
2-Methylnaphthalene	1.1E-08	8.1E-15	7.4E-05	3.6E-10	8.2E-06	1.3E-11	1.6E-12	1.01E-15	7.5E-05	5.8E-10	1.0E-06	7.5E-05	7.6E-05
Pyrene	4.6E-04	3.5E-10	2.3E-03	4.1E-06	2.7E-04	7.5E-08	9.6E-09	5.92E-12	3.1E-04	8.7E-07	4.6E-06	3.1E-04	3.2E-04
Non-carcinogenic PAH Mixture											1.1E-05	6.4E-04	6.5E-04
Metals and Metalloids													
Antimony	2.3E-03	1.7E-09	1.8E-04	6.9E-05	1.2E-05	3.7E-06	3.2E-07	2.92E-10	2.4E-04	1.5E-04	2.1E-06	3.9E-04	3.9E-04
Arsenic	3.0E-02	2.3E-08	1.6E-03	1.8E-04	1.6E-04	4.9E-04	1.3E-05	2.30E-08	2.2E-02	3.9E-03	1.6E-04	2.7E-02	2.7E-02
Barium	8.0E+00	6.1E-06	5.7E-01	1.8E-01	4.3E-02	1.9E-04	1.7E-05	6.11E-06	1.2E-02	5.8E-03	4.3E-02	1.8E-02	6.1E-02
Beryllium	4.9E-03	3.7E-09	2.4E-04	7.3E-06	3.0E-05	1.2E-05	1.0E-06	1.86E-07	4.9E-04	2.3E-05	1.5E-03	5.3E-04	2.0E-03
Cadmium	2.6E-03	1.9E-09	3.1E-04	1.9E-04	1.8E-05	1.5E-05	1.3E-07	1.94E-07	1.5E-03	1.5E-03	1.8E-03	3.0E-03	4.8E-03
Chromium(III)	2.3E-03	1.7E-09	1.1E-04	2.6E-06	3.7E-04	7.4E-09	6.4E-10	3.49E-10	3.1E-07	1.1E-08	7.4E-05	3.3E-07	7.4E-05
Chromium(VI)	6.9E-02	5.2E-08	3.4E-03	7.8E-05	3.0E-07	3.7E-04	3.2E-05	5.24E-07	1.5E-02	5.5E-04	3.0E-06	1.6E-02	1.6E-02
Cobalt	2.9E-02	2.2E-08	1.5E-03	8.7E-05	1.6E-04	1.4E-05	1.2E-06	2.20E-07	6.0E-04	5.5E-05	1.6E-03	6.7E-04	2.2E-03
Copper	3.0E-01	2.2E-07	3.2E-02	1.8E-02	1.6E-03	1.6E-05	8.1E-07	2.24E-07	1.4E-03	1.2E-03	1.6E-03	2.7E-03	4.3E-03
Lead	5.1E-02	3.9E-08	2.8E-03	3.4E-04	2.9E-04	4.1E-04	2.1E-06	2.57E-07	3.6E-03	6.9E-04	1.9E-03	4.7E-03	6.6E-03
Manganese	4.4E-01	3.4E-07	3.8E-02	1.7E-02	2.4E-03	1.6E-05	1.4E-06	6.71E-06	1.1E-03	7.7E-04	4.7E-02	1.9E-03	4.9E-02
Molybdenum	1.2E-02	8.8E-09	9.9E-04	4.3E-04	6.2E-05	2.4E-06	2.1E-08	7.31E-10	1.7E-04	1.2E-04	5.2E-06	3.0E-04	3.0E-04
Mercury	1.2E-02	1.4E-09	3.3E-04	2.4E-04	1.0E-05	1.9E-04	1.6E-05	4.54E-09	4.4E-03	5.1E-03	3.4E-05	9.8E-03	9.8E-03
Nickel	9.2E-02	7.0E-08	5.3E-03	8.3E-04	4.9E-04	4.1E-05	3.2E-06	2.00E-05	1.9E-03	4.8E-04	1.4E-01	2.5E-03	1.4E-01
Selenium	1.5E-02	1.1E-08	7.7E-04	5.6E-05	8.0E-05	1.2E-05	1.0E-07	5.63E-10	5.0E-04	5.7E-05	4.0E-06	5.7E-04	5.8E-04
Strontium	5.1E+00	3.9E-06	2.1E+00	1.9E+00	2.7E-02	4.1E-05	3.5E-06	3.23E-09	1.5E-02	2.0E-02	2.3E-05	3.5E-02	3.5E-02
Tin	5.2E-03	3.9E-09	2.7E-04	2.3E-05	2.8E-05	1.3E-08	1.1E-09	9.92E-13	5.6E-07	7.4E-08	7.0E-09	6.4E-07	6.5E-07
Uranium	8.1E-03	6.1E-09	4.0E-04	1.0E-05	4.3E-05	6.5E-05	5.6E-06	1.54E-07	2.7E-03	1.1E-04	1.1E-03	2.9E-03	4.0E-03
Vanadium	2.4E-01	1.8E-07	1.2E-02	2.0E-04	1.3E-03	7.8E-05	6.7E-06	1.83E-06	**3.2E-03**	8.4E-05	1.3E-02	3.4E-03	1.6E-02
Zinc	3.1E-01	2.4E-07	6.1E-02	4.6E-02	1.7E-03	3.0E-06	2.6E-07	2.37E-10	5.0E-04	5.9E-04	1.7E-06	1.1E-03	1.1E-03
Aluminum	5.2E+01	3.9E-05	2.5E+00	3.1E-02	2.8E-01	8.4E-04	7.2E-05	6.62E-08	3.4E-02	6.6E-04	4.7E-04	3.6E-02	3.6E-02
Boron	4.0E-01	3.0E-07	2.6E-01	2.4E-01	2.2E-03	1.1E-04	9.5E-07	1.01E-09	6.0E-02	8.7E-02	7.2E-06	1.5E-01	1.5E-01
Iron	5.8E+01	4.4E-05	2.8E+00	3.5E-02	3.1E-01	3.5E-04	3.0E-05	2.77E-08	1.4E-02	2.8E-04	2.0E-04	1.5E-02	1.5E-02
Titanium	3.6E+00	2.7E-06	1.8E-01	3.0E-03	1.9E-02	5.8E-06	5.0E-07	4.59E-10	2.4E-04	6.3E-06	3.2E-06	2.5E-04	2.5E-04
Dust Pallatives													
Epichlorohydrin	9.1E-14	6.9E-20	8.1E-05	2.9E-13	7.4E-02	7.4E-17	6.3E-17	6.93E-20	5.5E-05	3.1E-13	7.4E-02	5.5E-05	7.4E-02

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-5C: Cumulative (Background+Project) Scenario Risk Estimates for a Maximum Rail Corridor Residential Receptor, Toddler
(Based on Maximum Multi-Media Concentrations)

Scenario: Cumulative

	BASELINE HQ Air Inhalation	BASELINE HQ Soil/Vegetation	BASELINE HQ All Routes	PROJECT HQ Air Inhalation	PROJECT HQ Soil/Vegetation	PROJECT HQ All Routes	CUMULATIVE HQ Air Inhalation	CUMULATIVE HQ Soil/Vegetation	CUMULATIVE HQ All Routes
Polycyclic Aromatic Hydrocarbons									
Carcinogenic PAHs									
Benzo[a]anthracene	1.3E-05	3.8E-04	4.0E-04	1.2E-06	8.8E-05	8.9E-05	1.5E-05	4.7E-04	4.9E-04
Benzo[a]pyrene	6.7E-06	3.0E-04	3.1E-04	5.0E-07	3.6E-05	3.7E-05	7.2E-06	3.4E-04	3.5E-04
Benzo[b]fluoranthene	1.2E-05	6.6E-04	6.7E-04	1.8E-06	1.3E-04	1.3E-04	1.4E-05	7.9E-04	8.0E-04
Benzo[g,h,i]perylene	NA	1.2E-04	1.2E-04	1.4E-07	9.7E-06	9.8E-06	NC	1.3E-04	1.3E-04
Benzo[k]fluoranthene	5.0E-06	1.9E-04	1.9E-04	3.8E-07	2.7E-05	2.8E-05	5.4E-06	2.1E-04	2.2E-04
Chrysene	NA	4.3E-04	4.3E-04	7.6E-07	5.4E-05	5.5E-05	NC	4.8E-04	4.8E-04
Dibenzo[a,h]anthracene	NA	2.6E-05	2.6E-05	1.0E-07	7.6E-06	7.7E-06	NC	3.4E-05	3.4E-05
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	2.7E-06	1.8E-04	1.9E-04	8.8E-05	1.7E-03	1.7E-03
Indeno[1,2,3-c,d]pyrene	3.2E-05	1.2E-04	1.5E-04	3.6E-07	2.6E-05	2.7E-05	3.2E-05	1.5E-04	1.8E-04
Phenanthrene	2.7E-04	2.9E-03	3.1E-03	8.7E-06	6.0E-05	6.8E-05	2.8E-04	2.9E-03	3.2E-03
Carcinogenic PAH Mixture	4.2E-04	6.6E-03	3.9E-03	1.7E-05	6.2E-04	5.7E-04	4.4E-04	7.2E-03	7.6E-03
Non-carcinogenic PAHs									
Acenaphthene	1.8E-05	1.1E-04	1.3E-04	4.1E-07	2.7E-05	2.7E-05	1.9E-05	1.4E-04	1.6E-04
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	2.7E-06	4.5E-05	4.7E-05	7.1E-08	3.0E-06	3.0E-06	2.8E-06	4.8E-05	5.0E-05
Fluorene	6.4E-05	2.4E-04	3.0E-04	5.9E-07	1.9E-05	2.0E-05	6.5E-05	2.6E-04	3.2E-04
Fluoranthene	8.6E-05	1.5E-03	1.6E-03	2.7E-06	1.8E-04	1.9E-04	8.8E-05	1.7E-03	1.7E-03
Naphthalene	1.5E-02	7.5E-04	1.6E-02	1.7E-06	1.6E-05	1.8E-05	1.5E-02	7.7E-04	1.6E-02
2-Methylnaphthalene	NA	1.8E-03	1.8E-03	1.0E-06	7.5E-05	7.6E-05	NC	1.9E-03	1.9E-03
Pyrene	8.2E-05	2.3E-03	2.3E-03	4.6E-06	3.1E-04	3.2E-04	8.7E-05	2.6E-03	2.7E-03
Non-carcinogenic PAH Mixture	1.5E-02	6.7E-03	2.2E-02	1.1E-05	6.4E-04	6.5E-04	1.5E-02	7.3E-03	2.3E-02
Metals and Metalloids									
Antimony	NA	5.4E-02	5.4E-02	2.1E-06	3.9E-04	3.9E-04	NC	5.4E-02	5.4E-02
Arsenic	4.9E-04	**1.4E+00**	**1.4E+00**	1.6E-04	2.7E-02	2.7E-02	6.5E-04	**1.4E+00**	**1.4E+00**
Barium	3.6E-03	8.0E-02	8.3E-02	4.3E-02	1.8E-02	6.1E-02	4.7E-02	9.7E-02	1.4E-01
Beryllium	4.3E-05	2.5E-03	2.6E-03	1.5E-03	5.3E-04	2.0E-03	1.6E-03	3.0E-03	4.6E-03
Cadmium	1.7E-02	**2.1E-01**	**2.3E-01**	1.8E-03	3.0E-03	4.8E-03	1.9E-02	**2.1E-01**	**2.3E-01**
Chromium(III)	1.3E-04	3.0E-04	4.2E-04	7.4E-05	3.3E-07	7.4E-05	2.0E-04	3.0E-04	5.0E-04
Chromium(VI)	6.3E-03	3.0E-03	9.3E-03	3.0E-06	1.6E-02	1.6E-02	6.3E-03	1.9E-02	2.6E-02
Cobalt	2.6E-04	2.7E-02	2.7E-02	1.6E-03	6.7E-04	2.2E-03	1.8E-03	2.8E-02	3.0E-02
Copper	3.4E-03	1.7E-01	1.7E-01	1.6E-03	2.7E-03	4.3E-03	5.0E-03	1.7E-01	1.8E-01
Lead	2.1E-02	**6.7E-01**	**6.9E-01**	1.9E-03	4.7E-03	6.6E-03	2.3E-02	**6.7E-01**	**7.0E-01**
Manganese	5.4E-02	**1.0E+00**	**1.1E+00**	4.7E-02	1.9E-03	4.9E-02	1.0E-01	**1.0E+00**	**1.1E+00**
Molybdenum	1.9E-05	1.9E-02	1.9E-02	5.2E-06	3.0E-04	3.0E-04	2.4E-05	1.9E-02	1.9E-02
Mercury	1.2E-02	**2.4E-01**	**2.5E-01**	3.4E-05	9.8E-03	9.8E-03	1.2E-02	**2.5E-01**	**2.6E-01**
Nickel	**4.0E-01**	1.9E-01	**5.9E-01**	1.4E-01	2.5E-03	1.4E-01	**5.4E-01**	2.0E-01	**7.4E-01**
Selenium	9.0E-06	2.1E-03	2.1E-03	4.0E-06	5.7E-04	5.8E-04	1.3E-05	2.7E-03	2.7E-03
Strontium	5.5E-07	**4.4E-01**	**4.4E-01**	2.3E-05	3.5E-02	3.5E-02	2.3E-05	**4.8E-01**	**4.8E-01**
Tin	1.0E-07	5.4E-05	5.4E-05	7.0E-09	6.4E-07	6.5E-07	1.1E-07	5.5E-05	5.5E-05
Uranium	NA	2.3E-02	2.3E-02	1.1E-03	2.9E-03	4.0E-03	NC	2.6E-02	2.7E-02
Vanadium	2.6E-02	4.2E-02	6.8E-02	1.3E-02	3.4E-03	1.6E-02	3.9E-02	4.5E-02	8.4E-02
Zinc	1.2E-05	**2.6E-01**	**2.6E-01**	1.7E-06	1.1E-03	1.1E-03	1.4E-05	**2.6E-01**	**2.6E-01**
Aluminum	2.5E-07	**5.2E-01**	**5.2E-01**	4.7E-04	3.6E-02	3.6E-02	4.7E-04	**5.6E-01**	**5.6E-01**
Boron	NA	NA	NA	7.2E-06	1.5E-01	1.5E-01	NC	NC	NC
Iron	3.4E-05	**2.7E-01**	**2.7E-01**	2.0E-04	1.5E-02	1.5E-02	2.3E-04	**2.8E-01**	**2.8E-01**
Titanium	NA	3.4E-03	3.4E-03	3.2E-06	2.5E-04	2.5E-04	NC	3.7E-03	3.7E-03
Dust Pallatives									
Epichlorohydrin	ND	ND	ND	7.4E-02	5.5E-05	7.4E-02	NC	NC	NC

HQ = Hazard Quotient

NA = no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-6A: Baseline Scenario Risk Estimates for the Maximum Rail Corridor Residential Receptor (Adult)
(Based on Background Multi-Media Concentrations)

Scenario: Baseline

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04	3.2E-06	4.1E-06	2.02E-09	5.98E-05	8.20E-05	6.26E-06	1.49E-04	1.6E-04
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04	3.8E-06	4.8E-06	2.38E-09	4.30E-05	5.90E-05	3.13E-06	1.10E-04	1.1E-04
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04	5.7E-06	7.2E-06	3.57E-09	1.03E-04	1.41E-04	5.48E-06	2.56E-04	2.6E-04
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA	2.4E-06	3.0E-06	1.49E-09	1.38E-05	1.89E-05	**NA**	3.81E-05	3.8E-05
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04	2.3E-06	2.9E-06	1.43E-09	2.65E-05	3.63E-05	2.35E-06	6.79E-05	7.0E-05
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA	3.8E-06	4.8E-06	2.38E-09	6.58E-05	9.03E-05	**NA**	1.65E-04	1.6E-04
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA	5.7E-07	7.2E-07	3.57E-10	2.82E-06	3.88E-06	**NA**	7.98E-06	8.0E-06
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	6.1E-06	7.7E-06	3.84E-09	2.52E-04	3.46E-04	3.99E-05	6.12E-04	6.5E-04
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03	2.5E-06	3.2E-06	1.61E-09	1.34E-05	1.84E-05	1.49E-05	3.76E-05	5.2E-05
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02	4.9E-06	6.2E-06	3.09E-09	5.16E-04	7.08E-04	1.25E-04	1.23E-03	1.4E-03
Carcinogenic PAH Mixture											1.97E-04	2.68E-03	1.5E-03
Non-carcinogenic PAHs													
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03	9.4E-08	1.2E-07	5.95E-11	2.03E-05	2.79E-05	8.61E-06	4.85E-05	5.7E-05
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03	7.5E-08	9.6E-08	4.76E-11	8.04E-06	1.10E-05	1.25E-06	1.92E-05	2.0E-05
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03	2.8E-07	3.6E-07	1.78E-10	4.32E-05	5.93E-05	2.99E-05	1.03E-04	1.3E-04
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	6.1E-06	7.7E-06	3.84E-09	2.52E-04	3.46E-04	3.99E-05	6.12E-04	6.5E-04
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01	2.8E-07	3.6E-07	1.52E-09	1.39E-04	1.91E-04	1.50E-02	3.31E-04	1.5E-02
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA	1.4E-06	1.8E-06	8.92E-10	3.31E-04	4.54E-04	**NA**	7.87E-04	7.9E-04
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03	6.6E-06	8.4E-06	4.16E-09	3.97E-04	5.45E-04	3.83E-05	9.57E-04	9.9E-04
Non-carcinogenic PAH Mixture											1.51E-02	2.86E-03	1.8E-02
Metals and Metalloids													
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA	4.8E-05	4.1E-05	3.03E-08	9.88E-03	1.36E-02	**NA**	2.35E-02	2.4E-02
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04	5.7E-03	1.5E-03	4.61E-06	2.35E-01	3.23E-01	4.90E-04	**5.65E-01**	**5.7E-01**
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03	9.4E-05	8.1E-05	5.06E-05	1.45E-02	1.99E-02	3.60E-03	3.46E-02	3.8E-02
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07	3.4E-05	2.9E-05	9.12E-06	3.49E-04	4.79E-04	4.30E-05	9.00E-04	9.4E-04
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04	7.5E-05	6.4E-06	1.67E-05	3.85E-02	5.29E-02	1.70E-02	9.15E-02	1.1E-01
Chromium(III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	6.3E-04	4.9E-06	4.2E-06	3.92E-06	3.75E-05	5.15E-05	1.26E-04	1.02E-04	2.3E-04
Chromium(VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	6.3E-04	5.0E-05	4.3E-05	1.22E-06	3.88E-04	5.32E-04	6.30E-03	1.01E-03	7.3E-03
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05	2.1E-04	1.8E-04	5.63E-05	4.31E-03	5.91E-03	2.60E-04	1.07E-02	1.1E-02
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03	4.9E-05	2.5E-05	1.87E-05	2.03E-02	2.79E-02	3.40E-03	4.83E-02	5.2E-02
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03	3.9E-03	2.0E-04	1.11E-04	3.41E-02	4.68E-02	2.13E-02	8.50E-02	1.1E-01
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03	6.4E-04	5.5E-04	5.39E-03	1.65E-01	2.27E-01	5.40E-02	**3.99E-01**	**4.5E-01**
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04	1.1E-05	9.3E-07	6.84E-08	2.80E-03	3.85E-03	1.92E-05	6.66E-03	6.7E-03
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03	4.7E-05	4.0E-05	1.27E-07	4.36E-02	5.98E-02	1.23E-02	1.04E-01	1.2E-01
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03	5.4E-04	4.2E-04	4.54E-03	3.32E-02	4.55E-02	**4.00E-01**	8.42E-02	**4.8E-01**
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04	1.5E-05	1.3E-06	1.14E-08	3.82E-04	5.25E-04	9.00E-06	9.23E-04	9.3E-04
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04	3.2E-05	2.7E-05	2.02E-08	8.21E-02	1.13E-01	2.58E-07	1.95E-01	1.9E-01
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04	2.9E-07	2.5E-07	1.85E-10	9.03E-06	1.24E-05	4.70E-08	2.20E-05	2.2E-05
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA	3.5E-04	3.0E-04	1.43E-05	3.09E-03	4.24E-03	**NA**	8.00E-03	8.0E-03
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03	8.4E-04	7.2E-04	3.40E-04	4.76E-03	6.54E-03	2.60E-02	1.32E-02	3.9E-02
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02	3.9E-05	3.4E-05	2.48E-08	4.00E-02	5.49E-02	4.70E-06	9.49E-02	9.5E-02
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04	1.3E-02	1.1E-02	8.06E-06	5.25E-02	7.21E-02	1.17E-07	1.48E-01	1.5E-01
Boron	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	0.00E+00	**NA**
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02	6.6E-03	5.7E-03	4.17E-06	2.72E-02	3.73E-02	1.58E-05	7.68E-02	7.7E-02
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA	7.0E-05	6.0E-05	4.43E-08	3.97E-04	5.44E-04	**NA**	1.07E-03	1.1E-03
Dust Pallatives													
Epichlorohydrin	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	**NA**	**NA**	**NA**

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

[1] maximum outdoor air concentration assumed equal to the analytical detection limit

TABLE I-6B: Project Scenario Risk Estimates for the Maximum Rail Corridor Residential Scenario (Adult)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes	ILCR Soil Ingestion	ILCR Soil Dermal	ILCR Soil Dust Inhalation	ILCR Plant (Aboveground)	ILCR Plant (Belowground)	ILCR Air Inhalation	ILCR All Routes
Polycyclic Aromatic Hydrocarbons																				
Carcinogenic PAHs																				
Benzo[a]anthracene	5.5E-04	4.2E-10	6.4E-04	1.5E-06	7.4E-05	5.2E-09	6.5E-09	3.26E-12	4.2E-05	1.3E-07	5.8E-07	4.2E-05	4.2E-05	3.6E-11	4.5E-11	1.3E-15	2.9E-07	9.1E-10	2.9E-11	2.9E-07
Benzo[a]pyrene	1.0E-03	7.7E-10	2.6E-04	1.7E-06	3.0E-05	9.5E-09	1.2E-08	6.00E-12	1.7E-05	1.5E-07	2.3E-07	1.7E-05	1.7E-05	6.6E-10	8.3E-10	2.4E-14	1.2E-06	1.0E-08	1.1E-10	1.2E-06
Benzo[b]fluoranthene	2.1E-04	1.6E-10	9.4E-04	5.5E-07	1.1E-04	2.0E-09	2.5E-09	1.24E-12	6.1E-05	4.9E-08	8.2E-07	6.1E-05	6.1E-05	1.4E-11	1.7E-11	4.9E-16	4.2E-07	3.4E-10	4.1E-11	4.2E-07
Benzo[g,h,i]perylene	3.2E-04	2.5E-10	7.1E-05	2.8E-07	8.3E-06	3.1E-09	3.9E-09	1.93E-12	4.6E-06	2.5E-08	6.5E-08	4.6E-06	4.7E-06	2.1E-12	2.7E-12	7.6E-17	3.2E-09	1.7E-11	3.2E-13	3.2E-09
Benzo[k]fluoranthene	8.5E-04	6.4E-10	2.0E-04	1.4E-06	2.3E-05	8.0E-09	1.0E-08	5.04E-12	1.3E-05	1.3E-07	1.8E-07	1.3E-05	1.3E-05	5.5E-11	7.0E-11	2.0E-15	8.8E-08	8.9E-10	8.8E-12	8.9E-08
Chrysene	1.2E-03	8.8E-10	3.9E-04	3.0E-06	4.5E-05	1.1E-08	1.4E-08	6.92E-12	2.5E-05	2.6E-07	3.5E-07	2.6E-05	2.6E-05	7.6E-12	9.6E-12	2.7E-16	1.7E-08	1.8E-10	1.7E-12	1.8E-08
Dibenzo[a,h]anthracene	2.5E-04	1.9E-10	5.5E-05	1.8E-07	6.2E-06	2.4E-09	3.0E-09	1.51E-12	3.6E-06	1.6E-08	4.8E-08	3.6E-06	3.6E-06	1.6E-10	2.1E-10	6.0E-15	2.5E-07	1.1E-09	2.4E-11	2.5E-07
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04	2.5E-09	3.2E-09	1.57E-12	8.8E-05	1.4E-07	1.3E-06	8.8E-05	8.9E-05	2.3E-13	2.9E-13	8.3E-18	8.1E-09	1.3E-11	8.3E-13	8.1E-09
Indeno[1,2,3-c,d]pyrene	8.4E-04	6.4E-10	1.9E-04	6.4E-07	2.2E-05	7.9E-09	1.0E-08	4.98E-12	1.2E-05	5.7E-08	1.7E-07	1.2E-05	1.3E-05	5.4E-11	6.9E-11	2.0E-15	8.6E-08	3.9E-10	8.4E-12	8.6E-08
Phenanthrene	6.3E-06	4.8E-12	4.4E-04	9.7E-08	5.2E-04	6.0E-11	7.6E-11	3.77E-14	2.9E-05	8.6E-09	4.0E-06	2.9E-05	3.3E-05	4.1E-15	5.2E-15	1.5E-19	2.0E-09	5.9E-13	2.0E-12	2.0E-09
Carcinogenic PAH Mixture											7.7E-06	3.0E-04	2.7E-04							2.3E-06
Non-carcinogenic PAHs																				
Acenaphthene	4.7E-07	3.6E-13	4.0E-04	1.5E-08	4.9E-05	2.2E-12	2.8E-12	1.39E-15	1.3E-05	6.5E-10	1.9E-07	1.3E-05	1.3E-05	NA	NA	NA	NA	NA	NA	NA
Acenaphthylene	1.7E-07	1.3E-13	1.1E-04	5.1E-09	9.0E-05	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	3.0E-06	2.3E-12	2.2E-04	4.7E-08	4.2E-05	2.9E-12	3.6E-12	1.80E-15	1.4E-06	4.2E-10	3.3E-08	1.4E-06	1.4E-06	NA	NA	NA	NA	NA	NA	NA
Fluorene	6.4E-07	4.8E-13	1.9E-04	1.4E-08	4.7E-05	4.5E-12	5.7E-12	2.85E-15	9.3E-06	9.5E-10	2.8E-07	9.3E-06	9.6E-06	NA	NA	NA	NA	NA	NA	NA
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04	2.5E-09	3.2E-09	1.57E-12	8.8E-05	1.4E-07	1.3E-06	8.8E-05	8.9E-05	2.3E-13	2.9E-13	8.3E-18	8.1E-09	1.3E-11	8.3E-13	8.1E-09
Naphthalene	5.9E-09	4.5E-15	7.9E-05	4.2E-10	1.7E-05	8.3E-14	1.1E-13	4.48E-16	7.6E-06	5.6E-11	1.7E-06	7.6E-06	9.3E-06	NA	NA	NA	NA	NA	NA	NA
2-Methylnaphthalene	1.1E-08	8.1E-15	7.4E-05	3.6E-10	8.2E-06	7.5E-13	9.5E-13	4.73E-16	3.6E-05	2.4E-10	4.8E-07	3.6E-05	3.6E-05	NA	NA	NA	NA	NA	NA	NA
Pyrene	4.6E-04	3.5E-10	2.3E-03	4.1E-06	2.7E-04	4.4E-09	5.6E-09	2.77E-12	1.5E-04	3.6E-07	2.1E-06	1.5E-04	1.5E-04	NA	NA	NA	NA	NA	NA	NA
Non-carcinogenic PAH Mixture											6.0E-06	3.0E-04	3.1E-04							
Metals and Metalloids																				
Antimony	2.3E-03	1.7E-09	1.8E-04	6.9E-05	1.2E-05	2.2E-07	1.8E-07	1.36E-10	1.2E-04	6.1E-05	9.6E-07	1.8E-04	1.8E-04	NA	NA	NA	NA	NA	NA	NA
Arsenic	3.0E-02	2.3E-08	1.6E-03	1.8E-04	1.6E-04	2.9E-05	7.3E-06	2.30E-08	1.1E-02	1.6E-03	1.6E-04	1.2E-02	1.2E-02	1.5E-08	4.0E-09	1.5E-10	5.7E-06	8.7E-07	1.3E-07	6.7E-06
Barium	8.0E+00	6.1E-06	5.7E-01	1.8E-01	4.3E-02	1.1E-05	9.7E-06	6.11E-06	5.5E-03	2.4E-03	4.3E-02	7.9E-03	5.1E-02	NA	NA	NA	NA	NA	NA	NA
Beryllium	4.9E-03	3.7E-09	2.4E-04	7.3E-06	3.0E-05	6.9E-07	5.9E-07	1.86E-07	2.3E-04	9.8E-06	1.5E-03	2.5E-04	1.8E-03	NA	NA	8.9E-12	NA	NA	9.1E-09	9.1E-09
Cadmium	2.6E-03	1.9E-09	3.1E-04	1.9E-04	1.8E-05	8.7E-07	7.4E-08	1.94E-07	7.3E-04	6.1E-04	1.8E-03	1.3E-03	3.1E-03	NA	NA	1.9E-11	NA	NA	2.2E-08	2.2E-08
Chromium(III)	2.3E-03	1.7E-09	1.1E-04	2.6E-06	3.7E-04	4.3E-10	3.7E-10	3.49E-10	1.5E-07	4.6E-09	7.4E-05	1.5E-07	7.4E-05	NA	NA	NA	NA	NA	NA	NA
Chromium(VI)	6.9E-02	5.2E-08	3.4E-03	7.8E-05	3.0E-07	2.2E-05	1.9E-05	5.24E-07	7.3E-03	2.3E-04	3.0E-06	7.6E-03	7.6E-03	8.2E-09	7.0E-09	4.0E-09	2.8E-06	8.7E-08	2.9E-09	2.9E-06
Cobalt	2.9E-02	2.2E-08	1.5E-03	8.7E-05	1.6E-04	8.2E-07	7.0E-07	2.20E-07	2.9E-04	2.3E-05	1.6E-03	3.1E-04	1.9E-03	NA	NA	NA	NA	NA	NA	NA
Copper	3.0E-01	2.2E-07	3.2E-02	1.8E-02	1.6E-03	5.9E-07	3.0E-07	2.24E-07	4.4E-04	3.3E-04	1.6E-03	7.7E-04	2.4E-03	NA	NA	NA	NA	NA	NA	NA
Lead	5.1E-02	3.9E-08	2.8E-03	3.4E-04	2.9E-04	9.0E-06	4.6E-07	2.57E-07	6.4E-04	1.1E-04	1.9E-03	7.6E-04	2.7E-03	NA	NA	NA	NA	NA	NA	NA
Manganese	4.4E-01	3.4E-07	3.8E-02	1.7E-02	2.4E-03	8.0E-07	6.9E-07	6.71E-06	4.7E-04	2.8E-04	4.7E-02	7.6E-04	4.8E-02	NA	NA	NA	NA	NA	NA	NA
Molybdenum	1.2E-02	8.8E-09	9.9E-04	4.3E-04	6.2E-05	1.2E-07	1.0E-08	7.31E-10	6.8E-05	4.1E-05	5.2E-06	1.1E-04	1.1E-04	NA	NA	NA	NA	NA	NA	NA
Mercury	1.2E-02	1.4E-09	3.3E-04	2.4E-04	1.0E-05	1.1E-05	9.3E-06	4.54E-09	2.1E-03	2.1E-03	3.4E-05	4.3E-03	4.3E-03	NA	NA	NA	NA	NA	NA	NA
Nickel	9.2E-02	7.0E-08	5.3E-03	8.3E-04	4.9E-04	2.4E-06	1.8E-06	2.00E-05	9.3E-04	2.0E-04	1.4E-01	1.2E-03	1.4E-01	NA	NA	9.1E-11	NA	NA	8.0E-08	8.0E-08
Selenium	1.5E-02	1.1E-08	7.7E-04	5.6E-05	8.0E-05	7.4E-07	6.3E-08	5.63E-10	2.6E-04	2.6E-05	4.0E-06	2.9E-04	2.9E-04	NA	NA	NA	NA	NA	NA	NA
Strontium	5.1E+00	3.9E-06	2.1E+00	1.9E+00	2.7E-02	2.4E-06	2.0E-06	1.51E-09	6.9E-03	8.4E-03	1.1E-05	1.5E-02	1.5E-02	NA	NA	NA	NA	NA	NA	NA
Tin	5.2E-03	3.9E-09	2.7E-04	2.3E-05	2.8E-05	7.3E-10	6.3E-10	4.63E-13	2.7E-07	3.1E-08	3.3E-09	3.0E-07	3.0E-07	NA	NA	NA	NA	NA	NA	NA
Uranium	8.1E-03	6.1E-09	4.0E-04	1.0E-05	4.3E-05	3.8E-06	3.3E-06	1.54E-07	1.3E-03	4.6E-05	1.1E-03	1.3E-03	2.4E-03	NA	NA	NA	NA	NA	NA	NA
Vanadium	2.4E-01	1.8E-07	1.2E-02	2.0E-04	1.3E-03	4.5E-06	3.9E-06	1.83E-06	1.5E-03	3.5E-05	1.3E-02	1.6E-03	1.5E-02	NA	NA	NA	NA	NA	NA	NA
Zinc	3.1E-01	2.4E-07	6.1E-02	4.6E-02	1.7E-03	1.5E-07	1.3E-07	9.24E-11	2.0E-04	2.0E-04	6.5E-07	4.0E-04	4.0E-04	NA	NA	NA	NA	NA	NA	NA
Aluminum	5.2E+01	3.9E-05	2.5E+00	3.1E-02	2.8E-01	4.9E-05	4.2E-05	3.09E-08	1.6E-02	2.8E-04	2.2E-04	1.7E-02	1.7E-02	NA	NA	NA	NA	NA	NA	NA
Boron	4.0E-01	3.0E-07	2.6E-01	2.4E-01	2.2E-03	6.5E-06	5.5E-07	1.01E-09	2.9E-02	3.7E-02	7.2E-06	6.5E-02	6.5E-02	NA	NA	NA	NA	NA	NA	NA
Iron	5.8E+01	4.4E-05	2.8E+00	3.5E-02	3.1E-01	2.0E-05	1.8E-05	1.29E-08	6.8E-03	1.2E-04	9.1E-05	7.0E-03	7.1E-03	NA	NA	NA	NA	NA	NA	NA
Titanium	3.6E+00	2.7E-06	1.8E-01	3.0E-03	1.9E-02	3.4E-07	2.9E-07	2.14E-10	1.1E-04	2.6E-06	1.5E-06	1.2E-04	1.2E-04	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives																				
Epichlorohydrin	9.1E-14	6.9E-20	8.1E-05	2.9E-13	7.4E-02	4.3E-18	3.7E-17	6.93E-20	2.6E-05	1.3E-13	7.4E-02	2.6E-05	7.4E-02	2.6E-22	2.2E-21	8.3E-26	1.6E-09	7.7E-18	1.1E-08	1.3E-08

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

TABLE I-6C: Cumulative (Background+Project) Scenario Risk Estimates for a Maximum Rail Corridor Residential Receptor, Adult
(Based on Maximum Multi-Media Concentrations)

Scenario: Cumulative

	BASELINE HQ Air Inhalation	BASELINE HQ Soil/Vegetation	BASELINE HQ All Routes	PROJECT HQ Air Inhalation	PROJECT HQ Soil/Vegetation	PROJECT HQ All Routes	CUMULATIVE HQ Air Inhalation	CUMULATIVE HQ Soil/Vegetation	CUMULATIVE HQ All Routes
Polycyclic Aromatic Hydrocarbons									
Carcinogenic PAHs									
Benzo[a]anthracene	6.3E-06	1.5E-04	1.6E-04	5.8E-07	4.2E-05	4.2E-05	6.8E-06	1.9E-04	2.0E-04
Benzo[a]pyrene	3.1E-06	1.1E-04	1.1E-04	2.3E-07	1.7E-05	1.8E-05	3.4E-06	1.3E-04	1.3E-04
Benzo[b]fluoranthene	5.5E-06	2.6E-04	2.6E-04	8.2E-07	6.1E-05	6.1E-05	6.3E-06	3.2E-04	3.2E-04
Benzo[g,h,i]perylene	NA	3.8E-05	3.8E-05	6.5E-08	4.6E-06	4.7E-06	NC	4.3E-05	4.3E-05
Benzo[k]fluoranthene	2.3E-06	6.8E-05	7.0E-05	1.8E-07	1.3E-05	1.3E-05	2.5E-06	8.1E-05	8.3E-05
Chrysene	NA	1.6E-04	1.6E-04	3.5E-07	2.6E-05	2.6E-05	NC	1.9E-04	1.9E-04
Dibenzo[a,h]anthracene	NA	8.0E-06	8.0E-06	4.8E-08	3.6E-06	3.7E-06	NC	1.2E-05	1.2E-05
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	1.3E-06	8.8E-05	8.9E-05	4.1E-05	7.0E-04	7.4E-04
Indeno[1,2,3-c,d]pyrene	1.5E-05	3.8E-05	5.2E-05	1.7E-07	1.3E-05	1.3E-05	1.5E-05	5.0E-05	6.5E-05
Phenanthrene	1.3E-04	1.2E-03	1.4E-03	4.0E-06	2.9E-05	3.3E-05	1.3E-04	1.3E-03	1.4E-03
Carcinogenic PAH Mixture	2.0E-04	2.7E-03	1.5E-03	7.7E-06	3.0E-04	2.7E-04	2.0E-04	3.0E-03	3.2E-03
Non-carcinogenic PAHs									
Acenaphthene	8.6E-06	4.8E-05	5.7E-05	1.9E-07	1.3E-05	1.3E-05	8.8E-06	6.1E-05	7.0E-05
Acenaphthylene	NA	NA	NA	NA	NA	NA	NC	NA	NA
Anthracene	1.3E-06	1.9E-05	2.0E-05	3.3E-08	1.4E-06	1.4E-06	1.3E-06	2.1E-05	2.2E-05
Fluorene	3.0E-05	1.0E-04	1.3E-04	2.8E-07	9.3E-06	9.6E-06	3.0E-05	1.1E-04	1.4E-04
Fluoranthene	4.0E-05	6.1E-04	6.5E-04	1.3E-06	8.8E-05	8.9E-05	4.1E-05	7.0E-04	7.4E-04
Naphthalene	1.5E-02	3.3E-04	1.5E-02	1.7E-06	7.6E-06	9.3E-06	1.5E-02	3.4E-04	1.5E-02
2-Methylnaphthalene	NA	7.9E-04	7.9E-04	4.8E-07	3.6E-05	3.6E-05	NC	8.2E-04	8.2E-04
Pyrene	3.8E-05	9.6E-04	9.9E-04	2.1E-06	1.5E-04	1.5E-04	4.0E-05	1.1E-03	1.1E-03
Non-carcinogenic PAH Mixture	1.5E-02	2.9E-03	1.8E-02	6.0E-06	3.0E-04	3.1E-04	1.5E-02	3.2E-03	1.8E-02
Metals and Metalloids									
Antimony	NA	2.4E-02	2.4E-02	9.6E-07	1.8E-04	1.8E-04	NC	2.4E-02	2.4E-02
Arsenic	4.9E-04	**5.7E-01**	**5.7E-01**	1.6E-04	1.2E-02	1.3E-02	6.5E-04	**5.8E-01**	**5.8E-01**
Barium	3.6E-03	3.5E-02	3.8E-02	4.3E-02	8.0E-03	5.1E-02	4.7E-02	4.3E-02	8.9E-02
Beryllium	4.3E-05	9.0E-04	9.4E-04	1.5E-03	2.5E-04	4.0E-04	1.6E-03	1.2E-03	1.3E-03
Cadmium	1.7E-02	9.1E-02	1.1E-01	1.8E-03	1.4E-03	3.1E-03	1.9E-02	9.3E-02	1.1E-01
Chromium(III)	1.3E-04	1.0E-04	2.3E-04	7.4E-05	1.6E-07	7.4E-05	2.0E-04	1.0E-04	3.0E-04
Chromium(VI)	6.3E-03	1.0E-03	7.3E-03	3.0E-06	7.8E-03	7.8E-03	6.3E-03	8.8E-03	1.5E-02
Cobalt	2.6E-04	1.1E-02	1.1E-02	1.6E-03	3.2E-04	1.9E-03	1.8E-03	1.1E-02	1.3E-02
Copper	3.4E-03	4.8E-02	5.2E-02	1.6E-03	7.8E-04	2.4E-03	5.0E-03	4.9E-02	5.4E-02
Lead	2.1E-02	8.5E-02	1.1E-01	1.9E-03	8.4E-04	2.7E-03	2.3E-02	8.6E-02	1.1E-01
Manganese	5.4E-02	**4.0E-01**	**4.5E-01**	4.7E-02	7.7E-04	4.8E-02	1.0E-01	**4.0E-01**	**5.0E-01**
Molybdenum	1.9E-05	6.7E-03	6.7E-03	5.2E-06	1.1E-04	1.2E-04	2.4E-05	6.8E-03	6.8E-03
Mercury	1.2E-02	1.0E-01	1.2E-01	3.4E-05	4.4E-03	4.4E-03	1.2E-02	1.1E-01	1.2E-01
Nickel	**4.0E-01**	8.4E-02	**4.8E-01**	1.4E-01	1.2E-03	1.4E-01	**5.4E-01**	8.5E-02	**6.3E-01**
Selenium	9.0E-06	9.2E-04	9.3E-04	4.0E-06	2.9E-04	3.0E-04	1.3E-05	1.2E-03	1.2E-03
Strontium	2.6E-07	1.9E-01	1.9E-01	1.1E-05	1.5E-02	1.5E-02	1.1E-05	**2.1E-01**	**2.1E-01**
Tin	4.7E-08	2.2E-05	2.2E-05	3.3E-09	3.0E-07	3.1E-07	5.0E-08	2.2E-05	2.2E-05
Uranium	NA	8.0E-03	8.0E-03	1.1E-03	1.4E-03	1.4E-03	NC	9.4E-03	9.4E-03
Vanadium	2.6E-02	1.3E-02	3.9E-02	1.3E-02	1.6E-03	1.5E-02	3.9E-02	1.5E-02	5.4E-02
Zinc	4.7E-06	9.5E-02	9.5E-02	6.5E-07	4.0E-04	4.0E-04	5.3E-06	9.5E-02	9.5E-02
Aluminum	1.2E-07	1.5E-01	1.5E-01	2.2E-04	1.7E-02	1.7E-02	2.2E-04	1.7E-01	1.7E-01
Boron	NA	NA	NA	7.2E-06	6.5E-02	6.5E-02	NC	NC	NC
Iron	1.6E-05	7.7E-02	7.7E-02	9.1E-05	7.2E-03	7.2E-03	1.1E-04	8.4E-02	8.4E-02
Titanium	NA	1.1E-03	1.1E-03	1.5E-06	1.2E-04	1.2E-04	NC	1.2E-03	1.2E-03
Dust Pallatives									
Epichlorohydrin	NA	NA	NA	7.4E-02	2.6E-05	7.4E-02	NC	NC	NC

HQ = Hazard Quotient

NA = no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE I-7A: Baseline Scenario Risk Estimates for a Industrial Receptor (Adult)
(Based on Background Multi-Media Concentrations)

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Air Inhalation	HQ Soil Pathways	HQ All Routes
Polycyclic Aromatic Hydrocarbons											
Carcinogenic PAHs											
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04	2.1E-06	2.7E-06	5.56E-10	1.72E-06	4.79E-06	6.5E-06
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04	2.5E-06	3.2E-06	6.54E-10	8.60E-07	5.64E-06	6.5E-06
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04	3.7E-06	4.7E-06	9.80E-10	1.51E-06	8.46E-06	1.0E-05
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA	1.6E-06	2.0E-06	4.09E-10	NA	3.52E-06	3.5E-06
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04	1.5E-06	1.9E-06	3.92E-10	6.45E-07	3.38E-06	4.0E-06
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA	2.5E-06	3.2E-06	6.54E-10	NA	5.64E-06	5.6E-06
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA	3.7E-07	4.7E-07	9.80E-11	NA	8.46E-07	8.5E-07
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	4.0E-06	5.1E-06	1.05E-09	1.10E-05	9.09E-06	2.0E-05
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03	1.7E-06	2.1E-06	4.41E-10	4.09E-06	3.81E-06	7.9E-06
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02	3.2E-06	4.1E-06	8.50E-10	3.44E-05	7.33E-06	4.2E-05
Carcinogenic PAH Mixture									5.42E-05	5.25E-05	6.5E-05
Non-carcinogenic PAHs											
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03	6.2E-08	7.9E-08	1.63E-11	2.37E-06	1.41E-07	2.5E-06
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA	NA	NA	NA	NA	NA	NA
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03	5.0E-08	6.3E-08	1.31E-11	3.44E-07	1.13E-07	4.6E-07
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03	1.9E-07	2.4E-07	4.90E-11	8.22E-06	4.23E-07	8.6E-06
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03	4.0E-06	5.1E-06	1.05E-09	1.10E-05	9.09E-06	2.0E-05
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01	1.9E-07	2.4E-07	4.18E-10	4.12E-03	4.23E-07	4.1E-03
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA	9.3E-07	1.2E-06	2.45E-10	NA	2.11E-06	2.1E-06
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03	4.4E-06	5.5E-06	1.14E-09	1.05E-05	9.87E-06	2.0E-05
Non-carcinogenic PAH Mixture									4.15E-03	2.22E-05	4.2E-03
Metals and Metalloids											
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA	3.2E-05	2.7E-05	8.33E-09	NA	5.89E-05	5.9E-05
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04	3.8E-03	9.7E-04	1.27E-06	1.35E-04	4.74E-03	4.9E-03
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03	6.2E-05	5.3E-05	1.39E-05	9.89E-04	1.29E-04	1.1E-03
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07	2.2E-05	1.9E-05	2.51E-06	1.18E-05	4.40E-05	5.6E-05
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04	4.9E-05	4.2E-06	4.59E-06	4.67E-03	5.83E-05	4.7E-03
Chromium(III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	6.3E-04	3.2E-06	2.7E-06	1.08E-06	3.46E-05	7.04E-06	4.2E-05
Chromium(VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	6.3E-04	3.3E-05	2.8E-05	3.34E-07	1.73E-03	6.19E-05	1.8E-03
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05	1.4E-04	1.2E-04	1.55E-05	7.14E-05	2.72E-04	3.4E-04
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03	3.3E-05	1.7E-05	5.14E-06	9.34E-04	5.45E-05	9.9E-04
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03	2.6E-03	1.3E-04	3.05E-05	5.86E-03	2.72E-03	8.6E-03
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03	4.2E-04	3.6E-04	1.48E-03	1.48E-02	2.27E-03	1.7E-02
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04	7.2E-06	6.2E-07	1.88E-08	5.27E-06	7.83E-06	1.3E-05
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03	3.1E-05	2.7E-05	3.48E-08	3.39E-03	5.77E-05	3.4E-03
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03	3.5E-04	2.8E-04	1.25E-03	1.10E-01	1.88E-03	1.1E-01
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04	9.8E-06	8.4E-07	3.13E-09	2.47E-06	1.07E-05	1.3E-05
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04	2.1E-05	1.8E-05	5.54E-09	7.10E-08	3.91E-05	3.9E-05
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04	1.9E-07	1.7E-07	5.07E-11	1.29E-08	3.58E-07	3.7E-07
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA	2.3E-04	2.0E-04	3.91E-06	NA	4.37E-04	4.4E-04
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03	5.6E-04	4.8E-04	9.33E-05	7.14E-03	1.12E-03	8.3E-03
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02	2.6E-05	2.2E-05	6.81E-09	1.29E-06	4.81E-05	4.9E-05
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04	8.4E-03	7.2E-03	2.21E-06	3.23E-08	1.56E-02	1.6E-02
Boron	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02	4.4E-03	3.7E-03	1.15E-06	4.35E-06	8.10E-03	8.1E-03
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA	4.6E-05	4.0E-05	1.22E-08	NA	8.59E-05	8.6E-05
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Others											
Hexachlorobenzene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
PCBs	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sulphate	NA	NA	NA	NA	9.0E-01	NA	NA	NA	NA	NA	NA
Hexachlorobenzene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA
Naphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA
2-Methylnaphthalene (gas phase)	NA	NA	NA	NA *	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives											
Epichlorohydrin	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

[1] maximum outdoor air concentration assumed equal to the analytical detection limit

TABLE I-7B: Project Scenario Risk Estimates for a Industrial Receptor (Adult)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Air Inhalation	HQ Soil	HQ All Routes	ILCR Soil Ingestion	ILCR Soil Dermal	ILCR Soil Dust Inhalation	ILCR Air Inhalation	ILCR All Routes
Polycyclic Aromatic Hydrocarbons														
Carcinogenic PAHs														
Benzo[a]anthracene	2.3E-03	1.7E-09	4.3E-03	1.5E-08	1.9E-08	4.02E-12	9.88E-06	3.47E-08	9.9E-06	4.6E-11	5.9E-11	7.0E-16	4.9E-10	5.9E-10
Benzo[a]pyrene	4.2E-03	3.2E-09	7.5E-05	2.8E-08	3.6E-08	7.42E-12	1.75E-07	6.40E-08	2.4E-07	8.5E-10	1.1E-09	1.3E-14	8.6E-11	2.0E-09
Benzo[b]fluoranthene	8.7E-04	6.6E-10	2.7E-04	5.8E-09	7.4E-09	1.53E-12	6.17E-07	1.32E-08	6.3E-07	1.8E-11	2.2E-11	2.6E-16	3.1E-11	7.0E-11
Benzo[g,h,i]perylene	1.4E-03	1.0E-09	2.2E-05	9.1E-09	1.1E-08	2.38E-12	5.02E-08	2.05E-08	7.1E-08	2.7E-12	3.5E-12	4.1E-17	2.5E-13	6.4E-12
Benzo[k]fluoranthene	3.5E-03	2.7E-09	5.7E-05	2.4E-08	3.0E-08	6.23E-12	1.32E-07	5.37E-08	1.9E-07	7.2E-11	9.1E-11	1.1E-15	6.5E-12	1.7E-10
Chrysene	4.9E-03	3.7E-09	1.1E-04	3.3E-08	4.1E-08	8.55E-12	2.58E-07	7.38E-08	3.3E-07	9.8E-12	1.2E-11	1.5E-16	1.3E-12	2.4E-11
Dibenzo[a,h]anthracene	1.1E-03	8.0E-10	1.6E-05	7.1E-09	9.0E-09	1.86E-12	3.59E-08	1.61E-08	5.2E-08	2.1E-10	2.7E-10	3.2E-15	1.8E-11	5.0E-10
Fluoranthene	1.5E-03	1.1E-09	5.2E-04	7.4E-09	9.4E-09	1.94E-12	9.07E-07	1.67E-08	9.2E-07	3.0E-13	3.8E-13	4.5E-18	6.0E-13	1.3E-12
Indeno[1,2,3-c,d]pyrene	3.5E-03	2.7E-09	5.7E-05	2.3E-08	3.0E-08	6.16E-12	1.32E-07	5.32E-08	1.9E-07	7.1E-11	9.0E-11	1.1E-15	6.6E-12	1.7E-10
Phenanthrene	2.6E-05	2.0E-11	7.0E-04	1.8E-10	2.2E-10	4.64E-14	1.62E-06	4.00E-10	1.6E-06	5.3E-15	6.8E-15	8.0E-20	8.0E-13	8.2E-13
Carcinogenic PAH Mixture							1.38E-05	3.46E-07	1.3E-05				8.6E-11	3.6E-09
Non-carcinogenic PAHs														
Acenaphthene	2.0E-06	1.5E-12	1.2E-04	6.5E-12	8.3E-12	1.72E-15	1.40E-07	1.48E-11	1.4E-07	NA	NA	NA	NA	NA
Acenaphthylene	7.0E-07	5.3E-13	1.3E-04	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	1.3E-05	9.6E-12	4.2E-03	8.5E-12	1.1E-11	2.23E-15	9.62E-07	1.92E-11	9.6E-07	NA	NA	NA	NA	NA
Fluorene	2.7E-06	2.0E-12	9.5E-05	1.3E-11	1.7E-11	3.52E-15	1.65E-07	3.04E-11	1.7E-07	NA	NA	NA	NA	NA
Fluoranthene	1.5E-03	1.1E-09	5.2E-04	7.4E-09	9.4E-09	1.94E-12	9.07E-07	1.67E-08	9.2E-07	3.0E-13	3.8E-13	4.5E-18	6.0E-13	1.3E-12
Naphthalene	2.5E-08	1.9E-14	5.2E-04	2.6E-13	3.2E-13	5.73E-16	1.54E-05	5.80E-13	1.5E-05	NA	NA	NA	NA	NA
2-Methylnaphthalene	4.4E-08	3.4E-14	2.1E-05	2.2E-12	2.8E-12	5.85E-16	3.60E-07	5.04E-12	3.6E-07	NA	NA	NA	NA	NA
Pyrene	1.9E-03	1.5E-09	6.7E-04	1.3E-08	1.6E-08	3.42E-12	1.54E-06	2.95E-08	1.6E-06	NA	NA	NA	NA	NA
Non-carcinogenic PAH Mixture							1.95E-05	4.63E-08	2.0E-05					
Metals and Metalloids														
Antimony	9.9E-03	7.5E-09	2.2E-04	6.6E-07	5.7E-07	1.74E-10	5.10E-06	1.23E-06	6.3E-06	NA	NA	NA	NA	NA
Arsenic	1.3E-01	9.6E-08	4.2E-04	8.5E-05	2.2E-05	2.85E-08	1.24E-04	1.07E-04	2.3E-04	2.0E-08	5.1E-09	8.0E-11	9.9E-08	1.2E-07
Barium	3.4E+01	2.6E-05	1.1E-01	3.4E-05	2.9E-05	7.55E-06	3.22E-02	7.02E-05	3.2E-02	NA	NA	NA	NA	NA
Beryllium	2.0E-02	1.5E-08	7.1E-05	2.0E-06	1.8E-06	2.29E-07	1.05E-03	4.02E-06	1.1E-03	NA	NA	4.8E-12	6.3E-09	6.3E-09
Cadmium	1.1E-02	8.1E-09	1.1E-04	2.6E-06	2.2E-07	2.40E-07	3.24E-03	3.04E-06	3.2E-03	NA	NA	1.0E-11	4.0E-08	4.0E-08
Chromium(III)	9.6E-03	7.3E-09	9.4E-04	1.3E-09	1.1E-09	4.31E-10	5.59E-05	2.81E-09	5.6E-05	NA	NA	NA	NA	NA
Chromium(VI)	2.9E-01	2.2E-07	5.7E-06	6.4E-05	5.5E-05	6.48E-07	1.70E-05	1.20E-04	1.4E-04	1.1E-08	9.1E-09	2.2E-09	1.6E-08	3.8E-08
Cobalt	1.2E-01	9.2E-08	3.9E-04	2.4E-06	2.1E-06	2.72E-07	1.16E-03	4.78E-06	1.2E-03	NA	NA	NA	NA	NA
Copper	1.2E+00	9.4E-07	4.0E-03	1.8E-06	9.0E-07	2.78E-07	1.20E-03	2.94E-06	1.2E-03	NA	NA	NA	NA	NA
Lead	2.1E-01	1.6E-07	7.4E-04	2.7E-05	1.4E-06	3.18E-07	1.46E-03	2.83E-05	1.5E-03	NA	NA	NA	NA	NA
Manganese	1.8E+00	1.4E-06	6.0E-03	2.4E-06	2.0E-06	8.30E-06	3.54E-02	1.27E-05	3.5E-02	NA	NA	NA	NA	NA
Molybdenum	4.8E-02	3.7E-08	2.0E-04	3.5E-07	3.0E-08	9.06E-10	4.93E-06	3.77E-07	5.3E-06	NA	NA	NA	NA	NA
Mercury	7.5E-03	5.7E-09	2.7E-05	5.0E-06	4.3E-06	5.62E-09	2.68E-05	9.32E-06	3.6E-05	NA	NA	NA	NA	NA
Nickel	3.8E-01	2.9E-07	1.2E-03	7.0E-06	5.5E-06	2.47E-05	1.05E-01	3.72E-05	1.1E-01	NA	NA	4.9E-11	6.0E-08	6.0E-08
Selenium	6.2E-02	4.7E-08	2.0E-04	2.2E-06	1.9E-07	6.97E-10	2.97E-06	2.37E-06	5.3E-06	NA	NA	NA	NA	NA
Strontium	2.1E+01	1.6E-05	6.9E-02	7.1E-06	6.1E-06	1.86E-09	7.94E-06	1.32E-05	2.1E-05	NA	NA	NA	NA	NA
Tin	2.2E-02	1.7E-08	1.8E-04	2.2E-09	1.9E-09	5.78E-13	6.22E-09	4.08E-09	1.0E-08	NA	NA	NA	NA	NA
Uranium	3.4E-02	2.6E-08	1.1E-04	1.1E-05	9.7E-06	1.90E-07	8.10E-04	2.12E-05	8.3E-04	NA	NA	NA	NA	NA
Vanadium	1.0E+00	7.7E-07	3.5E-03	1.3E-05	1.2E-05	2.27E-06	1.04E-02	2.73E-05	1.0E-02	NA	NA	NA	NA	NA
Zinc	1.3E+00	9.9E-07	4.8E-03	4.4E-07	3.7E-07	1.14E-10	5.58E-07	8.07E-07	1.4E-06	NA	NA	NA	NA	NA
Aluminum	2.2E+02	1.6E-04	7.0E-01	1.5E-04	1.2E-04	3.82E-08	1.63E-04	2.70E-04	4.3E-04	NA	NA	NA	NA	NA
Boron	1.7E+00	1.3E-06	5.4E-03	1.9E-05	1.6E-06	1.26E-09	5.35E-06	2.09E-05	2.6E-05	NA	NA	NA	NA	NA
Iron	2.4E+02	1.8E-04	7.8E-01	6.1E-05	5.2E-05	1.60E-08	6.81E-05	1.13E-04	1.8E-04	NA	NA	NA	NA	NA
Titanium	1.5E+01	1.1E-05	4.9E-02	1.0E-06	8.6E-07	2.65E-10	1.13E-06	1.87E-06	3.0E-06	NA	NA	NA	NA	NA
Indium	2.1E-04	1.6E-10	1.2E-04	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	5.2E-04	4.0E-10	2.9E-04	2.1E-10	1.8E-10	5.50E-14	4.09E-08	3.89E-10	4.1E-08	NA	NA	NA	NA	NA
Others														
Hexachlorobenzene	8.6E-09	6.5E-15	2.9E-04	6.4E-12	5.5E-12	1.68E-15	7.57E-05	1.19E-11	7.6E-05	1.2E-15	1.0E-15	3.9E-19	5.0E-09	5.0E-09
PCBs	3.2E-07	2.4E-13	1.7E-07	2.8E-05	2.4E-05	7.34E-09	5.04E-03	5.18E-05	5.1E-03	NA	NA	NA	NA	NA
Sulphate	6.6E-02	5.0E-08	3.7E-02	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives														
Epichlorohydrin	6.0E-10	4.5E-16	6.0E-05	2.0E-14	1.7E-13	1.34E-16	1.78E-05	1.91E-13	1.8E-05	5.2E-19	4.4E-18	7.0E-23	2.7E-12	2.7E-12

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

TABLE I-7C: Cumulative (Background+Project) Scenario Risk Estimates for an Industrial Receptor, Adult
(Based on Maximum Multi-Media Concentrations)

Scenario: Cumulative

	BASELINE HQ Air Inhalation	BASELINE HQ Soil	BASELINE HQ All Routes	PROJECT HQ Air Inhalation	PROJECT HQ Soil	PROJECT HQ All Routes	CUMULATIVE HQ Air Inhalation	CUMULATIVE HQ Soil/Vegetation	CUMULATIVE HQ All Routes
Polycyclic Aromatic Hydrocarbons									
Carcinogenic PAHs									
Benzo[a]anthracene	1.7E-06	4.8E-06	6.5E-06	9.9E-06	3.5E-08	9.9E-06	1.2E-05	4.8E-06	1.6E-05
Benzo[a]pyrene	8.6E-07	5.6E-06	6.5E-06	1.7E-07	6.4E-08	2.4E-07	1.0E-06	5.7E-06	6.7E-06
Benzo[b]fluoranthene	1.5E-06	8.5E-06	1.0E-05	6.2E-07	1.3E-08	6.3E-07	2.1E-06	8.5E-06	1.1E-05
Benzo[g,h,i]perylene	NA	3.5E-06	3.5E-06	5.0E-08	2.1E-08	7.1E-08	NC	3.5E-06	3.5E-06
Benzo[k]fluoranthene	6.5E-07	3.4E-06	4.0E-06	1.3E-07	5.4E-08	1.9E-07	7.8E-07	3.4E-06	4.2E-06
Chrysene	NA	5.6E-06	5.6E-06	2.6E-07	7.4E-08	3.3E-07	NC	5.7E-06	5.7E-06
Dibenzo[a,h]anthracene	NA	8.5E-07	8.5E-07	3.6E-08	1.6E-08	5.2E-08	NC	8.6E-07	8.6E-07
Fluoranthene	1.1E-05	9.1E-06	2.0E-05	9.1E-07	1.7E-08	9.2E-07	1.2E-05	9.1E-06	2.1E-05
Indeno[1,2,3-c,d]pyrene	4.1E-06	3.8E-06	7.9E-06	1.3E-07	5.3E-08	1.9E-07	4.2E-06	3.9E-06	8.1E-06
Phenanthrene	3.4E-05	7.3E-06	4.2E-05	1.6E-06	4.0E-10	1.6E-06	3.6E-05	7.3E-06	4.3E-05
Carcinogenic PAH Mixture	5.4E-05	5.3E-05	6.5E-05	1.4E-05	3.5E-07	1.3E-05	6.8E-05	5.3E-05	1.2E-04
Non-carcinogenic PAHs									
Acenaphthene	2.4E-06	1.4E-07	2.5E-06	1.4E-07	1.5E-11	1.4E-07	2.5E-06	1.4E-07	2.6E-06
Acenaphthylene	NA	NA	NA	NA	NA	NA	NA	NA	NA
Anthracene	3.4E-07	1.1E-07	4.6E-07	9.6E-07	1.9E-11	9.6E-07	1.3E-06	1.1E-07	1.4E-06
Fluorene	8.2E-06	4.2E-07	8.6E-06	1.7E-07	3.0E-11	1.7E-07	8.4E-06	4.2E-07	8.8E-06
Fluoranthene	1.1E-05	9.1E-06	2.0E-05	9.1E-07	1.7E-08	9.2E-07	1.2E-05	9.1E-06	2.1E-05
Naphthalene	4.1E-03	4.2E-07	4.1E-03	1.5E-05	5.8E-13	1.5E-05	4.1E-03	4.2E-07	4.1E-03
2-Methylnaphthalene	NA	2.1E-06	2.1E-06	3.6E-07	5.0E-12	3.6E-07	NC	2.1E-06	2.1E-06
Pyrene	1.1E-05	9.9E-06	2.0E-05	1.5E-06	2.9E-08	1.6E-06	1.2E-05	9.9E-06	2.2E-05
Non-carcinogenic PAH Mixture	4.2E-03	2.2E-05	4.2E-03	1.9E-05	4.6E-08	2.0E-05	4.2E-03	2.2E-05	4.2E-03
Metals and Metalloids									
Antimony	NA	5.9E-05	5.9E-05	5.1E-06	1.2E-06	6.3E-06	NC	6.0E-05	6.5E-05
Arsenic	1.3E-04	4.7E-03	4.9E-03	1.2E-04	1.1E-04	2.3E-04	2.6E-04	4.9E-03	5.1E-03
Barium	9.9E-04	1.3E-04	1.1E-03	3.2E-02	7.0E-05	3.2E-02	3.3E-02	2.0E-04	3.3E-02
Beryllium	1.2E-05	4.4E-05	5.6E-05	1.1E-03	4.0E-06	1.1E-03	1.1E-03	4.8E-05	1.1E-03
Cadmium	4.7E-03	5.8E-05	4.7E-03	3.2E-03	3.0E-06	3.2E-03	7.9E-03	6.1E-05	8.0E-03
Chromium(III)	3.5E-05	7.0E-06	4.2E-05	5.6E-05	2.8E-09	5.6E-05	9.0E-05	7.0E-06	9.8E-05
Chromium(VI)	1.7E-03	6.2E-05	1.8E-03	1.7E-05	1.2E-04	1.4E-04	1.7E-03	1.8E-04	1.9E-03
Cobalt	7.1E-05	2.7E-04	3.4E-04	1.2E-03	4.8E-06	1.2E-03	1.2E-03	2.8E-04	1.5E-03
Copper	9.3E-04	5.4E-05	9.9E-04	1.2E-03	2.9E-06	1.2E-03	2.1E-03	5.7E-05	2.2E-03
Lead	5.9E-03	2.7E-03	8.6E-03	1.5E-03	2.8E-05	1.5E-03	7.3E-03	2.7E-03	1.0E-02
Manganese	1.5E-02	2.3E-03	1.7E-02	3.5E-02	1.3E-05	3.5E-02	5.0E-02	2.3E-03	5.3E-02
Molybdenum	5.3E-06	7.8E-06	1.3E-05	4.9E-06	3.8E-07	5.3E-06	1.0E-05	8.2E-06	1.8E-05
Mercury	3.4E-03	5.8E-05	3.4E-03	2.7E-05	9.3E-06	3.6E-05	3.4E-03	6.7E-05	3.5E-03
Nickel	1.1E-01	1.9E-03	1.1E-01	1.1E-01	3.7E-05	1.1E-01	**2.2E-01**	1.9E-03	**2.2E-01**
Selenium	2.5E-06	1.1E-05	1.3E-05	3.0E-06	2.4E-06	5.3E-06	5.4E-06	1.3E-05	1.8E-05
Strontium	7.1E-08	3.9E-05	3.9E-05	7.9E-06	1.3E-05	2.1E-05	8.0E-06	5.2E-05	6.0E-05
Tin	1.3E-08	3.6E-07	3.7E-07	6.2E-09	4.1E-09	1.0E-08	1.9E-08	3.6E-07	3.8E-07
Uranium	NA	4.4E-04	4.4E-04	8.1E-04	2.1E-05	8.3E-04	NC	4.6E-04	1.3E-03
Vanadium	7.1E-03	1.1E-03	8.3E-03	1.0E-02	2.7E-05	1.0E-02	1.8E-02	1.2E-03	1.9E-02
Zinc	1.3E-06	4.8E-05	4.9E-05	5.6E-07	8.1E-07	1.4E-06	1.8E-06	4.9E-05	5.1E-05
Aluminum	3.2E-08	1.6E-02	1.6E-02	1.6E-04	2.7E-04	4.3E-04	1.6E-04	1.6E-02	1.6E-02
Boron	NA	NA	NA	5.3E-06	2.1E-05	2.6E-05	NC	NC	NC
Iron	4.4E-06	8.1E-03	8.1E-03	6.8E-05	1.1E-04	1.8E-04	7.2E-05	8.2E-03	8.3E-03
Titanium	NA	8.6E-05	8.6E-05	1.1E-06	1.9E-06	3.0E-06	NC	8.8E-05	8.9E-05
Indium	NA	NA	NA	NA	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	4.1E-08	3.9E-10	4.1E-08	NC	NC	NC
Others									
Hexachlorobenzene	NA	NA	NA	7.6E-05	1.2E-11	7.6E-05	NC	NC	NC
PCBs	NA	NA	NA	5.0E-03	5.2E-05	5.1E-03	NC	NC	NC
Sulphate	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dust Pallatives									
Epichlorohydrin	NA	NA	NA	1.8E-05	1.9E-13	1.8E-05	NC	NC	NC

HQ = Hazard Quotient

NA = no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

APPENDIX II

Exposure Modelling Details

Table II-1A Summary of Multimedia Exposure Point Concentrations (EPCs) for the Baseline Scenario **Scenario: Baseline**

Parameter	C_s mg/kg	C_{dust} ug/m^3	$C_{plant\ (w/w)}$ mg/kg	$Pr_{root\ (w/w)}$ mg/kg	C_{air} ug/m^3
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	3.4E-01	2.6E-07	9.3E-04	9.3E-04	8.0E-04
Benzo[a]pyrene	4.0E-01	3.0E-07	6.7E-04	6.7E-04	4.0E-04
Benzo[b]fluoranthene	6.0E-01	4.6E-07	1.6E-03	1.6E-03	7.0E-04
Benzo[g,h,i]perylene	2.5E-01	1.9E-07	2.1E-04	2.1E-04	NA
Benzo[k]fluoranthene	2.4E-01	1.8E-07	4.1E-04	4.1E-04	3.0E-04
Chrysene	4.0E-01	3.0E-07	1.0E-03	1.0E-03	NA
Dibenzo[a,h]anthracene	6.0E-02	4.6E-08	4.4E-05	4.4E-05	NA
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03
Indeno[1,2,3-c,d]pyrene	2.7E-01	2.1E-07	2.1E-04	2.1E-04	1.9E-03
Phenanthrene	5.2E-01	4.0E-07	8.0E-03	8.0E-03	1.6E-02
Non-carcinogenic PAHs					
Acenaphthene	2.0E-02	1.5E-08	6.3E-04	6.3E-04	2.2E-03
Acenaphthylene	3.0E-02	2.3E-08	9.2E-04	9.2E-04	NA
Anthracene	8.0E-02	6.1E-08	1.2E-03	1.2E-03	1.6E-03
Fluorene	4.0E-02	3.0E-08	8.9E-04	8.9E-04	5.1E-03
Fluoranthene	8.6E-01	6.5E-07	5.2E-03	5.2E-03	6.8E-03
Naphthalene	2.0E-02	1.5E-08	1.4E-03	1.4E-03	1.5E-01
2-Methylnaphthalene	2.0E-02	1.5E-08	6.8E-04	6.8E-04	NA
Pyrene	7.0E-01	5.3E-07	6.1E-03	6.1E-03	4.9E-03
Metals and Metalliods					
Antimony	5.1E-01	3.9E-07	1.5E-02	1.5E-02	NA
Arsenic	6.1E+00	4.6E-06	3.6E-02	3.6E-02	4.9E-04
Barium	6.7E+01	5.1E-05	1.5E+00	1.5E+00	3.6E-03
Beryllium	2.4E-01	1.8E-07	3.6E-04	3.6E-04	8.6E-07
Cadmium	2.2E-01	1.7E-07	1.7E-02	1.7E-02	1.7E-04
Chromium (III)	2.6E+01	2.0E-05	2.9E-02	2.9E-02	*6.3E-04*
Chromium (VI)	1.6E-01	1.2E-07	1.8E-04	1.8E-04	*6.3E-04*
Cobalt	7.4E+00	5.6E-06	2.2E-02	2.2E-02	2.6E-05
Copper	2.5E+01	1.9E-05	1.5E+00	1.5E+00	3.4E-03
Lead	2.2E+01	1.7E-05	1.5E-01	1.5E-01	3.2E-03
Manganese	3.5E+02	2.7E-04	1.3E+01	1.3E+01	2.7E-03
Mercury	5.0E-02	3.8E-08	6.8E-03	6.8E-03	3.7E-03
Molybdenum	1.1E+00	8.2E-07	4.1E-02	4.1E-02	2.3E-04
Nickel	2.1E+01	1.6E-05	1.9E-01	1.9E-01	1.4E-03
Selenium	3.0E-01	2.3E-07	1.1E-03	1.1E-03	1.8E-04
Strontium	6.8E+01	5.1E-05	2.5E+01	2.5E+01	6.6E-04
Silver	1.0E-01	7.6E-08	6.0E-03	6.0E-03	NA
Tin	2.1E+00	1.6E-06	9.3E-03	9.3E-03	4.0E-04
Uranium	7.5E-01	5.7E-07	9.6E-04	9.6E-04	NA
Vanadium	4.5E+01	3.4E-05	3.7E-02	3.7E-02	2.6E-03
Zinc	8.3E+01	6.3E-05	1.2E+01	1.2E+01	1.2E-02
Aluminum	1.4E+04	1.0E-02	8.1E+00	8.1E+00	1.5E-04
Boron	NA	NA	NA	NA	NA
Iron	1.9E+04	1.4E-02	1.1E+01	1.1E+01	5.4E-02
Titanium	7.4E+02	5.7E-04	6.1E-01	6.1E-01	NA
Indium	NA	NA	NA	NA	NA
Lanthanum	NA	NA	NA	NA	NA
Others					
PCBs	NA	NA	NA	NA	NA
Sulfate	NA	NA	NA	NA	9.0E-01
Hexachlorobenzene (gas phase)	NA	NA	NA	NA *	NA
Dust Pallatives					
Epichlorohydrin	NA	NA	NA	NA	NA

Notes:
- NA / Not applicable/available for this parameter
Italics Concentation for total chromium
* Root vegetables are assumed to be protected from air-to-plant transfer (US EPA OWS 2005).

Table II-2A Soil Concentrations for Baseline Scenario **Scenario:** **Baseline**

Parameter	D_{tot} mg/m^2/yr	Zs m	BD kg/m^3	Ds mg/kg/yr	kt yrs^{-1}	tD yrs	† C_s mg/kg
Polycyclic Aromatic Hydrocarbons							
Carcinogenic PAHs							
Benzo[a]anthracene	-	-	-	-	-	-	3.4E-01
Benzo[a]pyrene	-	-	-	-	-	-	4.0E-01
Benzo[b]fluoranthene	-	-	-	-	-	-	6.0E-01
Benzo[g,h,i]perylene	-	-	-	-	-	-	2.5E-01
Benzo[k]fluoranthene	-	-	-	-	-	-	2.4E-01
Chrysene	-	-	-	-	-	-	4.0E-01
Dibenzo[a,h]anthracene	-	-	-	-	-	-	6.0E-02
Fluoranthene	-	-	-	-	-	-	8.6E-01
Indeno[1,2,3-c,d]pyrene	-	-	-	-	-	-	2.7E-01
Phenanthrene	-	-	-	-	-	-	5.2E-01
Non-carcinogenic PAHs							
Acenaphthene	-	-	-	-	-	-	2.0E-02
Acenaphthylene	-	-	-	-	-	-	3.0E-02
Anthracene	-	-	-	-	-	-	8.0E-02
Fluorene	-	-	-	-	-	-	4.0E-02
Fluoranthene	-	-	-	-	-	-	8.6E-01
Naphthalene	-	-	-	-	-	-	2.0E-02
2-Methylnaphthalene	-	-	-	-	-	-	2.0E-02
Pyrene	-	-	-	-	-	-	7.0E-01
Metals and Metalloids							
Antimony	-	-	-	-	-	-	5.1E-01
Arsenic	-	-	-	-	-	-	6.1E+00
Barium	-	-	-	-	-	-	6.7E+01
Beryllium	-	-	-	-	-	-	2.4E-01
Cadmium	-	-	-	-	-	-	2.2E-01
Chromium (III)	-	-	-	-	-	-	2.6E+01
Chromium (VI)	-	-	-	-	-	-	1.6E-01
Cobalt	-	-	-	-	-	-	7.4E+00
Copper	-	-	-	-	-	-	2.5E+01
Lead	-	-	-	-	-	-	2.2E+01
Lithium	-	-	-	-	-	-	9.2E+00
Manganese	-	-	-	-	-	-	3.5E+02
Mercury	-	-	-	-	-	-	5.0E-02
Molybdenum	-	-	-	-	-	-	1.1E+00
Nickel	-	-	-	-	-	-	2.1E+01
Selenium	-	-	-	-	-	-	3.0E-01
Strontium	-	-	-	-	-	-	6.8E+01
Tin	-	-	-	-	-	-	2.1E+00
Uranium	-	-	-	-	-	-	7.5E-01
Vanadium	-	-	-	-	-	-	4.5E+01
Zinc	-	-	-	-	-	-	8.3E+01
Aluminum	-	-	-	-	-	-	1.4E+04
Boron	-	-	-	-	-	-	NA
Iron	-	-	-	-	-	-	1.9E+04
Titanium	-	-	-	-	-	-	7.4E+02
Indium	-	-	-	-	-	-	NA
Lanthanum	-	-	-	-	-	-	NA
Others							
PCBs	-	-	-	-	-	-	NA
Sulphate	-	-	-	-	-	-	NA
Hexachlorobenzene (gas phase)	-	-	-	-	-	-	NA
Dust Pallatives							
Epichlorohydrin	-	-	-	-	-	-	NA

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)
- Not applicable as baseline soil concentration based on measured data and not modelled
† Based on laboratory analysis conducted for study area
NA Data not available for this parameter

Table II-3A Kt Parameters for Baseline Scenario

Scenario: Baseline

Parameter	CAS	Organic Carbon Partition Coefficient (L/kg)	Water Solubility (mg/L)	Vapor Pressure (mm Hg)	t 1/2 days due to volatilzation	Kv yrs^{-1}	t 1/2 days due to degradation (abiotic and/or biotic)	Source t 1/2 due to degradation (abiotic and/or biotic)	Ks yrs^{-1}	Kt yrs^{-1}
Acenaphthene	000083-32-9	5.03E+03	3.90E+00	2.15E-03	1.4E-01	1.8E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.8E+03
Acenaphthylene	000208-96-8	5.03E+03	1.61E+01	6.68E-03	1.9E-01	1.3E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.3E+03
Aluminum	007429-90-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Anthracene	000120-12-7	1.64E+04	4.34E-02	6.53E-06	1.7E+00	1.5E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.5E+02
Antimony (metallic)	007440-36-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Arsenic, Inorganic	007440-38-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Barium	007440-39-3	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Benz[a]anthracene	000056-55-3	1.77E+05	9.40E-03	2.10E-07	1.3E+02	2.0E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	2.4E+00
Benzo[a]pyrene	000050-32-8	5.87E+05	1.62E-03	5.49E-09	2.7E+03	9.2E-02	570	OEHHA 2000	4.4E-01	5.4E-01
Benzo[b]fluoranthene	000205-99-2	5.99E+05	1.50E-03	5.00E-07	2.8E+01	8.9E+00	570	OEHHA 2000	4.4E-01	9.4E+00
Benzo[g,h,i]perylene	000191-24-2	1.95E+06	2.60E-04	1.00E-10	8.0E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Benzo[k]fluoranthene	000207-08-9	5.87E+05	8.00E-04	9.65E-10	7.7E+03	3.3E-02	570	OEHHA 2000	4.4E-01	4.8E-01
Beryllium and compounds	007440-41-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Boron And Borates Only	007440-42-8	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Cadmium (Diet)	007440-43-9	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III) (Soluble Particulates)	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III), Insoluble Salts	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(VI)	018540-29-9	-	1.69E+06	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chrysene	000218-01-9	1.81E+05	2.00E-03	6.23E-09	9.2E+02	2.8E-01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	6.9E-01
Cobalt	007440-48-4	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Copper	007440-50-8	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Dibenz[a,h]anthracene	000053-70-3	1.91E+06	2.49E-03	9.55E-10	7.9E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Epichlorohydrin	000106-89-8	9.91E+00	6.59E+04	1.64E+01	6.3E-04	4.0E+05	2.8E+01	Environment Canada 2000	9.0E+00	4.0E+05
Ethylbenzene	000100-41-4	4.46E+02	1.69E+02	9.60E+00	1.2E-04	2.0E+06	na	na	na	2.0E+06
Ethylene							1.0E+08	assumed	2.5E-06	2.5E-06
Fluoranthene	000206-44-0	5.55E+04	2.60E-01	9.22E-06	2.5E+01	1.0E+01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.1E+01
Fluorene	000086-73-7	9.16E+03	1.69E+00	6.00E-04	4.1E-01	6.2E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	6.2E+02
Formaldehyde	000050-00-0	1.00E+00	4.00E+05	3.89E+03	1.6E-06	1.6E+08	na	na	na	1.6E+08
Hexane, N-	000110-54-3	1.32E+02	9.50E+00	1.51E+02	1.3E-07	1.9E+09	na	na	na	1.9E+09
Indeno[1,2,3-cd]pyrene	000193-39-5	3.47E+06	2.20E-05	1.25E-10	9.6E+03	2.6E-02	570	OEHHA 2000	4.4E-01	4.7E-01
Iron	007439-89-6	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lead and Compounds	007439-92-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lithium	007439-93-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Manganese (Diet)	007439-96-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Mercury (elemental)	007439-97-6	-	6.00E-02	1.96E-03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Methylnaphthalene, 2-	000091-57-6	2.48E+03	2.46E+01	5.50E-02	1.8E-02	1.4E+04	32.5	CCME 2008 based on Loehr and Webster 1997	7.8E+00	1.4E+04
Molybdenum	007439-98-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Naphthalene	000091-20-3	1.54E+03	3.10E+01	8.50E-02	8.9E-03	2.9E+04	2.1	Enviornment Canada 1996	1.2E+02	2.9E+04
Nickel Oxide	001313-99-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Refinery Dust	000000-00-7	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Soluble Salts	007440-02-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Phenanthrene	000085-01-8	1.67E+04	1.15E+00	1.21E-04	2.5E+00	1.0E+02	5.7	CCME 2008 based on CEPA 1993	4.4E+01	1.5E+02
Pyrene	000129-00-0	5.43E+04	1.35E-01	4.50E-06	2.6E+01	9.8E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.0E+01
Selenium	007782-49-2	-	-	9.12E+03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Silver	007440-22-4	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Strontium, Stable	007440-24-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Styrene	000100-42-5	4.46E+02	3.10E+02	6.40E+00	3.4E-04	7.4E+05	na	na	na	7.4E+05
Tin	007440-31-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Titanium	007440-32-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Toluene	000108-88-3	2.34E+02	5.26E+02	2.84E+01	6.8E-05	3.7E+06	na	na	na	3.7E+06
Uranium, Insoluble Compounds	007440-61-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Vanadium	000000-06-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Xylenes	001330-20-7	3.83E+02	1.06E+02	7.99E+00	8.0E-05	3.2E+06	na	na	na	3.2E+06
Zinc and Compounds	007440-66-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Indium	007440-74-6	-				na	1.0E+08	OEHHA 2001	2.5E-06	2.5E-06
Lanthanum	007439-91-0	-				na	1.0E+08	OEHHA 2002	2.5E-06	2.5E-06
Hexachlorobenzene	000118-74-1	6.20E+03	6.20E-03	1.80E-05	6.00E+02	4.2E-01	1.0E+09	OEHHA 2003	2.5E-07	4.2E-01
PCBs	001336-36-3	7.81E+04	7.00E-01	8.63E-05		na	3.23E+03	OEHHA 2004	7.8E-02	7.8E-02
Sulphate	014808-79-8	-	1.00E+06	5.93E-05		na	1.0E+08	OEHHA 2005	2.5E-06	2.5E-06

Notes:

Values from RAIS (2014) unless otherwise noted

CCME. 2008. Canadian Council of Ministers of the Enviroment. Canada-Wide Standard (CWS) for Petroleum Hydrocarbons (PHC) in Soil. Scientifc Rationale Supporting Technical Document. CCME. 2008

OEHHA 2000. Office of Environmental Health Hazard Assessment. California EPA. Technical Support Document for Exposure Assessment and Stochastic Analysis. Air Toxics Hot Spots Program Risk Assessment Guidelines. Oakland. California. 2000

Environment Canada. 2000. Screening Assessment: oxirane, (chloromethyl)-,epichlorohydrin. Chemical Abstracts Service Registry Number 106-89-8. Environment Canada. Health Canada. November 2008

	Calculated based on first order decay rate: k=0.693/(t1/2 / 365)
assumed	Based on lack of data from above sources, 10^8 t 1/2 based on professional judgement
na	Not availble from sources idenfied above

Table II-4A Soil Particulate/Dust Concentations for Baseline Scenario **Scenario: Baseline**

Parameter	DL kg/m^3	Cs mg/kg	CF ug/mg	C_{dust} ug/m^3
Polycyclic Aromatic Hydrocarbons				
Carcinogenic PAHs				
Benzo[a]anthracene	7.6E-10	0.34	1000	2.6E-07
Benzo[a]pyrene	7.6E-10	0.4	1000	3.0E-07
Benzo[b]fluoranthene	7.6E-10	0.6	1000	4.6E-07
Benzo[g,h,i]perylene	7.6E-10	0.25	1000	1.9E-07
Benzo[k]fluoranthene	7.6E-10	0.24	1000	1.8E-07
Chrysene	7.6E-10	0.4	1000	3.0E-07
Dibenzo[a,h]anthracene	7.6E-10	0.06	1000	4.6E-08
Fluoranthene	7.6E-10	0.86	1000	6.5E-07
Indeno[1,2,3-c,d]pyrene	7.6E-10	0.27	1000	2.1E-07
Phenanthrene	7.6E-10	0.52	1000	4.0E-07
Non-carcinogenic PAHs				
Acenaphthene	7.6E-10	0.02	1000	1.5E-08
Acenaphthylene	7.6E-10	0.03	1000	2.3E-08
Anthracene	7.6E-10	0.08	1000	6.1E-08
Fluorene	7.6E-10	0.04	1000	3.0E-08
Fluoranthene	7.6E-10	0.86	1000	6.5E-07
Naphthalene	7.6E-10	0.02	1000	1.5E-08
2-Methylnaphthalene	7.6E-10	0.02	1000	1.5E-08
Pyrene	7.6E-10	0.7	1000	5.3E-07
Metals and Metalloids				
Antimony	7.6E-10	0.51	1000	3.9E-07
Arsenic	7.6E-10	6.07	1000	4.6E-06
Barium	7.6E-10	66.52	1000	5.1E-05
Beryllium	7.6E-10	0.24	1000	1.8E-07
Cadmium	7.6E-10	0.22	1000	1.7E-07
Chromium (III)	7.6E-10	25.82	1000	2.0E-05
Chromium (VI)	7.6E-10	0.16	1000	1.2E-07
Cobalt	7.6E-10	7.41	1000	5.6E-06
Copper	7.6E-10	24.63	1000	1.9E-05
Lead	7.6E-10	21.94	1000	1.7E-05
Lithium	7.6E-10	9.2	1000	7.0E-06
Manganese	7.6E-10	354.67	1000	2.7E-04
Mercury	7.6E-10	0.05	1000	3.8E-08
Molybdenum	7.6E-10	1.08	1000	8.2E-07
Nickel	7.6E-10	20.93	1000	1.6E-05
Selenium	7.6E-10	0.3	1000	2.3E-07
Strontium	7.6E-10	67.76	1000	5.1E-05
Tin	7.6E-10	2.07	1000	1.6E-06
Uranium	7.6E-10	0.75	1000	5.7E-07
Vanadium	7.6E-10	44.7	1000	3.4E-05
Zinc	7.6E-10	83.36	1000	6.3E-05
Aluminum	7.6E-10	13549.79	1000	1.0E-02
Boron	7.6E-10	NA	1000	NA
Iron	7.6E-10	18715.42	1000	1.4E-02
Titanium	7.6E-10	744.44	1000	5.7E-04
Indium	7.6E-10	NA	1000	NA
Lanthanum	7.6E-10	NA	1000	NA
Others				
PCBs	7.6E-10	NA	1000	NA
Sulphate	7.6E-10	NA	1000	NA
Hexachlorobenzene (gas phase)	7.6E-10	NA	1000	NA
Dust Pallatives				
Epichlorohydrin	7.6E-10	NA	1000	NA

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2
NA - not available for this parameter

Table II-5A: Above Ground Plant Concentations **Scenario: Baseline**

Parameter	Dd mg/m^2/yr	Dw mg/m^2/yr	Fv percentage	Rp unitless	kp yr^{-1}	Tp yr	Yp kg DW/m^2	Pd mg/kg DW	Log K_{ow} unitless	BCF kg soil/kg plant DW	Cs mg/kg	BCF kg soil/kg plant DW	Pr mg/kg DW	Pd mg/kg	Pr mg/kg	‡ $C_{plant\ ww}$ mg/kg
Polycyclic Aromatic Hydrocarbons																
Carcinogenic PAHs																
Benzo[a]anthracene	-	-	-	-	-	-	-	-	5.76	1.8E-02	3.4E-01	1.8E-02	6.2E-03	-	6.2E-03	9.3E-04
Benzo[a]pyrene	-	-	-	-	-	-	-	-	6.13	1.1E-02	4.0E-01	1.1E-02	4.4E-03	-	4.4E-03	6.7E-04
Benzo[b]fluoranthene	-	-	-	-	-	-	-	-	5.78	1.8E-02	6.0E-01	1.8E-02	1.1E-02	-	1.1E-02	1.6E-03
Benzo[g,h,i]perylene	-	-	-	-	-	-	-	-	6.63	5.7E-03	2.5E-01	5.7E-03	1.4E-03	-	1.4E-03	2.1E-04
Benzo[k]fluoranthene	-	-	-	-	-	-	-	-	6.11	1.1E-02	2.4E-01	1.1E-02	2.7E-03	-	2.7E-03	4.1E-04
Chrysene	-	-	-	-	-	-	-	-	5.81	1.7E-02	4.0E-01	1.7E-02	6.8E-03	-	6.8E-03	1.0E-03
Dibenzo[a,h]anthracene	-	-	-	-	-	-	-	-	6.75	4.9E-03	6.0E-02	4.9E-03	2.9E-04	-	2.9E-04	4.4E-05
Fluoranthene	-	-	-	-	-	-	-	-	5.16	4.0E-02	8.6E-01	4.0E-02	3.5E-02	-	3.5E-02	5.2E-03
Indeno[1,2,3-c,d]pyrene	-	-	-	-	-	-	-	-	6.71	5.1E-03	2.7E-01	5.1E-03	1.4E-03	-	1.4E-03	2.1E-04
Phenanthrene	-	-	-	-	-	-	-	-	4.46	1.0E-01	5.2E-01	1.0E-01	5.3E-02	-	5.3E-02	8.0E-03
Non-carcinogenic PAHs																
Acenaphthene	-	-	-	-	-	-	-	-	3.92	2.1E-01	2.0E-02	2.1E-01	4.2E-03	-	4.2E-03	6.3E-04
Acenaphthylene	-	-	-	-	-	-	-	-	3.94	2.0E-01	3.0E-02	2.0E-01	6.1E-03	-	6.1E-03	9.2E-04
Anthracene	-	-	-	-	-	-	-	-	4.45	1.0E-01	8.0E-02	1.0E-01	8.3E-03	-	8.3E-03	1.2E-03
Fluorene	-	-	-	-	-	-	-	-	4.18	1.5E-01	4.0E-02	1.5E-01	5.9E-03	-	5.9E-03	8.9E-04
Fluoranthene	-	-	-	-	-	-	-	-	5.16	4.0E-02	8.6E-01	4.0E-02	3.5E-02	-	3.5E-02	5.2E-03
Naphthalene	-	-	-	-	-	-	-	-	3.3	4.8E-01	2.0E-02	4.8E-01	9.6E-03	-	9.6E-03	1.4E-03
2-Methylnaphthalene	-	-	-	-	-	-	-	-	3.86	2.3E-01	2.0E-02	2.3E-01	4.5E-03	-	4.5E-03	6.8E-04
Pyrene	-	-	-	-	-	-	-	-	4.88	5.9E-02	7.0E-01	5.9E-02	4.1E-02	-	4.1E-02	6.1E-03
Metals and Metalliods																
Antimony	-	-	-	-	-	-	-	-	-	2.0E-01	5.1E-01	2.0E-01	1.0E-01	-	1.0E-01	1.5E-02
Arsenic	-	-	-	-	-	-	-	-	-	4.0E-02	6.1E+00	4.0E-02	2.4E-01	-	2.4E-01	3.6E-02
Barium	-	-	-	-	-	-	-	-	-	1.5E-01	6.7E+01	1.5E-01	1.0E+01	-	1.0E+01	1.5E+00
Beryllium	-	-	-	-	-	-	-	-	-	1.0E-02	2.4E-01	1.0E-02	2.4E-03	-	2.4E-03	3.6E-04
Cadmium	-	-	-	-	-	-	-	-	-	5.0E-01	2.2E-01	5.0E-01	1.1E-01	-	1.1E-01	1.7E-02
Chromium (III)	-	-	-	-	-	-	-	-	-	7.5E-03	2.6E+01	7.5E-03	1.9E-01	-	1.9E-01	2.9E-02
Chromium (VI)	-	-	-	-	-	-	-	-	-	7.5E-03	1.6E-01	7.5E-03	1.2E-03	-	1.2E-03	1.8E-04
Cobalt	-	-	-	-	-	-	-	-	-	2.0E-02	7.4E+00	2.0E-02	1.5E-01	-	1.5E-01	2.2E-02
Copper	-	-	-	-	-	-	-	-	-	4.0E-01	2.5E+01	4.0E-01	9.9E+00	-	9.9E+00	1.5E+00
Lead	-	-	-	-	-	-	-	-	-	4.5E-02	2.2E+01	4.5E-02	9.9E-01	-	9.9E-01	1.5E-01
Lithium	-	-	-	-	-	-	-	-	-	2.5E-02	9.2E+00	2.5E-02	2.3E-01	-	2.3E-01	3.5E-02
Manganese	-	-	-	-	-	-	-	-	-	2.5E-01	3.5E+02	2.5E-01	8.9E+01	-	8.9E+01	1.3E+01
Mercury	-	-	-	-	-	-	-	-	-	9.0E-01	5.0E-02	9.0E-01	4.5E-02	-	4.5E-02	6.8E-03
Molybdenum	-	-	-	-	-	-	-	-	-	2.5E-01	1.1E+00	2.5E-01	2.7E-01	-	2.7E-01	4.1E-02
Nickel	-	-	-	-	-	-	-	-	-	6.0E-02	2.1E+01	6.0E-02	1.3E+00	-	1.3E+00	1.9E-01
Selenium	-	-	-	-	-	-	-	-	-	2.5E-02	3.0E-01	2.5E-02	7.5E-03	-	7.5E-03	1.1E-03
Strontium	-	-	-	-	-	-	-	-	-	2.5E+00	6.8E+01	2.5E+00	1.7E+02	-	1.7E+02	2.5E+01
Tin	-	-	-	-	-	-	-	-	-	3.0E-02	2.1E+00	3.0E-02	6.2E-02	-	6.2E-02	9.3E-03
Uranium	-	-	-	-	-	-	-	-	-	8.5E-03	7.5E-01	8.5E-03	6.4E-03	-	6.4E-03	9.6E-04
Vanadium	-	-	-	-	-	-	-	-	-	5.5E-03	4.5E+01	5.5E-03	2.5E-01	-	2.5E-01	3.7E-02
Zinc	-	-	-	-	-	-	-	-	-	9.9E-01	8.3E+01	9.9E-01	8.3E+01	-	8.3E+01	1.2E+01
Aluminum	-	-	-	-	-	-	-	-	-	4.0E-03	1.4E+04	4.0E-03	5.4E+01	-	5.4E+01	8.1E+00
Boron	-	-	-	-	-	-	-	-	-	4.0E+00	NA	4.0E+00	NA	-	NA	NA
Iron	-	-	-	-	-	-	-	-	-	4.0E-03	1.9E+04	4.0E-03	7.5E+01	-	7.5E+01	1.1E+01
Titanium	-	-	-	-	-	-	-	-	-	5.5E-03	7.4E+02	5.5E-03	4.1E+00	-	4.1E+00	6.1E-01
Indium	-	-	-	-	-	-	-	-		*4.0E-01*	NA	4.0E-01	NA	-	NA	NA
Lanthanum	-	-	-	-	-	-	-	-		*4.0E-01*	NA	4.0E-01	NA	-	NA	NA
Others																
PCBs	-	-	-	-	-	-	-	-	7.1E+00	2.9E-03	NA	2.9E-03	NA	-	NA	NA
Sulphate	-	-	-	-	-	-	-	-		*4.0E-01*	NA	4.0E-01	NA	-	NA	NA
Hexachlorobenzene (gas phase)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	NA
Dust Pallatives																
Epichlorohydrin	-	-	-	-	-	-	-	-	0.45	2.1E+01	NA	2.1E+01	NA	-	NA	NA

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)
[2] Log Kow based on US EPA (2004), RAIS (2014), HSDB (2014)
[3] Soil concentration from Equation 1.2
[4] For inorganics soil to dry plant uptake values from ORNL (2014)
- Not available for this parameter
‡ Background deposition data not available therefore, above ground plant concentation due to chemical root uptake. This is conservative as it assumes lower background vegetation concentrations.
Italics BCF not available BCF assumed to be equal to average of remaining metals

Table II-6A Below Ground Plant Concentrations Baseline Scenario **Scenario: Baseline**

Parameter	Cs mg/kg	BCF kg soil/kg plant DW	WPF unitless	WC percentage	Pr_{root} mg/kg ww
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	3.4E-01	1.8E-02	1	0.85	9.3E-04
Benzo[a]pyrene	4.0E-01	1.1E-02	1	0.85	6.7E-04
Benzo[b]fluoranthene	6.0E-01	1.8E-02	1	0.85	1.6E-03
Benzo[g,h,i]perylene	2.5E-01	5.7E-03	1	0.85	2.1E-04
Benzo[k]fluoranthene	2.4E-01	1.1E-02	1	0.85	4.1E-04
Chrysene	4.0E-01	1.7E-02	1	0.85	1.0E-03
Dibenzo[a,h]anthracene	6.0E-02	4.9E-03	1	0.85	4.4E-05
Fluoranthene	8.6E-01	4.0E-02	1	0.85	5.2E-03
Indeno[1,2,3-c,d]pyrene	2.7E-01	5.1E-03	1	0.85	2.1E-04
Phenanthrene	5.2E-01	1.0E-01	1	0.85	8.0E-03
Non-carcinogenic PAHs					
Acenaphthene	2.0E-02	2.1E-01	1	0.85	6.3E-04
Acenaphthylene	3.0E-02	2.0E-01	1	0.85	9.2E-04
Anthracene	8.0E-02	1.0E-01	1	0.85	1.2E-03
Fluorene	4.0E-02	1.5E-01	1	0.85	8.9E-04
Fluoranthene	8.6E-01	4.0E-02	1	0.85	5.2E-03
Naphthalene	2.0E-02	4.8E-01	1	0.85	1.4E-03
2-Methylnaphthalene	2.0E-02	2.3E-01	1	0.85	6.8E-04
Pyrene	7.0E-01	5.9E-02	1	0.85	6.1E-03
Metals and Metalliods					
Antimony	5.1E-01	2.0E-01	1	0.85	1.5E-02
Arsenic	6.1E+00	4.0E-02	1	0.85	3.6E-02
Barium	6.7E+01	1.5E-01	1	0.85	1.5E+00
Beryllium	2.4E-01	1.0E-02	1	0.85	3.6E-04
Cadmium	2.2E-01	5.0E-01	1	0.85	1.7E-02
Chromium (III)	2.6E+01	7.5E-03	1	0.85	2.9E-02
Chromium (VI)	1.6E-01	7.5E-03	1	0.85	1.8E-04
Cobalt	7.4E+00	2.0E-02	1	0.85	2.2E-02
Copper	2.5E+01	4.0E-01	1	0.85	1.5E+00
Lead	2.2E+01	4.5E-02	1	0.85	1.5E-01
Lithium	9.2E+00	2.5E-02	1	0.85	3.5E-02
Manganese	3.5E+02	2.5E-01	1	0.85	1.3E+01
Mercury	5.0E-02	9.0E-01	1	0.85	6.8E-03
Molybdenum	1.1E+00	2.5E-01	1	0.85	4.1E-02
Nickel	2.1E+01	6.0E-02	1	0.85	1.9E-01
Selenium	3.0E-01	2.5E-02	1	0.85	1.1E-03
Strontium	6.8E+01	2.5E+00	1	0.85	2.5E+01
Tin	2.1E+00	3.0E-02	1	0.85	9.3E-03
Uranium	7.5E-01	8.5E-03	1	0.85	9.6E-04
Vanadium	4.5E+01	5.5E-03	1	0.85	3.7E-02
Zinc	8.3E+01	9.9E-01	1	0.85	1.2E+01
Aluminum	1.4E+04	4.0E-03	1	0.85	8.1E+00
Boron	NA	4.0E+00	1	0.85	NA
Iron	1.9E+04	4.0E-03	1	0.85	1.1E+01
Titanium	7.4E+02	5.5E-03	1	0.85	6.1E-01
Crystalline Silica	NA	1.6E+00	1	0.85	NA
Indium	NA	4.0E-01	1	0.85	NA
Lanthanum	NA	4.0E-01	1	0.85	NA
Others					
PCBs	NA	2.9E-03	1	0.85	NA
Sulphate	NA	4.0E-01	1	0.85	NA
Hexachlorobenzene (gas phase)	-	-	-	-	-
Dust Pallatives					
Epichlorohydrin	NA	2.1E+01	1	0.85	NA

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2
[2] BCF from Equation 3.2.1 for inorganics, for organics soil to dry plant uptake value from ORNL (2014)
NA Background data not available for this parameter
- Vapour to root vegetation uptake assumed to be zero (US EPA OSW 2005)

Table II-1B Summary of Project Multimedia Exposure Point Concentrations (EPCs) for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	C_s mg/kg	C_{dust} ug/m^3	$C_{plant\ (w/w)}$ mg/kg	$Pr_{root\ (w/w)}$ mg/kg	C_{air} ug/m^3
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	3.7E-04	2.8E-10	1.3E-06	1.0E-06	3.8E-04
Benzo[a]pyrene	6.7E-04	5.1E-10	1.2E-06	1.1E-06	1.2E-05
Benzo[b]fluoranthene	1.4E-04	1.1E-10	7.1E-07	3.7E-07	2.6E-04
Benzo[g,h,i]perylene	2.2E-04	1.6E-10	2.2E-07	1.9E-07	2.0E-05
Benzo[k]fluoranthene	5.7E-04	4.3E-10	1.1E-06	9.7E-07	5.6E-05
Chrysene	7.8E-04	5.9E-10	2.1E-06	2.0E-06	1.1E-04
Dibenzo[a,h]anthracene	1.7E-04	1.3E-10	1.4E-07	1.2E-07	1.6E-05
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04
Indeno[1,2,3-c,d]pyrene	5.6E-04	4.3E-10	5.1E-07	4.3E-07	5.4E-05
Phenanthrene	4.2E-06	3.2E-12	3.3E-07	6.5E-08	5.2E-04
Non-carcinogenic PAHs					
Acenaphthene	3.1E-07	2.4E-13	1.7E-07	9.9E-09	1.2E-04
Acenaphthylene	1.1E-07	8.5E-14	4.6E-08	3.4E-09	9.8E-05
Anthracene	2.0E-06	1.5E-12	1.3E-07	3.2E-08	4.2E-04
Fluorene	4.3E-07	3.2E-13	8.4E-08	9.5E-09	7.7E-05
Fluoranthene	2.4E-04	1.8E-10	2.2E-06	1.4E-06	5.2E-04
Naphthalene	4.0E-09	3.0E-15	2.7E-07	2.9E-10	1.8E-04
2-Methylnaphthalene	7.1E-09	5.4E-15	2.6E-08	2.4E-10	2.1E-05
Pyrene	3.1E-04	2.4E-10	3.7E-06	2.7E-06	6.6E-04
Metals and Metalliods					
Antimony	1.6E-03	1.2E-09	4.7E-05	4.7E-05	4.1E-05
Arsenic	2.0E-02	1.5E-08	1.2E-04	1.2E-04	4.1E-04
Barium	5.4E+00	4.1E-06	1.2E-01	1.2E-01	1.1E-01
Beryllium	3.3E-03	2.5E-09	5.1E-06	4.9E-06	6.9E-05
Cadmium	1.7E-03	1.3E-09	1.3E-04	1.3E-04	1.2E-05
Chromium (III)	1.5E-03	1.2E-09	1.8E-06	1.7E-06	1.5E-04
Chromium (VI)	4.6E-02	3.5E-08	5.3E-05	5.2E-05	4.7E-07
Cobalt	1.9E-02	1.5E-08	5.8E-05	5.8E-05	6.3E-05
Copper	2.0E-01	1.5E-07	1.2E-02	1.2E-02	6.5E-04
Lead	3.4E-02	2.6E-08	2.3E-04	2.3E-04	1.2E-04
Manganese	3.0E-01	2.2E-07	1.1E-02	1.1E-02	9.6E-04
Mercury	1.2E-03	9.1E-10	1.6E-04	1.6E-04	5.8E-06
Molybdenum	7.7E-03	5.9E-09	2.9E-04	2.9E-04	2.7E-05
Nickel	6.2E-02	4.7E-08	5.6E-04	5.5E-04	2.0E-04
Selenium	9.9E-03	7.5E-09	3.7E-05	3.7E-05	3.2E-05
Strontium	3.4E+00	2.6E-06	1.3E+00	1.3E+00	1.1E-02
Tin	3.5E-03	2.6E-09	1.6E-05	1.6E-05	1.7E-05
Uranium	5.4E-03	4.1E-09	7.0E-06	6.9E-06	1.8E-05
Vanadium	1.6E-01	1.2E-07	1.4E-04	1.3E-04	5.4E-04
Zinc	2.1E-01	1.6E-07	3.1E-02	3.1E-02	7.1E-04
Aluminum	3.5E+01	2.6E-05	2.2E-02	2.1E-02	1.1E-01
Boron	2.7E-01	2.0E-07	1.6E-01	1.6E-01	8.8E-04
Iron	3.9E+01	2.9E-05	2.4E-02	2.3E-02	1.3E-01
Titanium	2.4E+00	1.8E-06	2.0E-03	2.0E-03	7.9E-03
Indium	1.4E-09	1.1E-15	6.8E-08	8.7E-11	5.9E-06
Lanthanum	3.7E-09	2.8E-15	1.7E-07	2.2E-10	1.5E-05
Others					
PCBs	3.0E-12	2.3E-18	2.2E-10	1.3E-15	8.4E-09
Sulfate	4.6E-07	3.5E-13	2.2E-05	2.8E-08	1.9E-03
Hexachlorobenzene (gas phase)	0.0E+00	0.0E+00	1.7E-13	0.0E+00	6.8E-10
Dust Pallatives					
Epichlorohydrin	3.8E-09	2.9E-15	4.7E-08	1.2E-08	1.3E-06

Notes:

- Not applicable for this parameter

[1] Parameter speciated into individual PAH and metal constituents above

[2] Operable exposure for this parameter limited to inhalation pathway

Table II-2B Project Soil Concentrations for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	D_{tot} mg/m^2/yr	Zs m	BD kg/m^3	Ds mg/kg/yr	kt yrs^{-1}	tD yrs	C_s mg/kg
Polycyclic Aromatic Hydrocarbons							
Carcinogenic PAHs							
Benzo[a]anthracene	2.7E-02	0.02	1500	8.9E-04	2.4E+00	10	3.7E-04
Benzo[a]pyrene	1.1E-02	0.02	1500	3.6E-04	5.4E-01	10	6.7E-04
Benzo[b]fluoranthene	3.9E-02	0.02	1500	1.3E-03	9.4E+00	10	1.4E-04
Benzo[g,h,i]perylene	2.9E-03	0.02	1500	9.8E-05	4.5E-01	10	2.2E-04
Benzo[k]fluoranthene	8.2E-03	0.02	1500	2.7E-04	4.8E-01	10	5.7E-04
Chrysene	1.6E-02	0.02	1500	5.4E-04	6.9E-01	10	7.8E-04
Dibenzo[a,h]anthracene	2.3E-03	0.02	1500	7.7E-05	4.5E-01	10	1.7E-04
Fluoranthene	7.5E-02	0.02	1500	2.5E-03	1.1E+01	10	2.4E-04
Indeno[1,2,3-c,d]pyrene	8.0E-03	0.02	1500	2.7E-04	4.7E-01	10	5.6E-04
Phenanthrene	1.8E-02	0.02	1500	6.1E-04	1.5E+02	10	4.2E-06
Non-carcinogenic PAHs							
Acenaphthene	1.6E-02	0.02	1500	5.5E-04	1.8E+03	10	3.1E-07
Acenaphthylene	4.4E-03	0.02	1500	1.5E-04	1.3E+03	10	1.1E-07
Anthracene	9.1E-03	0.02	1500	3.0E-04	1.5E+02	10	2.0E-06
Fluorene	8.0E-03	0.02	1500	2.7E-04	6.2E+02	10	4.3E-07
Fluoranthene	7.5E-02	0.02	1500	2.5E-03	1.1E+01	10	2.4E-04
Naphthalene	3.4E-03	0.02	1500	1.1E-04	2.9E+04	10	4.0E-09
2-Methylnaphthalene	3.1E-03	0.02	1500	1.0E-04	1.4E+04	10	7.1E-09
Pyrene	9.6E-02	0.02	1500	3.2E-03	1.0E+01	10	3.1E-04
Metals and Metalloids							
Antimony	4.7E-03	0.02	1500	1.6E-04	2.5E-06	10	1.6E-03
Arsenic	6.1E-02	0.02	1500	2.0E-03	2.5E-06	10	2.0E-02
Barium	1.6E+01	0.02	1500	5.4E-01	2.5E-06	10	5.4E+00
Beryllium	9.8E-03	0.02	1500	3.3E-04	2.5E-06	10	3.3E-03
Cadmium	5.1E-03	0.02	1500	1.7E-04	2.5E-06	10	1.7E-03
Chromium (III)	4.6E-03	0.02	1500	1.5E-04	2.5E-06	10	1.5E-03
Chromium (VI)	1.4E-01	0.02	1500	4.6E-03	2.5E-06	10	4.6E-02
Cobalt	5.8E-02	0.02	1500	1.9E-03	2.5E-06	10	1.9E-02
Copper	5.9E-01	0.02	1500	2.0E-02	2.5E-06	10	2.0E-01
Lead	1.0E-01	0.02	1500	3.4E-03	2.5E-06	10	3.4E-02
Lithium	2.3E-01	0.02	1500	7.7E-03	2.5E-06	10	7.7E-02
Manganese	8.9E-01	0.02	1500	3.0E-02	2.5E-06	10	3.0E-01
Mercury	3.6E-03	0.02	1500	1.2E-04	2.5E-06	10	1.2E-03
Molybdenum	2.3E-02	0.02	1500	7.7E-04	2.5E-06	10	7.7E-03
Nickel	1.8E-01	0.02	1500	6.2E-03	2.5E-06	10	6.2E-02
Selenium	3.0E-02	0.02	1500	9.9E-04	2.5E-06	10	9.9E-03
Strontium	1.0E+01	0.02	1500	3.4E-01	2.5E-06	10	3.4E+00
Tin	1.0E-02	0.02	1500	3.5E-04	2.5E-06	10	3.5E-03
Uranium	1.6E-02	0.02	1500	5.4E-04	2.5E-06	10	5.4E-03
Vanadium	4.8E-01	0.02	1500	1.6E-02	2.5E-06	10	1.6E-01
Zinc	6.2E-01	0.02	1500	2.1E-02	2.5E-06	10	2.1E-01
Aluminum	1.0E+02	0.02	1500	3.5E+00	2.5E-06	10	3.5E+01
Bismuth	4.6E-03	0.02	1500	1.5E-04	2.5E-06	10	1.5E-03
Boron	8.0E-01	0.02	1500	2.7E-02	2.5E-06	10	2.7E-01
Iron	1.2E+02	0.02	1500	3.9E+00	2.5E-06	10	3.9E+01
Titanium	7.2E+00	0.02	1500	2.4E-01	2.5E-06	10	2.4E+00
Indium	4.3E-09	0.02	1500	1.4E-10	2.5E-06	10	1.4E-09
Lanthanum	1.1E-08	0.02	1500	3.7E-10	2.5E-06	10	3.7E-09
Others							
PCBs	1.3E-11	0.02	1500	4.4E-13	7.8E-02	10	3.0E-12
Sulphate	1.4E-06	0.02	1500	4.6E-08	2.5E-06	10	4.6E-07
Hexachlorobenzene (gas phase)	0.0E+00	0.02	1500	0.0E+00	4.2E-01	10	0.0E+00
Dust Pallatives							
Epichlorohydrin	*4.6E-02*	0.02	1500	1.5E-03	4.0E+05	10	3.8E-09

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)
Italics conservatively assuming 100% of dustfall, note this is clearly an overestimate

Table II-3B Kt Parameters for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	CAS	Organic Carbon Partition Coefficient (L/kg)	Water Solubility (mg/L)	Vapor Pressure (mm Hg)	t 1/2 days due to volatilzation	Kv yrs^{-1}	t 1/2 days due to degradation (abiotic and/or biotic)	Source t 1/2 due to degradation (abiotic and/or biotic)	Ks yrs^{-1}	Kt yrs^{-1}
Acenaphthene	000083-32-9	5.03E+03	3.90E+00	2.15E-03	1.4E-01	1.8E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.8E+03
Acenaphthylene	000208-96-8	5.03E+03	1.61E+01	6.68E-03	1.9E-01	1.3E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.3E+03
Aluminum	007429-90-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Anthracene	000120-12-7	1.64E+04	4.34E-02	6.53E-06	1.7E+00	1.5E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.5E+02
Antimony (metallic)	007440-36-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Arsenic, Inorganic	007440-38-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Barium	007440-39-3	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Benz[a]anthracene	000056-55-3	1.77E+05	9.40E-03	2.10E-07	1.3E+02	2.0E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	2.4E+00
Benzene	000071-43-2	1.46E+02	1.79E+03	9.48E+01	4.4E-05	5.8E+06	68	CCME 2008 based on Baker and Mayfield 1980	3.7E+00	5.8E+06
Benzo[a]pyrene	000050-32-8	5.87E+05	1.62E-03	5.49E-09	2.7E+03	9.2E-02	570	OEHHA 2000	4.4E-01	5.4E-01
Benzo[b]fluoranthene	000205-99-2	5.99E+05	1.50E-03	5.00E-07	2.8E+01	8.9E+00	570	OEHHA 2000	4.4E-01	9.4E+00
Benzo[g,h,i]perylene	000191-24-2	1.95E+06	2.60E-04	1.00E-10	8.0E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Benzo[k]fluoranthene	000207-08-9	5.87E+05	8.00E-04	9.65E-10	7.7E+03	3.3E-02	570	OEHHA 2000	4.4E-01	4.8E-01
Beryllium and compounds	007440-41-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Boron And Borates Only	007440-42-8	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Cadmium (Diet)	007440-43-9	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III) (Soluble Particulates)	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III), Insoluble Salts	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(VI)	018540-29-9	-	1.69E+06	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chrysene	000218-01-9	1.81E+05	2.00E-03	6.23E-09	9.2E+02	2.8E-01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	6.9E-01
Cobalt	007440-48-4	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Copper	007440-50-8	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Dibenz[a,h]anthracene	000053-70-3	1.91E+06	2.49E-03	9.55E-10	7.9E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Epichlorohydrin	000106-89-8	9.91E+00	6.59E+04	1.64E+01	6.3E-04	4.0E+05	2.8E+01	Environment Canada 2000	9.0E+00	4.0E+05
Fluoranthene	000206-44-0	5.55E+04	2.60E-01	9.22E-06	2.5E+01	1.0E+01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.1E+01
Fluorene	000086-73-7	9.16E+03	1.69E+00	6.00E-04	4.1E-01	6.2E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	6.2E+02
Hexane, N-	000110-54-3	1.32E+02	9.50E+00	1.51E+02	1.3E-07	1.9E+09	na	na	na	1.9E+09
Indeno[1,2,3-cd]pyrene	000193-39-5	3.47E+06	2.20E-05	1.25E-10	9.6E+03	2.6E-02	570	OEHHA 2000	4.4E-01	4.7E-01
Iron	007439-89-6	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lead and Compounds	007439-92-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Linear Alkyl Sulfonate							1.0E+08	assumed	2.5E-06	2.5E-06
Lithium	007439-93-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Manganese (Diet)	007439-96-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Mercury (elemental)	007439-97-6	-	6.00E-02	1.96E-03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Methylnaphthalene, 2-	000091-57-6	2.48E+03	2.46E+01	5.50E-02	1.8E-02	1.4E+04	32.5	CCME 2008 based on Loehr and Webster 1997	7.8E+00	1.4E+04
Molybdenum	007439-98-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Naphthalene	000091-20-3	1.54E+03	3.10E+01	8.50E-02	8.9E-03	2.9E+04	2.1	Enviornment Canada 1996	1.2E+02	2.9E+04
Nickel Oxide	001313-99-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Refinery Dust	000000-00-7	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Soluble Salts	007440-02-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Phenanthrene	000085-01-8	1.67E+04	1.15E+00	1.21E-04	2.5E+00	1.0E+02	5.7	CCME 2008 based on CEPA 1993	4.4E+01	1.5E+02
Pyrene	000129-00-0	5.43E+04	1.35E-01	4.50E-06	2.6E+01	9.8E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.0E+01
Selenium	007782-49-2	-	-	9.12E+03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Strontium, Stable	007440-24-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Styrene	000100-42-5	4.46E+02	3.10E+02	6.40E+00	3.4E-04	7.4E+05	na	na	na	7.4E+05
Tin	007440-31-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Titanium	007440-32-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Toluene	000108-88-3	2.34E+02	5.26E+02	2.84E+01	6.8E-05	3.7E+06	na	na	na	3.7E+06
Uranium, Insoluble Compounds	007440-61-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Vanadium	000000-06-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Xylenes	001330-20-7	3.83E+02	1.06E+02	7.99E+00	8.0E-05	3.2E+06	na	na	na	3.2E+06
Zinc and Compounds	007440-66-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Indium	007440-74-6	-				na	1.0E+08	OEHHA 2001	2.5E-06	2.5E-06
Lanthanum	007439-91-0	-				na	1.0E+08	OEHHA 2002	2.5E-06	2.5E-06
Hexachlorobenzene	000118-74-1	6.20E+03	6.20E-03	1.80E-05	6.00E+02	4.2E-01	1.0E+09	OEHHA 2003	2.5E-07	4.2E-01
PCBs	001336-36-3	7.81E+04	7.00E-01	8.63E-05		na	3.23E+03	OEHHA 2004	7.8E-02	7.8E-02
Sulphate	014808-79-8	-	1.00E+06	5.93E-05		na	1.0E+08	OEHHA 2005	2.5E-06	2.5E-06

Notes:

Values from RAIS (2014) unless otherwise noted

CCME. 2008. Canadian Council of Ministers of the Enviroment. Canada-Wide Standard (CWS) for Petroleum Hydrocarbons (PHC) in Soil. Scientifc Rationale Supporting Technical Document. CCME. 2008

OEHHA 2000. Office of Environmental Health Hazard Assessment. California EPA. Technical Support Document for Exposure Assessment and Stochastic Analysis. Air Toxics Hot Spots Program Risk Assessment Guidelines. Oakland. California. 2000

Environment Canada. 2000. Screening Assessment: oxirane, (chloromethyl)-,epichlorohydrin. Chemical Abstracts Service Registry Number 106-89-8. Environment Canada. Health Canada. November 2008

	Calculated based on first order decay rate: k=0.693/(t1/2 / 365)
assumed	Based on lack of data from above sources, 10^8 t 1/2 based on professional judgement
na	Not availble from sources idenfied above

Table II-4B Project Soil Particulate/Dust Concentations for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	DL kg/m^3	Cs mg/kg	CF ug/mg	C_{dust} ug/m^3
Polycyclic Aromatic Hydrocarbons				
Carcinogenic PAHs				
Benzo[a]anthracene	7.6E-10	3.7E-04	1000	2.8E-10
Benzo[a]pyrene	7.6E-10	6.7E-04	1000	5.1E-10
Benzo[b]fluoranthene	7.6E-10	1.4E-04	1000	1.1E-10
Benzo[g,h,i]perylene	7.6E-10	2.2E-04	1000	1.6E-10
Benzo[k]fluoranthene	7.6E-10	5.7E-04	1000	4.3E-10
Chrysene	7.6E-10	7.8E-04	1000	5.9E-10
Dibenzo[a,h]anthracene	7.6E-10	1.7E-04	1000	1.3E-10
Fluoranthene	7.6E-10	2.4E-04	1000	1.8E-10
Indeno[1,2,3-c,d]pyrene	7.6E-10	5.6E-04	1000	4.3E-10
Phenanthrene	7.6E-10	4.2E-06	1000	3.2E-12
Non-carcinogenic PAHs				
Acenaphthene	7.6E-10	3.1E-07	1000	2.4E-13
Acenaphthylene	7.6E-10	1.1E-07	1000	8.5E-14
Anthracene	7.6E-10	2.0E-06	1000	1.5E-12
Fluorene	7.6E-10	4.3E-07	1000	3.2E-13
Fluoranthene	7.6E-10	2.4E-04	1000	1.8E-10
Naphthalene	7.6E-10	4.0E-09	1000	3.0E-15
2-Methylnaphthalene	7.6E-10	7.1E-09	1000	5.4E-15
Pyrene	7.6E-10	3.1E-04	1000	2.4E-10
Metals and Metalloids				
Antimony	7.6E-10	1.6E-03	1000	1.2E-09
Arsenic	7.6E-10	2.0E-02	1000	1.5E-08
Barium	7.6E-10	5.4E+00	1000	4.1E-06
Beryllium	7.6E-10	3.3E-03	1000	2.5E-09
Cadmium	7.6E-10	1.7E-03	1000	1.3E-09
Chromium (III)	7.6E-10	1.5E-03	1000	1.2E-09
Chromium (VI)	7.6E-10	4.6E-02	1000	3.5E-08
Cobalt	7.6E-10	1.9E-02	1000	1.5E-08
Copper	7.6E-10	2.0E-01	1000	1.5E-07
Lead	7.6E-10	3.4E-02	1000	2.6E-08
Lithium	7.6E-10	7.7E-02	1000	5.8E-08
Manganese	7.6E-10	3.0E-01	1000	2.2E-07
Mercury	7.6E-10	1.2E-03	1000	9.1E-10
Molybdenum	7.6E-10	7.7E-03	1000	5.9E-09
Nickel	7.6E-10	6.2E-02	1000	4.7E-08
Selenium	7.6E-10	9.9E-03	1000	7.5E-09
Strontium	7.6E-10	3.4E+00	1000	2.6E-06
Tin	7.6E-10	3.5E-03	1000	2.6E-09
Uranium	7.6E-10	5.4E-03	1000	4.1E-09
Vanadium	7.6E-10	1.6E-01	1000	1.2E-07
Zinc	7.6E-10	2.1E-01	1000	1.6E-07
Aluminum	7.6E-10	3.5E+01	1000	2.6E-05
Boron	7.6E-10	2.7E-01	1000	2.0E-07
Iron	7.6E-10	3.9E+01	1000	2.9E-05
Titanium	7.6E-10	2.4E+00	1000	1.8E-06
Indium	7.6E-10	1.4E-09	1000	1.1E-15
Lanthanum	7.6E-10	3.7E-09	1000	2.8E-15
Others				
PCBs	7.6E-10	3.0E-12	1000	2.3E-18
Sulphate	7.6E-10	4.6E-07	1000	3.5E-13
Hexachlorobenzene (gas phase)	7.6E-10	0.0E+00	1000	0.0E+00
Dust Pallatives				
Epichlorohydrin	7.6E-10	3.8E-09	1000	2.9E-15

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2

Table II-5B: Project Above Ground Plant Concentations for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	Dd mg/m^2/yr	Dw mg/m^2/yr	Fv percentage	Rp unitless	kp yr^{-1}	Tp yr	Yp kg DW/m^2	Pd mg/kg DW	Log K_{ow} unitless	BCF kg soil/kg plant DW	Cs mg/kg	BCF kg soil/kg plant DW	Pr mg/kg DW	Pd mg/kg	Pr mg/kg	$C_{plant\ ww}$ mg/kg
Polycyclic Aromatic Hydrocarbons																
Carcinogenic PAHs																
Benzo[a]anthracene	3.1E-05	2.0E-04	1%	0.5	18	0.164	2.24	1.8E-06	5.8E+00	1.8E-02	3.7E-04	1.8E-02	6.6E-06	1.8E-06	6.6E-06	1.3E-06
Benzo[a]pyrene	8.0E-06	8.1E-05	1%	0.5	18	0.164	2.24	6.6E-07	6.1E+00	1.1E-02	6.7E-04	1.1E-02	7.5E-06	6.6E-07	7.5E-06	1.2E-06
Benzo[b]fluoranthene	2.3E-05	2.9E-04	1%	0.5	18	0.164	2.24	2.3E-06	5.8E+00	1.8E-02	1.4E-04	1.8E-02	2.5E-06	2.3E-06	2.5E-06	7.1E-07
Benzo[g,h,i]perylene	5.1E-06	2.2E-05	1%	0.5	18	0.164	2.24	2.2E-07	6.6E+00	5.7E-03	2.2E-04	5.7E-03	1.2E-06	2.2E-07	1.2E-06	2.2E-07
Benzo[k]fluoranthene	1.0E-05	6.1E-05	1%	0.5	18	0.164	2.24	5.5E-07	6.1E+00	1.1E-02	5.7E-04	1.1E-02	6.5E-06	5.5E-07	6.5E-06	1.1E-06
Chrysene	2.2E-05	1.2E-04	1%	0.5	18	0.164	2.24	1.1E-06	5.8E+00	1.7E-02	7.8E-04	1.7E-02	1.3E-05	1.1E-06	1.3E-05	2.1E-06
Dibenzo[a,h]anthracene	7.7E-07	1.7E-05	1%	0.5	18	0.164	2.24	1.3E-07	6.8E+00	4.9E-03	1.7E-04	4.9E-03	8.2E-07	1.3E-07	8.2E-07	1.4E-07
Fluoranthene	7.9E-05	5.6E-04	1%	0.5	18	0.164	2.24	4.9E-06	5.2E+00	4.0E-02	2.4E-04	4.0E-02	9.5E-06	4.9E-06	9.5E-06	2.2E-06
Indeno[1,2,3-c,d]pyrene	7.9E-06	5.9E-05	1%	0.5	18	0.164	2.24	5.1E-07	6.7E+00	5.1E-03	5.6E-04	5.1E-03	2.9E-06	5.1E-07	2.9E-06	5.1E-07
Phenanthrene	6.3E-05	1.4E-04	1%	0.5	18	0.164	2.24	1.8E-06	4.5E+00	1.0E-01	4.2E-06	1.0E-01	4.3E-07	1.8E-06	4.3E-07	3.3E-07
Non-carcinogenic PAHs																
Acenaphthene	1.9E-05	1.2E-04	1%	0.5	18	0.164	2.24	1.1E-06	3.9E+00	2.1E-01	3.1E-07	2.1E-01	6.6E-08	1.1E-06	6.6E-08	1.7E-07
Acenaphthylene	4.1E-06	3.3E-05	1%	0.5	18	0.164	2.24	2.8E-07	3.9E+00	2.0E-01	1.1E-07	2.0E-01	2.3E-08	2.8E-07	2.3E-08	4.6E-08
Anthracene	1.4E-05	6.8E-05	1%	0.5	18	0.164	2.24	6.5E-07	4.5E+00	1.0E-01	2.0E-06	1.0E-01	2.1E-07	6.5E-07	2.1E-07	1.3E-07
Fluorene	6.5E-06	6.0E-05	1%	0.5	18	0.164	2.24	5.0E-07	4.2E+00	1.5E-01	4.3E-07	1.5E-01	6.3E-08	5.0E-07	6.3E-08	8.4E-08
Fluoranthene	7.9E-05	5.6E-04	1%	0.5	18	0.164	2.24	4.9E-06	5.2E+00	4.0E-02	2.4E-04	4.0E-02	9.5E-06	4.9E-06	9.5E-06	2.2E-06
Naphthalene	1.2E-04	5.9E-05	1%	0.5	18	0.164	2.24	1.8E-06	3.3E+00	4.8E-01	4.0E-09	4.8E-01	1.9E-09	1.8E-06	1.9E-09	2.7E-07
2-Methylnaphthalene	1.0E-06	2.3E-05	1%	0.5	18	0.164	2.24	1.7E-07	3.9E+00	2.3E-01	7.1E-09	2.3E-01	1.6E-09	1.7E-07	1.6E-09	2.6E-08
Pyrene	1.2E-04	7.1E-04	1%	0.5	18	0.164	2.24	6.5E-06	4.9E+00	5.9E-02	3.1E-04	5.9E-02	1.8E-05	6.5E-06	1.8E-05	3.7E-06
Metals and Metalliods																
Antimony	5.2E-05	5.4E-05	0%	0.5	18	0.164	2.24	9.9E-07	-	2.0E-01	1.6E-03	2.0E-01	3.1E-04	9.9E-07	3.1E-04	4.7E-05
Arsenic	2.7E-05	4.5E-04	0%	0.5	18	0.164	2.24	3.5E-06	-	4.0E-02	2.0E-02	4.0E-02	8.1E-04	3.5E-06	8.1E-04	1.2E-04
Barium	5.5E-03	1.2E-01	0%	0.5	18	0.164	2.24	9.0E-04	-	1.5E-01	5.4E+00	1.5E-01	8.1E-01	9.0E-04	8.1E-01	1.2E-01
Beryllium	4.4E-05	7.8E-05	0%	0.5	18	0.164	2.24	1.1E-06	-	1.0E-02	3.3E-03	1.0E-02	3.3E-05	1.1E-06	3.3E-05	5.1E-06
Cadmium	5.7E-05	4.9E-05	0%	0.5	18	0.164	2.24	1.0E-06	-	5.0E-01	1.7E-03	5.0E-01	8.5E-04	1.0E-06	8.5E-04	1.3E-04
Chromium (III)	1.4E-05	3.6E-05	0%	0.5	18	0.164	2.24	4.1E-07	-	7.5E-03	1.5E-03	7.5E-03	1.1E-05	4.1E-07	1.1E-05	1.8E-06
Chromium (VI)	5.3E-05	1.0E-03	0%	0.5	18	0.164	2.24	7.8E-06	-	7.5E-03	4.6E-02	7.5E-03	3.5E-04	7.8E-06	3.5E-04	5.3E-05
Cobalt	1.9E-05	4.3E-04	0%	0.5	18	0.164	2.24	3.2E-06	-	2.0E-02	1.9E-02	2.0E-02	3.9E-04	3.2E-06	3.9E-04	5.8E-05
Copper	2.1E-04	4.4E-03	0%	0.5	18	0.164	2.24	3.3E-05	-	4.0E-01	2.0E-01	4.0E-01	7.9E-02	3.3E-05	7.9E-02	1.2E-02
Lead	1.7E-04	7.7E-04	0%	0.5	18	0.164	2.24	7.5E-06	-	4.5E-02	3.4E-02	4.5E-02	1.5E-03	7.5E-06	1.5E-03	2.3E-04
Lithium	7.7E-05	1.7E-03	0%	0.5	18	0.164	2.24	1.3E-05	-	2.5E-02	7.7E-02	2.5E-02	1.9E-03	1.3E-05	1.9E-03	2.9E-04
Manganese	3.0E-04	6.5E-03	0%	0.5	18	0.164	2.24	4.9E-05	-	2.5E-01	3.0E-01	2.5E-01	7.4E-02	4.9E-05	7.4E-02	1.1E-02
Mercury	7.2E-06	2.7E-05	1%	0.5	18	0.164	2.24	2.8E-07	-	9.0E-01	1.2E-03	9.0E-01	1.1E-03	2.8E-07	1.1E-03	1.6E-04
Molybdenum	1.9E-05	1.7E-04	0%	0.5	18	0.164	2.24	1.5E-06	-	2.5E-01	7.7E-03	2.5E-01	1.9E-03	1.5E-06	1.9E-03	2.9E-04
Nickel	6.2E-05	1.4E-03	0%	0.5	18	0.164	2.24	1.0E-05	-	6.0E-02	6.2E-02	6.0E-02	3.7E-03	1.0E-05	3.7E-03	5.6E-04
Selenium	1.0E-05	2.2E-04	0%	0.5	18	0.164	2.24	1.7E-06	-	2.5E-02	9.9E-03	2.5E-02	2.5E-04	1.7E-06	2.5E-04	3.7E-05
Strontium	3.4E-03	7.5E-02	0%	0.5	18	0.164	2.24	5.7E-04	-	2.5E+00	3.4E+00	2.5E+00	8.5E+00	5.7E-04	8.5E+00	1.3E+00
Tin	3.2E-05	8.8E-05	0%	0.5	18	0.164	2.24	1.0E-06	-	3.0E-02	3.5E-03	3.0E-02	1.0E-04	1.0E-06	1.0E-04	1.6E-05
Uranium	5.4E-06	1.2E-04	0%	0.5	18	0.164	2.24	9.0E-07	-	8.5E-03	5.4E-03	8.5E-03	4.6E-05	9.0E-07	4.6E-05	7.0E-06
Vanadium	2.3E-04	3.6E-03	0%	0.5	18	0.164	2.24	2.8E-05	-	5.5E-03	1.6E-01	5.5E-03	8.9E-04	2.8E-05	8.9E-04	1.4E-04
Zinc	3.7E-04	4.6E-03	0%	0.5	18	0.164	2.24	3.7E-05	-	9.9E-01	2.1E-01	9.9E-01	2.1E-01	3.7E-05	2.1E-01	3.1E-02
Aluminum	3.5E-02	7.6E-01	0%	0.5	18	0.164	2.24	5.8E-03	-	4.0E-03	3.5E+01	4.0E-03	1.4E-01	5.8E-03	1.4E-01	2.2E-02
Bismuth	1.5E-06	3.4E-05	0%	0.5	18	0.164	2.24	2.6E-07	-		1.5E-03	0.0E+00	0.0E+00	2.6E-07	0.0E+00	3.8E-08
Boron	2.7E-04	5.9E-03	0%	0.5	18	0.164	2.24	4.5E-05	-	4.0E+00	2.7E-01	4.0E+00	1.1E+00	4.5E-05	1.1E+00	1.6E-01
Iron	3.9E-02	8.5E-01	0%	0.5	18	0.164	2.24	6.5E-03	-	4.0E-03	3.9E+01	4.0E-03	1.5E-01	6.5E-03	1.5E-01	2.4E-02
Titanium	2.4E-03	5.3E-02	0%	0.5	18	0.164	2.24	4.0E-04	-	5.5E-03	2.4E+00	5.5E-03	1.3E-02	4.0E-04	1.3E-02	2.0E-03
Indium	3.1E-05	1.2E-05	1%	0.5	18	0.164	2.24	4.5E-07	-	*4.0E-01*	1.4E-09	4.0E-01	5.8E-10	4.5E-07	5.8E-10	6.8E-08
Lanthanum	7.8E-05	3.2E-05	1%	0.5	18	0.164	2.24	1.1E-06	-	*4.0E-01*	3.7E-09	4.0E-01	1.5E-09	1.1E-06	1.5E-09	1.7E-07
Others																
PCBs	1.1E-07	2.3E-08	1%	0.5	18	0.164	2.24	1.4E-09	7.1E+00	2.9E-03	3.0E-12	2.9E-03	8.9E-15	1.4E-09	8.9E-15	2.2E-10
Sulphate	9.9E-03	4.0E-03	1%	0.5	18	0.164	2.24	1.4E-04	-	*4.0E-01*	4.6E-07	4.0E-01	1.9E-07	1.4E-04	1.9E-07	2.2E-05
Hexachlorobenzene (gas phase)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1.7E-13
Dust Pallatives																
Epichlorohydrin	1.2E-05	1.3E-05	1%	0.5	18	0.164	2.24	2.3E-07	4.5E-01	2.1E+01	3.8E-09	2.1E+01	8.1E-08	2.3E-07	8.1E-08	4.7E-08

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)
[2] Log Kow based on US EPA (2004), RAIS (2014), HSDB (2014)
[3] Soil concentration from Equation 1.2
[4] For inorganics soil to dry plant uptake values from ORNL (2014)
- Not available for this parameter
Italics BCF not available from RAIS (2014), BCF assumed to be equal to average of remaining metals

Table II-6B Project Below Ground Plant Concentrations for Maximum North Delta Residential Receptor **Scenario: Project**

Parameter	Cs mg/kg	BCF kg soil/kg plant DW	WPF unitless	WC percentage	Pr_{root} mg/kg ww
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	3.7E-04	1.8E-02	1	0.85	1.0E-06
Benzo[a]pyrene	6.7E-04	1.1E-02	1	0.85	1.1E-06
Benzo[b]fluoranthene	1.4E-04	1.8E-02	1	0.85	3.7E-07
Benzo[g,h,i]perylene	2.2E-04	5.7E-03	1	0.85	1.9E-07
Benzo[k]fluoranthene	5.7E-04	1.1E-02	1	0.85	9.7E-07
Chrysene	7.8E-04	1.7E-02	1	0.85	2.0E-06
Dibenzo[a,h]anthracene	1.7E-04	4.9E-03	1	0.85	1.2E-07
Fluoranthene	2.4E-04	4.0E-02	1	0.85	1.4E-06
Indeno[1,2,3-c,d]pyrene	5.6E-04	5.1E-03	1	0.85	4.3E-07
Phenanthrene	4.2E-06	1.0E-01	1	0.85	6.5E-08
Non-carcinogenic PAHs					
Acenaphthene	3.1E-07	2.1E-01	1	0.85	9.9E-09
Acenaphthylene	1.1E-07	2.0E-01	1	0.85	3.4E-09
Anthracene	2.0E-06	1.0E-01	1	0.85	3.2E-08
Fluorene	4.3E-07	1.5E-01	1	0.85	9.5E-09
Fluoranthene	2.4E-04	4.0E-02	1	0.85	1.4E-06
Naphthalene	4.0E-09	4.8E-01	1	0.85	2.9E-10
2-Methylnaphthalene	7.1E-09	2.3E-01	1	0.85	2.4E-10
Pyrene	3.1E-04	5.9E-02	1	0.85	2.7E-06
Metals and Metalliods					
Antimony	1.6E-03	2.0E-01	1	0.85	4.7E-05
Arsenic	2.0E-02	4.0E-02	1	0.85	1.2E-04
Barium	5.4E+00	1.5E-01	1	0.85	1.2E-01
Beryllium	3.3E-03	1.0E-02	1	0.85	4.9E-06
Cadmium	1.7E-03	5.0E-01	1	0.85	1.3E-04
Chromium (III)	1.5E-03	7.5E-03	1	0.85	1.7E-06
Chromium (VI)	4.6E-02	7.5E-03	1	0.85	5.2E-05
Cobalt	1.9E-02	2.0E-02	1	0.85	5.8E-05
Copper	2.0E-01	4.0E-01	1	0.85	1.2E-02
Lead	3.4E-02	4.5E-02	1	0.85	2.3E-04
Lithium	7.7E-02	2.5E-02	1	0.85	2.9E-04
Manganese	3.0E-01	2.5E-01	1	0.85	1.1E-02
Mercury	1.2E-03	9.0E-01	1	0.85	1.6E-04
Molybdenum	7.7E-03	2.5E-01	1	0.85	2.9E-04
Nickel	6.2E-02	6.0E-02	1	0.85	5.5E-04
Selenium	9.9E-03	2.5E-02	1	0.85	3.7E-05
Strontium	3.4E+00	2.5E+00	1	0.85	1.3E+00
Tin	3.5E-03	3.0E-02	1	0.85	1.6E-05
Uranium	5.4E-03	8.5E-03	1	0.85	6.9E-06
Vanadium	1.6E-01	5.5E-03	1	0.85	1.3E-04
Zinc	2.1E-01	9.9E-01	1	0.85	3.1E-02
Aluminum	3.5E+01	4.0E-03	1	0.85	2.1E-02
Boron	2.7E-01	4.0E+00	1	0.85	1.6E-01
Iron	3.9E+01	4.0E-03	1	0.85	2.3E-02
Titanium	2.4E+00	5.5E-03	1	0.85	2.0E-03
Indium	1.4E-09	4.0E-01	1	0.85	8.7E-11
Lanthanum	3.7E-09	4.0E-01	1	0.85	2.2E-10
Others					
PCBs	3.0E-12	2.9E-03	1	0.85	1.3E-15
Sulphate	4.6E-07	4.0E-01	1	0.85	2.8E-08
Hexachlorobenzene (gas phase)	-	-	-	-	0.0E+00
Dust Pallatives					
Epichlorohydrin	3.8E-09	2.1E+01	1	0.85	1.2E-08

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2
[2] BCF from Equation 3.2.1 for inorganics, for organics soil to dry plant uptake value from ORNL (2014)

Table II-1C Summary of Project Multimedia Exposure Point Concentrations (EPCs) for Maximum Rail Corridor Residential Receptor **Scenario: Project**

Parameter	C_s mg/kg	C_{dust} ug/m^3	C_{plant} mg/kg (wet weight)	Pr_{root} mg/kg	C_{air} ug/m^3
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	5.5E-04	4.2E-10	6.4E-04	1.5E-06	7.4E-05
Benzo[a]pyrene	1.0E-03	7.7E-10	2.6E-04	1.7E-06	3.0E-05
Benzo[b]fluoranthene	2.1E-04	1.6E-10	9.4E-04	5.5E-07	1.1E-04
Benzo[g,h,i]perylene	3.2E-04	2.5E-10	7.1E-05	2.8E-07	8.3E-06
Benzo[k]fluoranthene	8.5E-04	6.4E-10	2.0E-04	1.4E-06	2.3E-05
Chrysene	1.2E-03	8.8E-10	3.9E-04	3.0E-06	4.5E-05
Dibenzo[a,h]anthracene	2.5E-04	1.9E-10	5.5E-05	1.8E-07	6.2E-06
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04
Indeno[1,2,3-c,d]pyrene	8.4E-04	6.4E-10	1.9E-04	6.4E-07	2.2E-05
Phenanthrene	6.3E-06	4.8E-12	4.4E-04	9.7E-08	5.2E-04
Non-carcinogenic PAHs					
Acenaphthene	4.7E-07	3.6E-13	4.0E-04	1.5E-08	4.9E-05
Acenaphthylene	1.7E-07	1.3E-13	1.1E-04	5.1E-09	9.0E-05
Anthracene	3.0E-06	2.3E-12	2.2E-04	4.7E-08	4.2E-05
Fluorene	6.4E-07	4.8E-13	1.9E-04	1.4E-08	4.7E-05
Fluoranthene	3.5E-04	2.7E-10	1.8E-03	2.1E-06	2.1E-04
Naphthalene	5.9E-09	4.5E-15	7.9E-05	4.2E-10	1.7E-05
2-Methylnaphthalene	1.1E-08	8.1E-15	7.4E-05	3.6E-10	8.2E-06
Pyrene	4.6E-04	3.5E-10	2.3E-03	4.1E-06	2.7E-04
Metals and Metalliods					
Antimony	2.3E-03	1.7E-09	1.8E-04	6.9E-05	1.2E-05
Arsenic	3.0E-02	2.3E-08	1.6E-03	1.8E-04	1.6E-04
Barium	8.0E+00	6.1E-06	5.7E-01	1.8E-01	4.3E-02
Beryllium	4.9E-03	3.7E-09	2.4E-04	7.3E-06	3.0E-05
Cadmium	2.6E-03	1.9E-09	3.1E-04	1.9E-04	1.8E-05
Chromium (III)	2.3E-03	1.7E-09	1.1E-04	2.6E-06	3.7E-04
Chromium (VI)	6.9E-02	5.2E-08	3.4E-03	7.8E-05	3.0E-07
Cobalt	2.9E-02	2.2E-08	1.5E-03	8.7E-05	1.6E-04
Copper	3.0E-01	2.2E-07	3.2E-02	1.8E-02	1.6E-03
Lead	5.1E-02	3.9E-08	2.8E-03	3.4E-04	2.9E-04
Manganese	4.4E-01	3.4E-07	3.8E-02	1.7E-02	2.4E-03
Mercury	1.8E-03	1.4E-09	3.3E-04	2.4E-04	1.0E-05
Molybdenum	1.2E-02	8.8E-09	9.9E-04	4.3E-04	6.2E-05
Nickel	9.2E-02	7.0E-08	5.3E-03	8.3E-04	4.9E-04
Selenium	1.5E-02	1.1E-08	7.7E-04	5.6E-05	8.0E-05
Strontium	5.1E+00	3.9E-06	2.1E+00	1.9E+00	2.7E-02
Tin	5.2E-03	3.9E-09	2.7E-04	2.3E-05	2.8E-05
Uranium	8.1E-03	6.1E-09	4.0E-04	1.0E-05	4.3E-05
Vanadium	2.4E-01	1.8E-07	1.2E-02	2.0E-04	1.3E-03
Zinc	3.1E-01	2.4E-07	6.1E-02	4.6E-02	1.7E-03
Aluminum	5.2E+01	3.9E-05	2.5E+00	3.1E-02	2.8E-01
Boron	4.0E-01	3.0E-07	2.6E-01	2.4E-01	2.2E-03
Iron	5.8E+01	4.4E-05	2.8E+00	3.5E-02	3.1E-01
Titanium	3.6E+00	2.7E-06	1.8E-01	3.0E-03	1.9E-02
Others					
PCBs	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Sulfate	0.0E+00	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Hexachlorobenzene (gas phase)	0.0E+00	0.0E+00	0.0E+00	-	
Dust Pallatives					
Epichlorohydrin	9.1E-14	6.9E-20	8.1E-05	2.9E-13	7.4E-02

Notes:

- Not applicable for this parameter

[1] Parameter speciated into individual PAH and metal constituents above

[2] Operable exposure for this parameter limited to inhalation pathway

Table II-2C Project Soil Concentrations for Maximum Rail Corridor Residential Receptor **Scenario: Project**

Parameter	D_{tot} mg/m^2/yr	Zs m	BD kg/m^3	Ds mg/kg/yr	kt yrs^{-1}	tD yrs	C_s mg/kg
Polycyclic Aromatic Hydrocarbons							
Carcinogenic PAHs							
Benzo[a]anthracene	4.0E-02	0.02	1500	1.3E-03	2.4E+00	10	5.5E-04
Benzo[a]pyrene	1.6E-02	0.02	1500	5.4E-04	5.4E-01	10	1.0E-03
Benzo[b]fluoranthene	5.8E-02	0.02	1500	1.9E-03	9.4E+00	10	2.1E-04
Benzo[g,h,i]perylene	4.4E-03	0.02	1500	1.5E-04	4.5E-01	10	3.2E-04
Benzo[k]fluoranthene	1.2E-02	0.02	1500	4.1E-04	4.8E-01	10	8.5E-04
Chrysene	2.4E-02	0.02	1500	8.1E-04	6.9E-01	10	1.2E-03
Dibenzo[a,h]anthracene	3.4E-03	0.02	1500	1.1E-04	4.5E-01	10	2.5E-04
Fluoranthene	1.1E-01	0.02	1500	3.7E-03	1.1E+01	10	3.5E-04
Indeno[1,2,3-c,d]pyrene	1.2E-02	0.02	1500	4.0E-04	4.7E-01	10	8.4E-04
Phenanthrene	2.8E-02	0.02	1500	9.2E-04	1.5E+02	10	6.3E-06
Non-carcinogenic PAHs							
Acenaphthene	2.5E-02	0.02	1500	8.2E-04	1.8E+03	10	4.7E-07
Acenaphthylene	6.6E-03	0.02	1500	2.2E-04	1.3E+03	10	1.7E-07
Anthracene	1.4E-02	0.02	1500	4.5E-04	1.5E+02	10	3.0E-06
Fluorene	1.2E-02	0.02	1500	4.0E-04	6.2E+02	10	6.4E-07
Fluoranthene	1.1E-01	0.02	1500	3.7E-03	1.1E+01	10	3.5E-04
Naphthalene	5.1E-03	0.02	1500	1.7E-04	2.9E+04	10	5.9E-09
2-Methylnaphthalene	4.6E-03	0.02	1500	1.5E-04	1.4E+04	10	1.1E-08
Pyrene	1.4E-01	0.02	1500	4.8E-03	1.0E+01	10	4.6E-04
Metals and Metalloids							
Antimony	6.9E-03	0.02	1500	2.3E-04	2.5E-06	10	2.3E-03
Arsenic	9.1E-02	0.02	1500	3.0E-03	2.5E-06	10	3.0E-02
Barium	2.4E+01	0.02	1500	8.0E-01	2.5E-06	10	8.0E+00
Beryllium	1.5E-02	0.02	1500	4.9E-04	2.5E-06	10	4.9E-03
Cadmium	7.7E-03	0.02	1500	2.6E-04	2.5E-06	10	2.6E-03
Chromium (III)	6.9E-03	0.02	1500	2.3E-04	2.5E-06	10	2.3E-03
Chromium (VI)	2.1E-01	0.02	1500	6.9E-03	2.5E-06	10	6.9E-02
Cobalt	8.7E-02	0.02	1500	2.9E-03	2.5E-06	10	2.9E-02
Copper	8.9E-01	0.02	1500	3.0E-02	2.5E-06	10	3.0E-01
Lead	1.5E-01	0.02	1500	5.1E-03	2.5E-06	10	5.1E-02
Lithium	3.4E-01	0.02	1500	1.1E-02	2.5E-06	10	1.1E-01
Manganese	1.3E+00	0.02	1500	4.4E-02	2.5E-06	10	4.4E-01
Mercury	5.4E-03	0.02	1500	1.8E-04	2.5E-06	10	1.8E-03
Molybdenum	3.5E-02	0.02	1500	1.2E-03	2.5E-06	10	1.2E-02
Nickel	2.8E-01	0.02	1500	9.2E-03	2.5E-06	10	9.2E-02
Selenium	4.4E-02	0.02	1500	1.5E-03	2.5E-06	10	1.5E-02
Strontium	1.5E+01	0.02	1500	5.1E-01	2.5E-06	10	5.1E+00
Tin	1.6E-02	0.02	1500	5.2E-04	2.5E-06	10	5.2E-03
Uranium	2.4E-02	0.02	1500	8.1E-04	2.5E-06	10	8.1E-03
Vanadium	7.2E-01	0.02	1500	2.4E-02	2.5E-06	10	2.4E-01
Zinc	9.3E-01	0.02	1500	3.1E-02	2.5E-06	10	3.1E-01
Aluminum	1.6E+02	0.02	1500	5.2E+00	2.5E-06	10	5.2E+01
Boron	1.2E+00	0.02	1500	4.0E-02	2.5E-06	10	4.0E-01
Iron	1.7E+02	0.02	1500	5.8E+00	2.5E-06	10	5.8E+01
Titanium	1.1E+01	0.02	1500	3.6E-01	2.5E-06	10	3.6E+00
Indium	0.0E+00	0.02	1500	0.0E+00	2.5E-06	10	0.0E+00
Lanthanum	0.0E+00	0.02	1500	0.0E+00	2.5E-06	10	0.0E+00
Others							
PCBs		0.02	1500	0.0E+00	7.8E-02	10	0.0E+00
Sulphate		0.02	1500	0.0E+00	2.5E-06	10	0.0E+00
Hexachlorobenzene (gas phase)		0.02	1500	0.0E+00	4.2E-01	10	0.0E+00
Dust Pallatives							
Epichlorohydrin	1.1E-06	0.02	1500	3.7E-08	4.0E+05	10	9.1E-14

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)

Table II-3C Kt Parameters for Maximum Rail Corridor Residential Receptor **Scenario: Project**

Parameter	CAS	Organic Carbon Partition Coefficient (L/kg)	Water Solubility (mg/L)	Vapor Pressure (mm Hg)	t 1/2 days due to volatilzation	Kv yrs^{-1}	t 1/2 days due to degradation (abiotic and/or biotic)	Source t 1/2 due to degradation (abiotic and/or biotic)	Ks yrs^{-1}	Kt yrs^{-1}
Acenaphthene	000083-32-9	5.03E+03	3.90E+00	2.15E-03	1.4E-01	1.8E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.8E+03
Acenaphthylene	000208-96-8	5.03E+03	1.61E+01	6.68E-03	1.9E-01	1.3E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.3E+03
Aluminum	007429-90-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Anthracene	000120-12-7	1.64E+04	4.34E-02	6.53E-06	1.7E+00	1.5E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.5E+02
Antimony (metallic)	007440-36-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Arsenic, Inorganic	007440-38-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Barium	007440-39-3	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Benz[a]anthracene	000056-55-3	1.77E+05	9.40E-03	2.10E-07	1.3E+02	2.0E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	2.4E+00
Benzo[a]pyrene	000050-32-8	5.87E+05	1.62E-03	5.49E-09	2.7E+03	9.2E-02	570	OEHHA 2000	4.4E-01	5.4E-01
Benzo[b]fluoranthene	000205-99-2	5.99E+05	1.50E-03	5.00E-07	2.8E+01	8.9E+00	570	OEHHA 2000	4.4E-01	9.4E+00
Benzo[g,h,i]perylene	000191-24-2	1.95E+06	2.60E-04	1.00E-10	8.0E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Benzo[k]fluoranthene	000207-08-9	5.87E+05	8.00E-04	9.65E-10	7.7E+03	3.3E-02	570	OEHHA 2000	4.4E-01	4.8E-01
Beryllium and compounds	007440-41-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Boron And Borates Only	007440-42-8	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Butadiene, 1,3-	000106-99-0	3.96E+01	7.35E+02	2.11E+03	2.2E-07	1.2E+09	na	na	na	1.2E+09
Cadmium (Diet)	007440-43-9	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III) (Soluble Particulates)	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III), Insoluble Salts	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(VI)	018540-29-9	-	1.69E+06	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chrysene	000218-01-9	1.81E+05	2.00E-03	6.23E-09	9.2E+02	2.8E-01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	6.9E-01
Cobalt	007440-48-4	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Copper	007440-50-8	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Dibenz[a,h]anthracene	000053-70-3	1.91E+06	2.49E-03	9.55E-10	7.9E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Diethanolamine	000111-42-2	1.00E+00	1.00E+06	2.80E-04	5.6E+01	4.5E+00	na	na	na	4.5E+00
Diethylenetriamine							1.0E+08	assumed	2.5E-06	2.5E-06
Epichlorohydrin	000106-89-8	9.91E+00	6.59E+04	1.64E+01	6.3E-04	4.0E+05	2.8E+01	Environment Canada 2000	9.0E+00	4.0E+05
Fluoranthene	000206-44-0	5.55E+04	2.60E-01	9.22E-06	2.5E+01	1.0E+01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.1E+01
Fluorene	000086-73-7	9.16E+03	1.69E+00	6.00E-04	4.1E-01	6.2E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	6.2E+02
Hexane, N-	000110-54-3	1.32E+02	9.50E+00	1.51E+02	1.3E-07	1.9E+09	na	na	na	1.9E+09
Indeno[1,2,3-cd]pyrene	000193-39-5	3.47E+06	2.20E-05	1.25E-10	9.6E+03	2.6E-02	570	OEHHA 2000	4.4E-01	4.7E-01
Iron	007439-89-6	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lead and Compounds	007439-92-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lithium	007439-93-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Manganese (Diet)	007439-96-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Mercury (elemental)	007439-97-6	-	6.00E-02	1.96E-03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Methylnaphthalene, 2-	000091-57-6	2.48E+03	2.46E+01	5.50E-02	1.8E-02	1.4E+04	32.5	CCME 2008 based on Loehr and Webster 1997	7.8E+00	1.4E+04
Molybdenum	007439-98-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Naphthalene	000091-20-3	1.54E+03	3.10E+01	8.50E-02	8.9E-03	2.9E+04	2.1	Enviornment Canada 1996	1.2E+02	2.9E+04
Nickel Oxide	001313-99-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Refinery Dust	000000-00-7	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Soluble Salts	007440-02-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Phenanthrene	000085-01-8	1.67E+04	1.15E+00	1.21E-04	2.5E+00	1.0E+02	5.7	CCME 2008 based on CEPA 1993	4.4E+01	1.5E+02
Pyrene	000129-00-0	5.43E+04	1.35E-01	4.50E-06	2.6E+01	9.8E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.0E+01
Selenium	007782-49-2	-	-	9.12E+03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Strontium, Stable	007440-24-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Styrene	000100-42-5	4.46E+02	3.10E+02	6.40E+00	3.4E-04	7.4E+05	na	na	na	7.4E+05
Tin	007440-31-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Titanium	007440-32-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Uranium, Insoluble Compounds	007440-61-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Vanadium	000000-06-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Xylenes	001330-20-7	3.83E+02	1.06E+02	7.99E+00	8.0E-05	3.2E+06	na	na	na	3.2E+06
Zinc and Compounds	007440-66-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Indium	007440-74-6	-				na	1.0E+08	OEHHA 2001	2.5E-06	2.5E-06
Lanthanum	007439-91-0	-				na	1.0E+08	OEHHA 2002	2.5E-06	2.5E-06
Hexachlorobenzene	000118-74-1	6.20E+03	6.20E-03	1.80E-05	6.00E+02	4.2E-01	1.0E+09	OEHHA 2003	2.5E-07	4.2E-01
PCBs	001336-36-3	7.81E+04	7.00E-01	8.63E-05		na	3.23E+03	OEHHA 2004	7.8E-02	7.8E-02
Sulphate	014808-79-8	-	1.00E+06	5.93E-05		na	1.0E+08	OEHHA 2005	2.5E-06	2.5E-06

Notes:

Values from RAIS (2014) unless otherwise noted

CCME. 2008. Canadian Council of Ministers of the Enviroment. Canada-Wide Standard (CWS) for Petroleum Hydrocarbons (PHC) in Soil. Scientifc Rationale Supporting Technical Document. CCME. 2008

OEHHA 2000. Office of Environmental Health Hazard Assessment. California EPA. Technical Support Document for Exposure Assessment and Stochastic Analysis. Air Toxics Hot Spots Program Risk Assessment Guidelines. Oakland. California. 2000

Environment Canada. 2000. Screening Assessment: oxirane, (chloromethyl)-,epichlorohydrin. Chemical Abstracts Service Registry Number 106-89-8. Environment Canada. Health Canada. November 2008

	Calculated based on first order decay rate: k=0.693/(t1/2 / 365)
assumed	Based on lack of data from above sources, 10^8 t 1/2 based on professional judgement
na	Not availble from sources idenfied above

Table II-4C Project Soil Particulate/Dust Concentations for Maximum Rail Corridor Residential Receptor **Scenario: Project**

Parameter	DL kg/m^3	Cs mg/kg	CF ug/mg	C_{dust} ug/m^3
Polycyclic Aromatic Hydrocarbons				
Carcinogenic PAHs				
Benzo[a]anthracene	7.6E-10	5.5E-04	1000	4.2E-10
Benzo[a]pyrene	7.6E-10	1.0E-03	1000	7.7E-10
Benzo[b]fluoranthene	7.6E-10	2.1E-04	1000	1.6E-10
Benzo[g,h,i]perylene	7.6E-10	3.2E-04	1000	2.5E-10
Benzo[k]fluoranthene	7.6E-10	8.5E-04	1000	6.4E-10
Chrysene	7.6E-10	1.2E-03	1000	8.8E-10
Dibenzo[a,h]anthracene	7.6E-10	2.5E-04	1000	1.9E-10
Fluoranthene	7.6E-10	3.5E-04	1000	2.7E-10
Indeno[1,2,3-c,d]pyrene	7.6E-10	8.4E-04	1000	6.4E-10
Phenanthrene	7.6E-10	6.3E-06	1000	4.8E-12
Non-carcinogenic PAHs				
Acenaphthene	7.6E-10	4.7E-07	1000	3.6E-13
Acenaphthylene	7.6E-10	1.7E-07	1000	1.3E-13
Anthracene	7.6E-10	3.0E-06	1000	2.3E-12
Fluorene	7.6E-10	6.4E-07	1000	4.8E-13
Fluoranthene	7.6E-10	3.5E-04	1000	2.7E-10
Naphthalene	7.6E-10	5.9E-09	1000	4.5E-15
2-Methylnaphthalene	7.6E-10	1.1E-08	1000	8.1E-15
Pyrene	7.6E-10	4.6E-04	1000	3.5E-10
Metals and Metalloids				
Antimony	7.6E-10	2.3E-03	1000	1.7E-09
Arsenic	7.6E-10	3.0E-02	1000	2.3E-08
Barium	7.6E-10	8.0E+00	1000	6.1E-06
Beryllium	7.6E-10	4.9E-03	1000	3.7E-09
Cadmium	7.6E-10	2.6E-03	1000	1.9E-09
Chromium (III)	7.6E-10	2.3E-03	1000	1.7E-09
Chromium (VI)	7.6E-10	6.9E-02	1000	5.2E-08
Cobalt	7.6E-10	2.9E-02	1000	2.2E-08
Copper	7.6E-10	3.0E-01	1000	2.2E-07
Lead	7.6E-10	5.1E-02	1000	3.9E-08
Lithium	7.6E-10	1.1E-01	1000	8.7E-08
Manganese	7.6E-10	4.4E-01	1000	3.4E-07
Mercury	7.6E-10	1.8E-03	1000	1.4E-09
Molybdenum	7.6E-10	1.2E-02	1000	8.8E-09
Nickel	7.6E-10	9.2E-02	1000	7.0E-08
Selenium	7.6E-10	1.5E-02	1000	1.1E-08
Strontium	7.6E-10	5.1E+00	1000	3.9E-06
Tin	7.6E-10	5.2E-03	1000	3.9E-09
Uranium	7.6E-10	8.1E-03	1000	6.1E-09
Vanadium	7.6E-10	2.4E-01	1000	1.8E-07
Zinc	7.6E-10	3.1E-01	1000	2.4E-07
Aluminum	7.6E-10	5.2E+01	1000	3.9E-05
Boron	7.6E-10	4.0E-01	1000	3.0E-07
Iron	7.6E-10	5.8E+01	1000	4.4E-05
Titanium	7.6E-10	3.6E+00	1000	2.7E-06
Indium	7.6E-10	0.0E+00	1000	0.0E+00
Lanthanum	7.6E-10	0.0E+00	1000	0.0E+00
Others				
PCBs	7.6E-10	0.0E+00	1000	0.0E+00
Sulphate	7.6E-10	0.0E+00	1000	0.0E+00
Hexachlorobenzene (gas phase)	7.6E-10	0.0E+00	1000	0.0E+00
Dust Pallatives				
Epichlorohydrin	7.6E-10	9.1E-14	1000	6.9E-20

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2

Table II-5C: Project Above Ground Plant Concentations for Maximum Rail Corridor Receptor **Scenario: Project**

Parameter	Dd mg/m^2/yr	Dw mg/m^2/yr	Fv percentage	Rp unitless	kp yr^{-1}	Tp yr	Yp kg DW/m^2	Pd mg/kg DW	Log K_{ow} unitless	BCF kg soil/kg plant DW	Cs mg/kg	BCF kg soil/kg plant DW	Pr mg/kg DW	Pd mg/kg	Pr mg/kg	$C_{plant\ ww}$ mg/kg
Polycyclic Aromatic Hydrocarbons																
Carcinogenic PAHs																
Benzo[a]anthracene	3.6E-01	5.4E-04	1%	0.5	18	0.164	2.24	4.3E-03	5.8E+00	1.8E-02	5.5E-04	1.8E-02	9.9E-06	4.3E-03	9.9E-06	6.4E-04
Benzo[a]pyrene	1.5E-01	2.2E-04	1%	0.5	18	0.164	2.24	1.7E-03	6.1E+00	1.1E-02	1.0E-03	1.1E-02	1.1E-05	1.7E-03	1.1E-05	2.6E-04
Benzo[b]fluoranthene	5.3E-01	7.8E-04	1%	0.5	18	0.164	2.24	6.2E-03	5.8E+00	1.8E-02	2.1E-04	1.8E-02	3.7E-06	6.2E-03	3.7E-06	9.4E-04
Benzo[g,h,i]perylene	4.0E-02	5.9E-05	1%	0.5	18	0.164	2.24	4.7E-04	6.6E+00	5.7E-03	3.2E-04	5.7E-03	1.8E-06	4.7E-04	1.8E-06	7.1E-05
Benzo[k]fluoranthene	1.1E-01	1.6E-04	1%	0.5	18	0.164	2.24	1.3E-03	6.1E+00	1.1E-02	8.5E-04	1.1E-02	9.7E-06	1.3E-03	9.7E-06	2.0E-04
Chrysene	2.2E-01	3.3E-04	1%	0.5	18	0.164	2.24	2.6E-03	5.8E+00	1.7E-02	1.2E-03	1.7E-02	2.0E-05	2.6E-03	2.0E-05	3.9E-04
Dibenzo[a,h]anthracene	3.1E-02	4.6E-05	1%	0.5	18	0.164	2.24	3.7E-04	6.8E+00	4.9E-03	2.5E-04	4.9E-03	1.2E-06	3.7E-04	1.2E-06	5.5E-05
Fluoranthene	1.0E+00	1.5E-03	1%	0.5	18	0.164	2.24	1.2E-02	5.2E+00	4.0E-02	3.5E-04	4.0E-02	1.4E-05	1.2E-02	1.4E-05	1.8E-03
Indeno[1,2,3-c,d]pyrene	1.1E-01	1.6E-04	1%	0.5	18	0.164	2.24	1.3E-03	6.7E+00	5.1E-03	8.4E-04	5.1E-03	4.3E-06	1.3E-03	4.3E-06	1.9E-04
Phenanthrene	2.5E-01	3.7E-04	1%	0.5	18	0.164	2.24	2.9E-03	4.5E+00	1.0E-01	6.3E-06	1.0E-01	6.5E-07	2.9E-03	6.5E-07	4.4E-04
Non-carcinogenic PAHs																
Acenaphthene	2.2E-01	3.3E-04	1%	0.5	18	0.164	2.24	2.6E-03	3.9E+00	2.1E-01	4.7E-07	2.1E-01	9.8E-08	2.6E-03	9.8E-08	4.0E-04
Acenaphthylene	6.0E-02	8.9E-05	1%	0.5	18	0.164	2.24	7.1E-04	3.9E+00	2.0E-01	1.7E-07	2.0E-01	3.4E-08	7.1E-04	3.4E-08	1.1E-04
Anthracene	1.2E-01	1.8E-04	1%	0.5	18	0.164	2.24	1.5E-03	4.5E+00	1.0E-01	3.0E-06	1.0E-01	3.1E-07	1.5E-03	3.1E-07	2.2E-04
Fluorene	1.1E-01	1.6E-04	1%	0.5	18	0.164	2.24	1.3E-03	4.2E+00	1.5E-01	6.4E-07	1.5E-01	9.5E-08	1.3E-03	9.5E-08	1.9E-04
Fluoranthene	1.0E+00	1.5E-03	1%	0.5	18	0.164	2.24	1.2E-02	5.2E+00	4.0E-02	3.5E-04	4.0E-02	1.4E-05	1.2E-02	1.4E-05	1.8E-03
Naphthalene	4.5E-02	7.5E-05	1%	0.5	18	0.164	2.24	5.3E-04	3.3E+00	4.8E-01	5.9E-09	4.8E-01	2.8E-09	5.3E-04	2.8E-09	7.9E-05
2-Methylnaphthalene	4.2E-02	6.1E-05	1%	0.5	18	0.164	2.24	4.9E-04	3.9E+00	2.3E-01	1.1E-08	2.3E-01	2.4E-09	4.9E-04	2.4E-09	7.4E-05
Pyrene	1.3E+00	1.9E-03	1%	0.5	18	0.164	2.24	1.5E-02	4.9E+00	5.9E-02	4.6E-04	5.9E-02	2.7E-05	1.5E-02	2.7E-05	2.3E-03
Metals and Metalliods																
Antimony	6.3E-02	9.2E-05	0%	0.5	18	0.164	2.24	7.4E-04	-	2.0E-01	2.3E-03	2.0E-01	4.6E-04	7.4E-04	4.6E-04	1.8E-04
Arsenic	8.3E-01	1.2E-03	0%	0.5	18	0.164	2.24	9.7E-03	-	4.0E-02	3.0E-02	4.0E-02	1.2E-03	9.7E-03	1.2E-03	1.6E-03
Barium	2.2E+02	3.2E-01	0%	0.5	18	0.164	2.24	2.6E+00	-	1.5E-01	8.0E+00	1.5E-01	1.2E+00	2.6E+00	1.2E+00	5.7E-01
Beryllium	1.3E-01	2.0E-04	0%	0.5	18	0.164	2.24	1.6E-03	-	1.0E-02	4.9E-03	1.0E-02	4.9E-05	1.6E-03	4.9E-05	2.4E-04
Cadmium	6.9E-02	1.1E-04	0%	0.5	18	0.164	2.24	8.1E-04	-	5.0E-01	2.6E-03	5.0E-01	1.3E-03	8.1E-04	1.3E-03	3.1E-04
Chromium (III)	6.3E-02	9.2E-05	0%	0.5	18	0.164	2.24	7.4E-04	-	7.5E-03	2.3E-03	7.5E-03	1.7E-05	7.4E-04	1.7E-05	1.1E-04
Chromium (VI)	1.9E+00	2.8E-03	0%	0.5	18	0.164	2.24	2.2E-02	-	7.5E-03	6.9E-02	7.5E-03	5.2E-04	2.2E-02	5.2E-04	3.4E-03
Cobalt	7.9E-01	1.2E-03	0%	0.5	18	0.164	2.24	9.3E-03	-	2.0E-02	2.9E-02	2.0E-02	5.8E-04	9.3E-03	5.8E-04	1.5E-03
Copper	8.1E+00	1.2E-02	0%	0.5	18	0.164	2.24	9.5E-02	-	4.0E-01	3.0E-01	4.0E-01	1.2E-01	9.5E-02	1.2E-01	3.2E-02
Lead	1.4E+00	2.1E-03	0%	0.5	18	0.164	2.24	1.6E-02	-	4.5E-02	5.1E-02	4.5E-02	2.3E-03	1.6E-02	2.3E-03	2.8E-03
Lithium	3.1E+00	4.6E-03	0%	0.5	18	0.164	2.24	3.7E-02	-	2.5E-02	1.1E-01	2.5E-02	2.9E-03	3.7E-02	2.9E-03	5.9E-03
Manganese	1.2E+01	1.8E-02	0%	0.5	18	0.164	2.24	1.4E-01	-	2.5E-01	4.4E-01	2.5E-01	1.1E-01	1.4E-01	1.1E-01	3.8E-02
Mercury	4.9E-02	7.3E-05	1%	0.5	18	0.164	2.24	5.8E-04	-	1.8E-03	5.8E-04	9.0E-01	1.6E-03	5.8E-04	1.6E-03	3.3E-04
Molybdenum	3.2E-01	4.6E-04	0%	0.5	18	0.164	2.24	3.7E-03	-	2.5E-01	1.2E-02	2.5E-01	2.9E-03	3.7E-03	2.9E-03	9.9E-04
Nickel	2.5E+00	3.7E-03	0%	0.5	18	0.164	2.24	3.0E-02	-	6.0E-02	9.2E-02	6.0E-02	5.5E-03	3.0E-02	5.5E-03	5.3E-03
Selenium	4.1E-01	6.0E-04	0%	0.5	18	0.164	2.24	4.8E-03	-	2.5E-02	1.5E-02	2.5E-02	3.7E-04	4.8E-03	3.7E-04	7.7E-04
Strontium	1.4E+02	2.0E-01	0%	0.5	18	0.164	2.24	1.6E+00	-	2.5E+00	5.1E+00	2.5E+00	1.3E+01	1.6E+00	1.3E+01	2.1E+00
Tin	1.4E-01	2.1E-04	0%	0.5	18	0.164	2.24	1.7E-03	-	3.0E-02	5.2E-03	3.0E-02	1.6E-04	1.7E-03	1.6E-04	2.7E-04
Uranium	2.2E-01	3.2E-04	0%	0.5	18	0.164	2.24	2.6E-03	-	8.5E-03	8.1E-03	8.5E-03	6.9E-05	2.6E-03	6.9E-05	4.0E-04
Vanadium	6.6E+00	9.7E-03	0%	0.5	18	0.164	2.24	7.7E-02	-	5.5E-03	2.4E-01	5.5E-03	1.3E-03	7.7E-02	1.3E-03	1.2E-02
Zinc	8.5E+00	1.2E-02	0%	0.5	18	0.164	2.24	1.0E-01	-	9.9E-01	3.1E-01	9.9E-01	3.1E-01	1.0E-01	3.1E-01	6.1E-02
Aluminum	1.4E+03	2.1E+00	0%	0.5	18	0.164	2.24	1.7E+01	-	4.0E-03	5.2E+01	4.0E-03	2.1E-01	1.7E+01	2.1E-01	2.5E+00
Boron	1.1E+01	1.6E-02	0%	0.5	18	0.164	2.24	1.3E-01	-	4.0E+00	4.0E-01	4.0E+00	1.6E+00	1.3E-01	1.6E+00	2.6E-01
Iron	1.6E+03	2.3E+00	0%	0.5	18	0.164	2.24	1.9E+01	-	4.0E-03	5.8E+01	4.0E-03	2.3E-01	1.9E+01	2.3E-01	2.8E+00
Titanium	9.8E+01	1.4E-01	0%	0.5	18	0.164	2.24	1.2E+00	-	5.5E-03	3.6E+00	5.5E-03	2.0E-02	1.2E+00	2.0E-02	1.8E-01
Indium	0.0E+00	0.0E+00	1%	0.5	18	0.164	2.24	0.0E+00	-	*4.0E-01*	0.0E+00	4.0E-01	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Lanthanum	0.0E+00	0.0E+00	1%	0.5	18	0.164	2.24	0.0E+00	-	*4.0E-01*	0.0E+00	4.0E-01	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Others																
PCBs	0.0E+00	0.0E+00	1%	0.5	18	0.164	2.24	0.0E+00	6.1E+00	2.9E-03	0.0E+00	2.9E-03	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Sulphate	0.0E+00	0.0E+00	1%	0.5	18	0.164	2.24	0.0E+00	7.1E+00	*4.0E-01*	0.0E+00	4.0E-01	0.0E+00	0.0E+00	0.0E+00	0.0E+00
Hexachlorobenzene (gas phase)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	0.0E+00
Dust Pallatives																
Epichlorohydrin	4.6E-02	3.6E-04	1%	0.5	18	0.164	2.24	5.4E-04	4.5E-01	2.1E+01	9.1E-14	2.1E+01	1.9E-12	5.4E-04	1.9E-12	8.1E-05

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)
[2] Log Kow based on US EPA (2004), RAIS (2014), HSDB (2014)
[3] Soil concentration from Equation 1.2
[4] For inorganics soil to dry plant uptake values from ORNL (2014)
- Not available/required for this parameter
Italics BCF assumed to be equal to average of remaining metals

Table II-6C Project Below Ground Plant Concentrations for Maximum Rail Corridor Residential Receptor **Scenario: Project**

Parameter	Cs mg/kg	BCF kg soil/kg plant DW	WPF unitless	WC percentage	Pr_{root} mg/kg ww
Polycyclic Aromatic Hydrocarbons					
Carcinogenic PAHs					
Benzo[a]anthracene	5.5E-04	1.8E-02	1	0.85	1.5E-06
Benzo[a]pyrene	1.0E-03	1.1E-02	1	0.85	1.7E-06
Benzo[b]fluoranthene	2.1E-04	1.8E-02	1	0.85	5.5E-07
Benzo[g,h,i]perylene	3.2E-04	5.7E-03	1	0.85	2.8E-07
Benzo[k]fluoranthene	8.5E-04	1.1E-02	1	0.85	1.4E-06
Chrysene	1.2E-03	1.7E-02	1	0.85	3.0E-06
Dibenzo[a,h]anthracene	2.5E-04	4.9E-03	1	0.85	1.8E-07
Fluoranthene	3.5E-04	4.0E-02	1	0.85	2.1E-06
Indeno[1,2,3-c,d]pyrene	8.4E-04	5.1E-03	1	0.85	6.4E-07
Phenanthrene	6.3E-06	1.0E-01	1	0.85	9.7E-08
Non-carcinogenic PAHs					
Acenaphthene	4.7E-07	2.1E-01	1	0.85	1.5E-08
Acenaphthylene	1.7E-07	2.0E-01	1	0.85	5.1E-09
Anthracene	3.0E-06	1.0E-01	1	0.85	4.7E-08
Fluorene	6.4E-07	1.5E-01	1	0.85	1.4E-08
Fluoranthene	3.5E-04	4.0E-02	1	0.85	2.1E-06
Naphthalene	5.9E-09	4.8E-01	1	0.85	4.2E-10
2-Methylnaphthalene	1.1E-08	2.3E-01	1	0.85	3.6E-10
Pyrene	4.6E-04	5.9E-02	1	0.85	4.1E-06
Metals and Metalliods					
Antimony	2.3E-03	2.0E-01	1	0.85	6.9E-05
Arsenic	3.0E-02	4.0E-02	1	0.85	1.8E-04
Barium	8.0E+00	1.5E-01	1	0.85	1.8E-01
Beryllium	4.9E-03	1.0E-02	1	0.85	7.3E-06
Cadmium	2.6E-03	5.0E-01	1	0.85	1.9E-04
Chromium (III)	2.3E-03	7.5E-03	1	0.85	2.6E-06
Chromium (VI)	6.9E-02	7.5E-03	1	0.85	7.8E-05
Cobalt	2.9E-02	2.0E-02	1	0.85	8.7E-05
Copper	3.0E-01	4.0E-01	1	0.85	1.8E-02
Lead	5.1E-02	4.5E-02	1	0.85	3.4E-04
Lithium	1.1E-01	2.5E-02	1	0.85	4.3E-04
Manganese	4.4E-01	2.5E-01	1	0.85	1.7E-02
Mercury	1.8E-03	9.0E-01	1	0.85	2.4E-04
Molybdenum	1.2E-02	2.5E-01	1	0.85	4.3E-04
Nickel	9.2E-02	6.0E-02	1	0.85	8.3E-04
Selenium	1.5E-02	2.5E-02	1	0.85	5.6E-05
Strontium	5.1E+00	2.5E+00	1	0.85	1.9E+00
Tin	5.2E-03	3.0E-02	1	0.85	2.3E-05
Uranium	8.1E-03	8.5E-03	1	0.85	1.0E-05
Vanadium	2.4E-01	5.5E-03	1	0.85	2.0E-04
Zinc	3.1E-01	9.9E-01	1	0.85	4.6E-02
Aluminum	5.2E+01	4.0E-03	1	0.85	3.1E-02
Boron	4.0E-01	4.0E+00	1	0.85	2.4E-01
Iron	5.8E+01	4.0E-03	1	0.85	3.5E-02
Titanium	3.6E+00	5.5E-03	1	0.85	3.0E-03
Indium	0.0E+00	4.0E-01	1	0.85	0.0E+00
Lanthanum	0.0E+00	4.0E-01	1	0.85	0.0E+00
Others					
PCBs	0.0E+00	2.9E-03	1	0.85	0.0E+00
Sulphate	0.0E+00	4.0E-01	1	0.85	0.0E+00
Hexachlorobenzene (gas phase)	-	-	-	-	-
Dust Pallatives					
Epichlorohydrin	9.1E-14	2.1E+01	1	0.85	2.9E-13

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2
[2] BCF from Equation 3.2.1 for inorganics, for organics soil to dry plant uptake value from ORNL (2014)

Table II-1D Summary of Project Multimedia Exposure Point Concentrations (EPCs) for Industrial Receptor

Parameter	C_s mg/kg	C_{dust} ug/m^3	C_{air} ug/m^3
Polycyclic Aromatic Hydrocarbons			
Carcinogenic PAHs			
Benzo[a]anthracene	2.3E-03	1.7E-09	4.3E-03
Benzo[a]pyrene	4.2E-03	3.2E-09	7.5E-05
Benzo[b]fluoranthene	8.7E-04	6.6E-10	2.7E-04
Benzo[g,h,i]perylene	1.4E-03	1.0E-09	2.2E-05
Benzo[k]fluoranthene	3.5E-03	2.7E-09	5.7E-05
Chrysene	4.9E-03	3.7E-09	1.1E-04
Dibenzo[a,h]anthracene	1.1E-03	8.0E-10	1.6E-05
Fluoranthene	1.5E-03	1.1E-09	5.2E-04
Indeno[1,2,3-c,d]pyrene	3.5E-03	2.7E-09	5.7E-05
Phenanthrene	2.6E-05	2.0E-11	7.0E-04
Non-carcinogenic PAHs			
Acenaphthene	2.0E-06	1.5E-12	1.2E-04
Acenaphthylene	7.0E-07	5.3E-13	1.3E-04
Anthracene	1.3E-05	9.6E-12	4.2E-03
Fluorene	2.7E-06	2.0E-12	9.5E-05
Fluoranthene	1.5E-03	1.1E-09	5.2E-04
Naphthalene	2.5E-08	1.9E-14	5.2E-04
2-Methylnaphthalene	4.4E-08	3.4E-14	2.1E-05
Pyrene	1.9E-03	1.5E-09	6.7E-04
Metals and Metalliods			
Antimony	9.9E-03	7.5E-09	2.2E-04
Arsenic	1.3E-01	9.6E-08	4.2E-04
Barium	3.4E+01	2.6E-05	1.1E-01
Beryllium	2.0E-02	1.5E-08	7.1E-05
Cadmium	1.1E-02	8.1E-09	1.1E-04
Chromium (III)	9.6E-03	7.3E-09	9.4E-04
Chromium (VI)	2.9E-01	2.2E-07	5.7E-06
Cobalt	1.2E-01	9.2E-08	3.9E-04
Copper	1.2E+00	9.4E-07	4.0E-03
Lead	2.1E-01	1.6E-07	7.4E-04
Manganese	1.8E+00	1.4E-06	6.0E-03
Mercury	7.5E-03	5.7E-09	2.7E-05
Molybdenum	4.8E-02	3.7E-08	2.0E-04
Nickel	3.8E-01	2.9E-07	1.2E-03
Selenium	6.2E-02	4.7E-08	2.0E-04
Strontium	2.1E+01	1.6E-05	6.9E-02
Tin	2.2E-02	1.7E-08	1.8E-04
Uranium	3.4E-02	2.6E-08	1.1E-04
Vanadium	1.0E+00	7.7E-07	3.5E-03
Zinc	1.3E+00	9.9E-07	4.8E-03
Aluminum	2.2E+02	1.6E-04	7.0E-01
Boron	1.7E+00	1.3E-06	5.4E-03
Iron	2.4E+02	1.8E-04	7.8E-01
Titanium	1.5E+01	1.1E-05	4.9E-02
Indium	2.1E-04	1.6E-10	1.2E-04
Lanthanum	5.2E-04	4.0E-10	2.9E-04
Others			
Hexachlorobenzene	8.6E-09	6.5E-15	2.9E-04
PCBs	3.2E-07	2.4E-13	1.7E-07
Sulfate	6.6E-02	5.0E-08	3.7E-02
Hexachlorobenzene (gas phase)	8.6E-09	6.5E-15	2.9E-04
Dust Pallatives			
Epichlorohydrin	6.0E-10	4.5E-16	6.0E-05

Notes:

- Not applicable for this parameter

[1] Parameter speciated into individual PAH and metal constituents above

[2] Operable exposure for this parameter limited to inhalation pathway

Table II-2D Project Soil Concentrations for Industrial Receptor **Scenario: Project**

Parameter	D_{tot} mg/m^2/yr	Zs m	BD kg/m^3	Ds mg/kg/yr	kt yrs^{-1}	tD yrs	C_s mg/kg
Polycyclic Aromatic Hydrocarbons							
Carcinogenic PAHs							
Benzo[a]anthracene	1.7E-01	0.02	1500	5.6E-03	2.4E+00	10	2.3E-03
Benzo[a]pyrene	6.8E-02	0.02	1500	2.3E-03	5.4E-01	10	4.2E-03
Benzo[b]fluoranthene	2.4E-01	0.02	1500	8.1E-03	9.4E+00	10	8.7E-04
Benzo[g,h,i]perylene	1.8E-02	0.02	1500	6.1E-04	4.5E-01	10	1.4E-03
Benzo[k]fluoranthene	5.1E-02	0.02	1500	1.7E-03	4.8E-01	10	3.5E-03
Chrysene	1.0E-01	0.02	1500	3.4E-03	6.9E-01	10	4.9E-03
Dibenzo[a,h]anthracene	1.4E-02	0.02	1500	4.8E-04	4.5E-01	10	1.1E-03
Fluoranthene	4.7E-01	0.02	1500	1.6E-02	1.1E+01	10	1.5E-03
Indeno[1,2,3-c,d]pyrene	5.0E-02	0.02	1500	1.7E-03	4.7E-01	10	3.5E-03
Phenanthrene	1.1E-01	0.02	1500	3.8E-03	1.5E+02	10	2.6E-05
Non-carcinogenic PAHs							
Acenaphthene	1.0E-01	0.02	1500	3.4E-03	1.8E+03	10	2.0E-06
Acenaphthylene	2.8E-02	0.02	1500	9.3E-04	1.3E+03	10	7.0E-07
Anthracene	5.7E-02	0.02	1500	1.9E-03	1.5E+02	10	1.3E-05
Fluorene	5.0E-02	0.02	1500	1.7E-03	6.2E+02	10	2.7E-06
Fluoranthene	4.7E-01	0.02	1500	1.6E-02	1.1E+01	10	1.5E-03
Naphthalene	2.2E-02	0.02	1500	7.3E-04	2.9E+04	10	2.5E-08
2-Methylnaphthalene	1.9E-02	0.02	1500	6.4E-04	1.4E+04	10	4.4E-08
Pyrene	6.0E-01	0.02	1500	2.0E-02	1.0E+01	10	1.9E-03
Metals and Metalloids							
Antimony	3.0E-02	0.02	1500	9.9E-04	2.5E-06	10	9.9E-03
Arsenic	3.8E-01	0.02	1500	1.3E-02	2.5E-06	10	1.3E-01
Barium	1.0E+02	0.02	1500	3.4E+00	2.5E-06	10	3.4E+01
Beryllium	6.1E-02	0.02	1500	2.0E-03	2.5E-06	10	2.0E-02
Cadmium	3.2E-02	0.02	1500	1.1E-03	2.5E-06	10	1.1E-02
Chromium (III)	2.9E-02	0.02	1500	9.6E-04	2.5E-06	10	9.6E-03
Chromium (VI)	8.6E-01	0.02	1500	2.9E-02	2.5E-06	10	2.9E-01
Cobalt	3.6E-01	0.02	1500	1.2E-02	2.5E-06	10	1.2E-01
Copper	3.7E+00	0.02	1500	1.2E-01	2.5E-06	10	1.2E+00
Lead	6.4E-01	0.02	1500	2.1E-02	2.5E-06	10	2.1E-01
Lithium	1.4E+00	0.02	1500	4.8E-02	2.5E-06	10	4.8E-01
Manganese	5.5E+00	0.02	1500	1.8E-01	2.5E-06	10	1.8E+00
Mercury	2.2E-02	0.02	1500	7.5E-04	2.5E-06	10	7.5E-03
Molybdenum	1.4E-01	0.02	1500	4.8E-03	2.5E-06	10	4.8E-02
Nickel	1.2E+00	0.02	1500	3.8E-02	2.5E-06	10	3.8E-01
Selenium	1.9E-01	0.02	1500	6.2E-03	2.5E-06	10	6.2E-02
Strontium	6.4E+01	0.02	1500	2.1E+00	2.5E-06	10	2.1E+01
Tin	6.6E-02	0.02	1500	2.2E-03	2.5E-06	10	2.2E-02
Uranium	1.0E-01	0.02	1500	3.4E-03	2.5E-06	10	3.4E-02
Vanadium	3.0E+00	0.02	1500	1.0E-01	2.5E-06	10	1.0E+00
Zinc	3.9E+00	0.02	1500	1.3E-01	2.5E-06	10	1.3E+00
Aluminum	6.5E+02	0.02	1500	2.2E+01	2.5E-06	10	2.2E+02
Boron	5.0E+00	0.02	1500	1.7E-01	2.5E-06	10	1.7E+00
Iron	7.3E+02	0.02	1500	2.4E+01	2.5E-06	10	2.4E+02
Titanium	4.5E+01	0.02	1500	1.5E+00	2.5E-06	10	1.5E+01
Indium	6.2E-04	0.02	1500	2.1E-05	2.5E-06	10	2.1E-04
Lanthanum	1.6E-03	0.02	1500	5.2E-05	2.5E-06	10	5.2E-04
Others							
PCBs	1.4E-06	0.02	1500	4.6E-08	7.8E-02	10	3.2E-07
Sulphate	2.0E-01	0.02	1500	6.6E-03	2.5E-06	10	6.6E-02
Hexachlorobenzene (gas phase)	1.1E-07	0.02	1500	3.7E-09	4.2E-01	10	8.6E-09
Dust Pallatives							
Epichlorohydrin	7.2E-03	0.02	1500	2.4E-04	4.0E+05	10	6.0E-10

Notes:
Equations based on US EPA OWS (2005)
[1] Based on Levelton (2014)

Table II-3D Kt Parameters for Industrial Receptor

Scenario: Project

Parameter	CAS	Organic Carbon Partition Coefficient (L/kg)	Water Solubility (mg/L)	Vapor Pressure (mm Hg)	t 1/2 days due to volatilzation	Kv yrs^{-1}	t 1/2 days due to degradation (abiotic and/or biotic)	Source t 1/2 due to degradation (abiotic and/or biotic)	Ks yrs^{-1}	Kt yrs^{-1}
Acenaphthene	000083-32-9	5.03E+03	3.90E+00	2.15E-03	1.4E-01	1.8E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.8E+03
Acenaphthylene	000208-96-8	5.03E+03	1.61E+01	6.68E-03	1.9E-01	1.3E+03	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.3E+03
Aluminum	007429-90-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Anthracene	000120-12-7	1.64E+04	4.34E-02	6.53E-06	1.7E+00	1.5E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	1.5E+02
Antimony (metallic)	007440-36-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Arsenic, Inorganic	007440-38-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Barium	007440-39-3	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Benz[a]anthracene	000056-55-3	1.77E+05	9.40E-03	2.10E-07	1.3E+02	2.0E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	2.4E+00
Benzo[a]pyrene	000050-32-8	5.87E+05	1.62E-03	5.49E-09	2.7E+03	9.2E-02	570	OEHHA 2000	4.4E-01	5.4E-01
Benzo[b]fluoranthene	000205-99-2	5.99E+05	1.50E-03	5.00E-07	2.8E+01	8.9E+00	570	OEHHA 2000	4.4E-01	9.4E+00
Benzo[g,h,i]perylene	000191-24-2	1.95E+06	2.60E-04	1.00E-10	8.0E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Benzo[k]fluoranthene	000207-08-9	5.87E+05	8.00E-04	9.65E-10	7.7E+03	3.3E-02	570	OEHHA 2000	4.4E-01	4.8E-01
Beryllium and compounds	007440-41-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Boron And Borates Only	007440-42-8	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Butadiene, 1,3-	000106-99-0	3.96E+01	7.35E+02	2.11E+03	2.2E-07	1.2E+09	na	na	na	1.2E+09
Cadmium (Diet)	007440-43-9	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III) (Soluble Particulates)	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(III), Insoluble Salts	016065-83-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chromium(VI)	018540-29-9	-	1.69E+06	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Chrysene	000218-01-9	1.81E+05	2.00E-03	6.23E-09	9.2E+02	2.8E-01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	6.9E-01
Cobalt	007440-48-4	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Copper	007440-50-8	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Dibenz[a,h]anthracene	000053-70-3	1.91E+06	2.49E-03	9.55E-10	7.9E+04	3.2E-03	570	OEHHA 2000	4.4E-01	4.5E-01
Epichlorohydrin	000106-89-8	9.91E+00	6.59E+04	1.64E+01	6.3E-04	4.0E+05	2.8E+01	Environment Canada 2000	9.0E+00	4.0E+05
Fluoranthene	000206-44-0	5.55E+04	2.60E-01	9.22E-06	2.5E+01	1.0E+01	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.1E+01
Fluorene	000086-73-7	9.16E+03	1.69E+00	6.00E-04	4.1E-01	6.2E+02	103.5	CCME 2008 based on Loehr and Webster 1997	2.4E+00	6.2E+02
Hexane, N-	000110-54-3	1.32E+02	9.50E+00	1.51E+02	1.3E-07	1.9E+09	na	na	na	1.9E+09
Indeno[1,2,3-cd]pyrene	000193-39-5	3.47E+06	2.20E-05	1.25E-10	9.6E+03	2.6E-02	570	OEHHA 2000	4.4E-01	4.7E-01
Iron	007439-89-6	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lead and Compounds	007439-92-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Lithium	007439-93-2	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Manganese (Diet)	007439-96-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Mercury (elemental)	007439-97-6	-	6.00E-02	1.96E-03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Methylnaphthalene, 2-	000091-57-6	2.48E+03	2.46E+01	5.50E-02	1.8E-02	1.4E+04	32.5	CCME 2008 based on Loehr and Webster 1997	7.8E+00	1.4E+04
Molybdenum	007439-98-7	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Naphthalene	000091-20-3	1.54E+03	3.10E+01	8.50E-02	8.9E-03	2.9E+04	2.1	Enviornment Canada 1996	1.2E+02	2.9E+04
Nickel Oxide	001313-99-1	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Refinery Dust	000000-00-7	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Nickel Soluble Salts	007440-02-0	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Phenanthrene	000085-01-8	1.67E+04	1.15E+00	1.21E-04	2.5E+00	1.0E+02	5.7	CCME 2008 based on CEPA 1993	4.4E+01	1.5E+02
Pyrene	000129-00-0	5.43E+04	1.35E-01	4.50E-06	2.6E+01	9.8E+00	604.5	CCME 2008 based on Howard et al 1991	4.2E-01	1.0E+01
Selenium	007782-49-2	-	-	9.12E+03		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Succinic Acid							1.0E+08	assumed	2.5E-06	2.5E-06
Strontium, Stable	007440-24-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Styrene	000100-42-5	4.46E+02	3.10E+02	6.40E+00	3.4E-04	7.4E+05	na	na	na	7.4E+05
Tin	007440-31-5	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Titanium	007440-32-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Uranium, Insoluble Compounds	007440-61-1	-	-	0.00E+00		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Vanadium	000000-06-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Zinc and Compounds	007440-66-6	-	-	-		na	1.0E+08	OEHHA 2000	2.5E-06	2.5E-06
Indium	007440-74-6	-				na	1.0E+08	OEHHA 2001	2.5E-06	2.5E-06
Lanthanum	007439-91-0	-				na	1.0E+08	OEHHA 2002	2.5E-06	2.5E-06
Hexachlorobenzene	000118-74-1	6.20E+03	6.20E-03	1.80E-05	6.00E+02	4.2E-01	1.0E+09	OEHHA 2003	2.5E-07	4.2E-01
PCBs	001336-36-3	7.81E+04	7.00E-01	8.63E-05		na	3.23E+03	OEHHA 2004	7.8E-02	7.8E-02
Sulphate	014808-79-8	-	1.00E+06	5.93E-05		na	1.0E+08	OEHHA 2005	2.5E-06	2.5E-06

Notes:

Values from RAIS (2014) unless otherwise noted

CCME. 2008. Canadian Council of Ministers of the Enviroment. Canada-Wide Standard (CWS) for Petroleum Hydrocarbons (PHC) in Soil. Scientifc Rationale Supporting Technical Document. CCME. 2008

OEHHA 2000. Office of Environmental Health Hazard Assessment. California EPA. Technical Support Document for Exposure Assessment and Stochastic Analysis. Air Toxics Hot Spots Program Risk Assessment Guidelines. Oakland. California. 2000

Environment Canada. 2000. Screening Assessment: oxirane, (chloromethyl)-,epichlorohydrin. Chemical Abstracts Service Registry Number 106-89-8. Environment Canada. Health Canada. November 2008

	Calculated based on first order decay rate: k=0.693/(t1/2 / 365)
assumed	Based on lack of data from above sources, 10^8 t 1/2 based on professional judgement
na	Not availble from sources idenfied above

Table II-4D Project Soil Particulate/Dust Concentations for Industrial Receptor **Scenario: Project**

Parameter	DL kg/m^3	Cs mg/kg	CF ug/mg	C_{dust} ug/m^3
Polycyclic Aromatic Hydrocarbons				
Carcinogenic PAHs				
Benzo[a]anthracene	7.6E-10	2.3E-03	1000	1.7E-09
Benzo[a]pyrene	7.6E-10	4.2E-03	1000	3.2E-09
Benzo[b]fluoranthene	7.6E-10	8.7E-04	1000	6.6E-10
Benzo[g,h,i]perylene	7.6E-10	1.4E-03	1000	1.0E-09
Benzo[k]fluoranthene	7.6E-10	3.5E-03	1000	2.7E-09
Chrysene	7.6E-10	4.9E-03	1000	3.7E-09
Dibenzo[a,h]anthracene	7.6E-10	1.1E-03	1000	8.0E-10
Fluoranthene	7.6E-10	1.5E-03	1000	1.1E-09
Indeno[1,2,3-c,d]pyrene	7.6E-10	3.5E-03	1000	2.7E-09
Phenanthrene	7.6E-10	2.6E-05	1000	2.0E-11
Non-carcinogenic PAHs				
Acenaphthene	7.6E-10	2.0E-06	1000	1.5E-12
Acenaphthylene	7.6E-10	7.0E-07	1000	5.3E-13
Anthracene	7.6E-10	1.3E-05	1000	9.6E-12
Fluorene	7.6E-10	2.7E-06	1000	2.0E-12
Fluoranthene	7.6E-10	1.5E-03	1000	1.1E-09
Naphthalene	7.6E-10	2.5E-08	1000	1.9E-14
2-Methylnaphthalene	7.6E-10	4.4E-08	1000	3.4E-14
Pyrene	7.6E-10	1.9E-03	1000	1.5E-09
Metals and Metalloids				
Antimony	7.6E-10	9.9E-03	1000	7.5E-09
Arsenic	7.6E-10	1.3E-01	1000	9.6E-08
Barium	7.6E-10	3.4E+01	1000	2.6E-05
Beryllium	7.6E-10	2.0E-02	1000	1.5E-08
Cadmium	7.6E-10	1.1E-02	1000	8.1E-09
Chromium (III)	7.6E-10	9.6E-03	1000	7.3E-09
Chromium (VI)	7.6E-10	2.9E-01	1000	2.2E-07
Cobalt	7.6E-10	1.2E-01	1000	9.2E-08
Copper	7.6E-10	1.2E+00	1000	9.4E-07
Lead	7.6E-10	2.1E-01	1000	1.6E-07
Lithium	7.6E-10	4.8E-01	1000	3.6E-07
Manganese	7.6E-10	1.8E+00	1000	1.4E-06
Mercury	7.6E-10	7.5E-03	1000	5.7E-09
Molybdenum	7.6E-10	4.8E-02	1000	3.7E-08
Nickel	7.6E-10	3.8E-01	1000	2.9E-07
Selenium	7.6E-10	6.2E-02	1000	4.7E-08
Strontium	7.6E-10	2.1E+01	1000	1.6E-05
Tin	7.6E-10	2.2E-02	1000	1.7E-08
Uranium	7.6E-10	3.4E-02	1000	2.6E-08
Vanadium	7.6E-10	1.0E+00	1000	7.7E-07
Zinc	7.6E-10	1.3E+00	1000	9.9E-07
Aluminum	7.6E-10	2.2E+02	1000	1.6E-04
Boron	7.6E-10	1.7E+00	1000	1.3E-06
Iron	7.6E-10	2.4E+02	1000	1.8E-04
Titanium	7.6E-10	1.5E+01	1000	1.1E-05
Indium	7.6E-10	2.1E-04	1000	1.6E-10
Lanthanum	7.6E-10	5.2E-04	1000	4.0E-10
Others				
PCBs	7.6E-10	3.2E-07	1000	2.4E-13
Sulphate	7.6E-10	6.6E-02	1000	5.0E-08
Hexachlorobenzene (gas phase)	7.6E-10	8.6E-09	1000	6.5E-15
Dust Pallatives				
Epichlorohydrin	7.6E-10	6.0E-10	1000	4.5E-16

Notes:
Equations based on US EPA OWS (2005)
[1] Soil concentration from Equation 1.2

APPENDIX III

Worked Example: Exposure Modeling and Risk Estimates

WORKED EXAMPLE CALCULATIONS

PARAMETER: EPICHLOROHYDIN

RECEPTOR: Maximum North Delta Residential Receptor; SCENARIO: PROJECT

Equation 1.1: Deposition to Soil

$$Ds = \frac{Dtot}{Zs \text{ x } BD}$$

D_s = chemical-specific deposition (mg/kg/yr)

D_{tot} = chemical-specific deposition rate (4.6E-02 mg/m^2/yr) (conservatively assuming 100% of dustfall, note this is clearly an overestimate)

Z_s = soil mixing depth (0.02 m) (US EPA OSW, 2005)

B_D = soil bulk density (1,500 kg/m^3) (US EPA OSW, 2005)

Worked Example:

$$Ds = \frac{4.6E-02}{0.02 \times 1500} = 1.5E-03 \text{ mg/kg/yr}$$

Equation 1.2: Soil Concentrations

$$Cs = \frac{Ds \times [1 - \exp(-kt \times tD)]}{kt}$$

C_s = average soil concentration over exposure (mg/kg)

D_s = deposition to soil (1.5E-03 mg chemical/kg soil/yr)

K_t = chemical soil loss due to degradation or volatilization 4.0E+05 yrs^{-1}

t_D = time period over which deposition occurs (10 yrs)

Worked Example:

$$Cs = \frac{1.5E\text{-}03 \times [1-\exp(-4.0E+05 \times 10)]}{4.0E+05}$$

Cs = 3.8E-09 mg/kg

Equation 1.2.1: Total Soil Degradation Rate

$$kt = kv + ks$$

kt = chemical soil loss due to degradation and volatilization (yrs^{-1})

kv = chemical loss due to volatilization (yrs^{-1})

ks = chemical loss due to abiotic and biotic biodegradation (yrs^{-1})

Loss from Volatilization:

Chemical loss from volatilization was predicted as follows:

$$t\,{}^{1}/_{2} = 1.58E-08 \times \left(\frac{Koc \times S}{VP}\right)$$

The half-life is converted to a rate constant (yrs-1) using the following equation:

$$k = 0.693/\left(\frac{t½}{365}\right)$$

Koc = organic carbon portioning coefficient (9.9E+00 L/kg) (ORNL, 2014)

S = water solubility (6.6E+04 mg/L) (ORNL, 2014)

VP = vapour pressure (1.6E+01 mm Hg) (ORNL, 2014)

t ½ =soil half-life due to biodegradation 28 days (Health Canada/Environment Canada, 2008)

Worked Example:

$$1.58-08 \times \left(\frac{9.91 \times 6.59\text{E}+04}{1.64\text{E}+01}\right) = 6.29\text{E}-04$$

kv = 0.693 / ((6.29E-04)/365) = 4.0E+05 yrs^{-1}

ks = 0.693 / (28/365) = 9.0 yrs^{-1}

kt = 4.0E+05 yrs^{-1} + 9.0 yrs^{-1}

kt= 4.0E+05 yrs^{-1}

Equation 2.0: Chemical Concentration in Soil Dust

$$C_{dust} = DL \times Cs \times CF$$

C_{dust} = chemical concentration in soil generated dust (µg/m^3)

DL = dust level (Health Canada 2010) (7.60E-10 kg/m^3)

C_s = surface soil concentration from deposition over time (3.8E-09 mg/kg)

CF = conversion factor (mg to µg) (1,000 µg/mg)

Worked Example:

C_{dust} = 7.6E-10 × 3.8E-09 × 1,000
C_{dust} = 2.9E-15 µg/m^3

Equation 3.1: Plant Concentration due to Direct Deposition

$$\text{Pd} = \frac{[\text{Dd} + (\text{Dw} \times 0.6)] \times \text{Rp} \times [1.0 - \exp(-\text{kp} \times \text{Tp})]}{\text{Yp} \times \text{kp}}$$

P_d = forage concentration due to deposition mg/kg DW

D_d = dry deposition particle fraction (1.2E-05 mg/m^2/yr - based on the deposition rate for coal provided in Levelton (2014) and the percent composition epichlorohydrin of 2.5E-06, estimated from the manufacturers application rate for the dust suppressant)

D_w = wet deposition particle fraction (1.3E-05 mg/m^2/yr - based on deposition rate for coal provided in Levelton (2014) and the percent composition epichlorohydrin of 2.5E-06)

R_p = intercept fraction of edible portions of plant (0.5) (conservative US EPA OSW 2005) (See below discussion regarding the selection of the R_p value)

k_p = plant surface loss coefficient (18 yrs^{-1}) (US EPA OWS 2005, corresponding to 14 day 1/2 life)

t_p = length of plant exposure to deposition per harvest (0.164 yrs) (US EPA OWS 2005)

Y_p = yield/productivity (2.24 kg DW/m^2) (US EPA OSW 2005)

Worked Example:

$$Pd = \frac{[1.2E\text{-}05\ mg/m^2/yr + (1.3E\text{-}05\ mg/m^2/yr \times 0.6)] \times 0.5 \times [1.0 - \exp(-18 yrs^{-1} \times 0.164 yrs)}{2.24\ kg/m^2 \times 18\ yrs^{-1}}$$

$$Pd = \frac{9.4E\text{-}06}{40.3}$$

Pd = 2.3E-07 mg/kg DW

Selection of R_p value:

The US EPA OSW (2005) recommends an R_p value of 0.05 for fruits, vegetables and legumes; however, the later sections of the document detail the limitations/uncertainty in predicting vegetable COPC concentrations arising from the R_p parameter. The US EPA OSW (2005) document also recommends a more conservative R_p value of 0.5 based on Baes et al. (1984) as indicated below:

Expert from Section 5.4.1 of US EPA OSW (2005):

- ***Recommended Value for: Interception Fraction of the Edible Portion of Plant (Rp) Forage = 0.5***

Given the US EPA identified uncertainties in the R_p value, and as an effort was made to protect for a wide variety of produce that may be grown within the Study Area, the more conservative R_p value was used in the HHRA. As discussed in Appendix B of US EPA OSW (2005), when determining COPC concentrations in above ground produce, the uncertainties associated with R_p are introduced "*when the calculated parameter values don't accurately represent aboveground produce-specific values"* while "*uncertainties associated with Y_p are not expected to be significant"*. The use of the US EPA OSW (2005) R_p of 0.5 is considered to be conservative and is considered to address uncertainly associated with variable food type.

Equation 3.2.1: Soil to Plant Bioconcentration Factor

$$\log BCF = 1.588 - 0.578 \times \log K_{ow}$$

BCF = plant-soil bioconcentration factor (kg soil/kg plant DW)
log K_{ow} = log of octanol-water partitioning coefficient (0.45 unitless) (ORNL, 2014)

Worked Example:

$$\log BCF = 1.588 - 0.578 \times 0.45$$
$$\log BCF = 1.3279$$
$$BCF = 21.3$$

Equation 3.2.2: Plant Concentration in Aboveground Forage due to Root Uptake

$$Pr = Cs \times BCF$$

Pr = chemical concentration in above ground plant as a result of root uptake (mg/kg DW)

Cs = chemical concentration in soil (3.8E-09 mg/kg)

BCF = plant-soil bioconcentration factor (21.3 kg soil/kg plant DW)

Worked Example:

$$Pr = 3.8E\text{-}09 \times 21.3$$

$$Pr = 8.1E\text{-}08 \text{ mg/kg}$$

Equation 3.3: Total Chemical Concentration in Plants

$$C_{plant} = (Pd+Pr) \times (1-WC)$$

C_{plant} = total chemical concentration in aboveground plant (mg/kg WW)

Pd = plant concentration due to direct deposition (2.3E-07 mg/kg DW)

Pr = chemical concentration due in above ground forage due to root uptake (8.1E-08 mg/kg DW)

WC = water content (DW to WW conversion) 0.85 unitless

Worked Example:

$$C_{plant} = (2.3E-07 + 8.1E-08) \times (1-0.85)$$

$$C_{plant} = 4.7E-08 \text{ mg/kg WW}$$

Equation 3.3: Belowground Plant Concentration

$$Pr_{root}=Cs \times BCF \times WPF \times (1-WC)$$

Pr_{root} = chemical concentration in belowground produce due to root uptake (mg/kg WW)

Cs = chemical concentration in soil (3.8E-09 mg/kg)

BCF = plant-soil bioconcentration factor for aboveground produce (21.3 kg soil/kg plant DW)

WPF = washing/peeling factor (no washing/peeling assumed)

WC = moisture content of root vegetable (85%)

Worked Example:

$$Pr_{root} = 3.8E-09 \times 21.3 \times 1 \times (1-0.85)$$

$$Pr_{root} = 1.2E-08 \text{ mg/kg WW}$$

Equation 4.0: Air Concentration

$$C_{air} = TPM \times PC$$

C_{air} = concentration of COPC in air (µg/m^3)

TPM = total particulate matter (0.498 µg/m^3) (Levelton, 2014, maximum annual coal concentration at maximum North Delta residential receptor)

PC = percent composition (2.5E-06) (based on manufacturers application rate for dust suppressant DC9148)

Worked Example:

$$C_{air} = 0.498 \times 2.5E\text{-}06$$

$$C_{air} = 1.3E\text{-}06 \text{ µg/m}^3$$

WORKED EXAMPLES OF RISK CALCULATIONS

The following worked examples of the risk calculations illustrate the procedures used to estimate risks for the following scenarios:

Food Exposures:

- A toddler consuming 67 grams per day of above ground vegetation of 4.7E-08 µg/g (wet weight) epichlorohydin and 105 grams per day of below ground vegetation containing 1.2E-08 µg/g (wet weight) epichlorohydin; and,
- An adult consuming 137 grams per day of above ground vegetation of 4.7E-08 µg/g (wet weight) epichlorohydin and 188 grams per day of below ground vegetation containing 1.2E-08 µg/g (wet weight) epichlorohydin.

Exposures to Air and Soil:

- A toddler and an adult inhaling 1.3E-06 µg/m^3 epichlorohydin;
- A toddler and an adult in direct contact (dermal contact, incidental ingestion) with 3.8E-09 µg/g epichlorohydin in soil; and

A toddler and adult in direct contact (particulate inhalation) with 2.9E-15 µg/m^3 epichlorohydin in suspended dust arising from soil.

FOOD EXPOSURES

Non-Cancer Risks to Toddler from Epichlorohydin in Vegetation

In order to estimate exposure from ingestion, the following equation was applied:

$$EIF = \frac{CF \times SS \times BAIG \times NSSW \times NWSY \times AAF \times LAF}{BW \times NDW \times NWY}$$

where:

EIF = exposure from the food ingestion pathway (µg/kg body weight/day)

CF = chemical concentration in food source (above ground 4.7E-08 µg/g)

SS = serving size of vegetation for person (toddler: 67 g/serving)

BAIG = bioavailable fraction via the ingestion route (assumed to be 1.0)

NSSW = number of servings per week that food (7 serving/week)

NWSY= number of weeks vegetation is consumed per year (assumed to be 52 weeks/year)

NWY = number of weeks in a year (52 weeks/year)

AAF = annual amortization factor (assumed to be 1)

LAF = lifetime amortization factor (assumed to be 1 for non-carcinogens)

BW = body weight of person (kg) (toddler: 16.5 kg)

NDW = number of days in a week (7 days per week)

$$EIF = \frac{4.7E\text{-}08\ \mu g/g \times 67\ g/serving \times 1.0 \times 7\ serving/wk \times 52\ wk/yr \times 1 \times 1}{16.5\ kg \times 7\ day/wk \times 52\ wk/yr}$$

= 1.9E-07 μg/kg body weight/day

Exposure to epichlorohydin from ingestion of above ground vegetation once per day was estimated to be 1.9E-07 μg/kg body weight/day for the toddler receptor.

When the US EPA IRIS (2014) RfD of 6.0 μg/kg bw/day is used, risks are estimated as Hazard Quotient (HQ) value according to the following formula:

$$HQ = \frac{\text{Estimated Exposure (μg/kg body weight/day)}}{\text{Tolerable Daily Intake (μg/kg body weight/day)}}$$

where:

Exposure to epichlorohydin = 1.9E-07 μg/kg body weight/day

RfD for epichlorohydin = 6.0 μg/kg body weight/day

$$HQ = \frac{1.9E\text{-}07}{6.0}$$

$$HQ = 3.1E\text{-}08$$

Thus, a HQ value of 3.1E-08 was estimated for toddlers consuming above ground vegetation with an epichlorohydin concentration of 4.7E-08 μg/g at a rate of 67 gram serving per day over their entire life stage as a toddler when the RfD of 6.0 μg/kg bw/day was assumed.

Similarly, a HQ of 1.3E-08 was estimated for toddlers consuming below ground vegetation with an epichlorohydin concentration of 1.2E-08 μg/g at a rate of 105 gram serving per day over their entire life stage as a toddler when the RfD of 6.0 μg/kg bw/day was assumed.

Cancer Risks to Adults from Epichlorohydin in Vegetation

The following example calculations illustrates the procedures used to estimate risks for an adult consuming 137 grams per day of above ground vegetation with epichlorohydin concentrations of 4.7E-08 μg/g (wet weight). Although atmospheric deposition was assumed to occur over the Project lifetime (10 years), it was assumed that vegetation grown for consumption would continue to have COPCs deposited on it from COPCs in surface soils and that therefore, the exposure would continue to occur over a lifetime (i.e., it was conservatively assumed that the average soil and produce concentrations would remain constant after deposition had ceased). This is conservative as soil and produce concentrations would decrease following the Project, however, in an effort to not

underestimate potential risks to human health, this conservative approach was undertaken when determining risks due to food consumption exposures.

In order to estimate exposure from ingestion of vegetation, the following equation was applied:

$$EIF = \frac{CF \times SS \times BAIG \times NSSW \times NWSY \times AAF \times LAF}{BW \times NDW \times NWY}$$

where:

EIF = exposure from the food ingestion pathway (μg/kg body weight/day)

CF = chemical concentration in vegetation source (above ground 4.7E-08 μg/g)

SS = serving size of vegetation for person (g/meal) (137 g/meal)

BAIG = bioavailable fraction via the ingestion route (assumed to be 1.0)

NSSW = number of servings of vegetation consumed per week (assumed to be 7 days per week)

NWSY = number of weeks vegetation consumed per year (assumed to be 52 weeks/year)

NWY = number of weeks in a year (52 weeks/year)

AAF = annual amortization factor (assumed to be 1)

LAF = lifetime amortization factor (assumed to be 1 for carcinogens)

BW = body weight of person (kg) (adult receptor: 70.7 kg)

NDW= number of days in a week (7 days per week)

$$EIF = \frac{4.7E\text{-}08\ \mu g/g \times 137\ g/meal \times 1.0 \times 7\ days/wk \times 52\ wk/yr \times 1.0 \times 1.0}{70.7\ kg \times 7\ day/wk \times 52\ wk/yr}$$

$$= 9.1E\text{-}08\ \mu g/kg\ body\ weight/day$$

Thus, for this example, exposures to epichlorohydin from ingestion of above ground vegetation of 137 grams per day would be estimated to be 9.1E-08 μg/kg body weight/day for the adult when vegetation concentrations of epichlorohydin. Once again, it is noted that persons were assumed to spend their entire lives in the vicinity of Project such that no amortization of cancer risks for less than lifetime exposures was considered.

Risks were then estimated as ILCR estimates according to the following formula:

ILCR = Estimated Daily Exposure (μg/kg/day) x Cancer Potency Factor $(\mu g/kg/day)^{-1}$

where:

Exposure to epichlorohydin = 9.1E-08 μg/kg body weight/day

Potency factor for epichlorohydin = 9.9 E-06 per μg/kg body weight/day

Using the US EPA IRIS (2014) oral potency factor, an ILCR estimate of 9.0-13 was estimated for adults consuming vegetation with epichlorohydin concentrations of 4.7E-08 μg/g at a rate of 137 grams per day over an entire lifetime.

Similarly, an ILCR of 3.2E-13 was estimated for adults consuming below ground vegetation with an epichlorohydin concentration of 2.1E-06 μg/g at a rate of 188 gram serving per day over their entire lifetime.

ENVIRONMENTAL EXPOSURES

Non-cancer Risk due to Inhalation of Epichlorohydin

Epichlorohydin was estimated to be present at the nearest residential receptor at a concentration of 1.3E-06 $\mu g/m^3$ conservatively assuming that epichlorohydin formed the entirety of the dust suppressant applied to coal.

When the US EPA IRIS (2014) RfC of 1 $\mu g/m^3$ was assumed, risks were then estimated as Hazard Quotient (HQ) values according to the following formula:

$$HQ = \frac{\text{Estimated Exposure } (\mu g/m^3)}{\text{Tolerable Concentration } (\mu g/m^3)}$$

where:

Exposure to epichlorohydin = $1.3 \times 10^{-6}\ \mu g/m^3$

RfC for epichlorohydin = $1.0\ \mu g/m^3$

$$HQ = \frac{1.3 \times 10^{-6}\ \mu g/m^3}{1.0\ \mu g/m^3}$$

$$= 1.3E\text{-}06$$

Thus, a HQ value of 1.3E-06 was estimated for residents inhaling epichlorohydin at concentrations of 1.3×10^{-6} μg/m^3 at a rate of 24 hours per day when the RfC 1.0 μg/m^3 was assumed.

Similarly, a HQ value of 2.9E-15 was estimated for residents inhaling epichlorohydin dust (arising from soil) concentrations of 2.9×10^{-15} μg/m^3 for 24 hours per day when the RfC of 1.0 μg/m^3 was assumed.

Cancer Risk due to Inhalation of Epichlorohydin

Epichlorohydin was estimated to be present at the nearest residential receptor at a concentration of 1.3E-06 μg/m^3 conservatively assuming that epichlorohydin formed the entirety of the dust suppressant. Based on a Project lifetime 10 years, the inhalation exposures were amortized, 10 years over an 80 year lifetime.

Risks were then estimated as ILCR estimates according to the following formula:

ILCR = Estimated Daily Exposure (μg/m^3) x Cancer Potency Factor (μg/m^3)$^{-1}$

where:

Amortized epichlorohydin air concentration = 1.3×10^{-6} μg/m^3 *10 years/80 years

= 1.6×10^{-7} μg/m^3

Potency factor for epichlorohydin = 1.2×10^{-6} per μg/m^3

ILCR = 1.6×10^{-7} μg/m^3 x 1.2×10^{-6} per μg/m^3

ILCR = 1.9E-13

When the US EPA IRIS (2014) potency factor is assumed, an ILCR estimate of 1.9E-13 was estimated for adults inhaling with epichlorohydin concentrations of 1.3×10^{-6} μg/m^3 for the duration of the Project (10 years).

Similarly, an ILCR estimate of 3.5E-21 was estimated for adults inhaling with epichlorohydin in dust arising from soil at concentrations of 2.9×10^{-15} μg/m^3 over a lifetime. Similar to produce consumption, no amortization was assumed for soil exposures.

Non-cancer Risk due to Direct Contact with Soil Containing of Epichlorohydin

Epichlorohydin was estimated to be present at the nearest residential receptor at a concentration of 3.8E-09 µg/g in soil, conservatively assuming that epichlorohydin formed the entirety of the dust suppressant.

Exposure following incidental ingestion of soil was estimated as per the following Health Canada (2012) equation:

$$EIG = \frac{C_S \times IR_S \times RAF_{Oral} \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EIG = exposure from the soil ingestion pathway (µg/kg body weight/day)

C_S = soil chemical concentration (3.8E-09 µg/g)

IR_S = soil ingestion rate of person (0.08 g/day)

RAF_{Oral} = relative bioavailability fraction via the ingestion route (100%)

D_2 = days per week exposed (7 d/7 d)

D_3 = weeks exposed per year (52/52 weeks, no amortization assumed)

D_4 = years exposed (carcinogens only)

BW = body weight of person (16.5 kg)

LE = life expectancy (years, carcinogens only)

Exposure to epichlorohydin from the soil ingestion route was estimated to be 1.8E-11 µg/kg body weight/day. Soil ingestion risks were then estimated as Hazard Quotient (HQ) values according to the following formula:

$$\text{Hazard Quotient} = \frac{\text{Estimated Exposure (1.8E-11 µg/kg/day)}}{\text{Toxicity Reference Values (TRV) (6.0 µg/kg/day)}}$$

A Hazard Quotient of 3.1E-12 was estimated for a toddler exposed via incidental ingestion to concentrations of epichlorohydin in soil.

Dermal exposure to soil was estimated according to the following Health Canada (2012) equation:

$$EDS = [(C_s \times SA_H \times SL_H) + (C_s \times SA_O \times SL_O)] \times RAF_{Derm} \times D_2 \times D_3 \times D_4$$

BW x LE

Where:

EDS = exposure from the dermal pathway for soils (µg/kg/day)

C_S = soil chemical concentration (3.8E-09 µg/g)

SA_H = surface area of hands exposed for soil loading (0.043 m^2)

SA_O = surface area exposed other than hands (0.258 m^2)

SL_H = soil loading rate to exposed skin of hands (1 g/m^2/event)

SL_O = soil loading rate to exposed skin other than hands (0.1 g/m^2/event)

RAF_{Dermal} = relative bioavailability fraction via the dermal route (1.0 assumed)

D_2 = days per week exposed (7 d/7 d)

D_3 = weeks exposed per year (52/52 weeks, no amortization assumed)

D_4 = years exposed (carcinogens only)

BW = body weight of person (16.5 kg)

LE = life expectancy (years, carcinogens only)

Exposure to epichlorohydin from dermal contact with soil was estimated to be 1.6E-11 µg/kg body weight/day. Soil dermal contact risks were then estimated as HQ values according to the following formula:

$$\text{Hazard Quotient} = \frac{\text{Estimated Exposure (1.6E-11 µg/kg/day)}}{\text{TRV (6.0 µg/kg/day) x Study Bioavailability (1.0)}}$$

A HQ of 2.6E-12 was estimated for a toddler dermally exposed to concentrations of epichlorohydin in soil.

Cancer Risk due to Direct Contact with Soil Containing of Epichlorohydin

Epichlorohydin was estimated to be present at the nearest residential receptor at a concentration of 3.8E-09 µg/g in soil, conservatively assuming that epichlorohydin formed the entirety of the dust suppressant.

Exposure following incidental ingestion of soil was estimated as per the following Health Canada (2012) equation:

$$EIG = \frac{C_S \times IR_S \times RAF_{Oral} \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EIG = exposure from the soil ingestion pathway (µg/kg body weight/day)

C_S = soil chemical concentration (3.8E-09 µg/g)

IR_S = soil ingestion rate of person (0.02 g/day)

RAF_{Oral} = relative bioavailability fraction via the ingestion route (100%)

D_2 = days per week exposed (7 d/7 d)

D_3 = weeks exposed per year (52/52 weeks, no amortization assumed)

D_4 = years exposed (80 years, carcinogens only)

BW = body weight of person (70.7 kg)

LE = life expectancy (80 years, carcinogens only)

Carcinogenic exposure to epichlorohydin from the soil ingestion route was estimated to be 1.1E-12 µg/kg body weight/day. Risks were then estimated as ILCR estimates according to the following formula:

$$ILCR = \text{Estimated Daily Exposure } (\mu g/kg/day) \times \text{Cancer Potency Factor } (\mu g/kg/day)^{-1}$$

where:

Exposure to epichlorohydin = 1.1×10^{-12} µg/kg/day

Potency factor for epichlorohydin = 9.9×10^{-6} per µg/kg/day

Using the US EPA IRIS (2014) potency factor, an ILCR estimate of 1.1E-17 was estimated for adults incidentally ingesting soil with epichlorohydin concentrations of 3.8E-09 µg/g over an entire lifetime. Once again, it is noted that persons were assumed to spend their entire lives in the vicinity of Project facility and that no degradation of soil concentrations would occur overtime. As such, no amortization of cancer risks for less than lifetime exposures was considered.

Dermal exposure to soil was estimated according to the following Health Canada (2012) equation:

$$EDS = \frac{[(C_s \times SA_H \times SL_H) + (C_s \times SA_O \times SL_O)] \times RAF_{Derm} \times D_2 \times D_3 \times D_4}{BW \times LE}$$

Where:

EDS = exposure from the dermal pathway for soils (µg/kg/day)

C_S = soil chemical concentration (3.8E-09 µg/g)

SA_H = surface area of hands exposed for soil loading (0.089 m^2)

SA_O = surface area exposed other than hands (0.822 m^2)

SL_H = soil loading rate to exposed skin of hands (1 g/m^2/event)

SL_O = soil loading rate to exposed skin other than hands (0.1 g/m^2/event)

RAF_{Dermal} = relative bioavailability fraction via the dermal route (1.0 assumed)

D_2 = days per week exposed (7 d/7 d)

D_3 = weeks exposed per year (52/52 weeks, no amortization assumed)

D_4 = years exposed (80 years)

BW = body weight of person (70.7 kg)

LE = life expectancy (80 years)

Carcinogenic exposures to epichlorohydin from dermal contact with soil were estimated to be 9.2E-12 µg/kg body weight/day. Risks were then estimated as ILCR estimates according to the following formula:

ILCR = Estimated Daily Exposure (µg/kg/day) x Cancer Potency Factor $(\mu g/kg/day)^{-1}$

where:

Exposure to epichlorohydin = 9.1 E-12 µg/kg/day

Potency factor for epichlorohydin = 9.9×10^{-6} per μg/kg/day

Using the US EPA IRIS (2014) potency factor, an ILCR estimate of 9.1E-17 was estimated for adults in daily dermal contact with soil with epichlorohydin concentrations of 3.8E-09 µg/g over an entire lifetime.

Overall Non-cancer Risk due to Exposure with Media containing Epichlorohydin

Overall non-cancer risks due to exposure to epichlorohydin at the maximum North Delta residential receptor were determined by summing risk estimates from all operable pathways. The following assumes that above and below ground vegetation are consumed on a daily basis, soil dermal and incidental ingestion exposures occur on a daily basis and that inhalation of particulate in air and re-suspended dust occur on over a 24 hour period each day. Therefore, as commercial produce is anticipated to supplement home gardens and a portion of time is anticipated to occur away from the project facility, the predicted risks are conservative.

HQ above-ground produce = 3.2E-08

HQ below ground produce = 1.3E-08

HQ air inhalation = 1.3E-06

HQ dust inhalation = 2.9E-15

HQ soil ingestion = 3.1E-12

HQ soil dermal contact = 2.6E-12

Overall HQ = 1.3E-06

Overall Cancer Risk due to Exposure with Media containing Epichlorohydin

Overall cancer risks due to exposure with above and below ground produce, soil, particulate and re-suspended dust containing epichlorohydin concentrations at the nearest residential receptor were determined by summing incremental lifetime cancer risks from all operable pathways. It is noted that persons were assumed to spend their entire lives in the vicinity of project facility such that no amortization of cancer risks for less than lifetime exposures was considered for contact with soil or produce consumption following project. It was also assumed that all produce was supplied by a home garden and residents were present in the maximum emission locations for a period of 24 hours per day (i.e., all time was spent at home). Therefore, the following is conservative.

ILCR above-ground produce = 9.0E-13

ILCR below ground produce = 3.2E-13

ILCR air inhalation = 1.9E-13

ILCR dust inhalation = 3.5E-21

ILCR soil ingestion = 1.1E-17

ILCR soil dermal contact = 9.1E-17

Total ILCR = 1.4E-12

APPENDIX IV

SNC·LAVALIN

Regional Health Status

APPENDIX IV: REGIONAL HEALTH STATUS

IV.1. BACKGROUND

The following appendix provides a discussion on the current human health in the region. The summary focuses on $PM_{2.5}$. The following information is intended to provide a reference (or baseline) to evaluate if the Project Facility could impact human health. The information in the following studies was not used to adjust the quantitative risk estimates presented in the HHRA, but rather to evaluate the current health status in the area with respect to $PM_{2.5}$. It is stressed that the following information is provided for the sole purpose of establishing a baseline in the region and is not intended to be a comprehensive review of the pertinent literature.

Health based air quality reports by health/regulatory agencies related to $PM_{2.5}$ air quality were reviewed. The following reports were relied upon:

- *Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide, Global Updates.* 2005. Summary of Risk Assessment. World Health Organization. Geneva, Switzerland. 2006.
- *Development of Options for a New Provincial $PM_{2.5}$ Air Quality Objective.* SENES Consultants and Bates, D. V. Prepared for BC Lung Association[1]. 2005.
- *Canada Wide Standards for Particulate Matter (PM) and Ozone.* Canadian Council of Ministers of the Environment (CCME). 2000.

Studies evaluating ambient $PM_{2.5}$ pollution and health response within the Greater Vancouver area were also reviewed with preference given to reviewing recent studies (≤ 10 years old). The following documents were relied upon:

- *Associations of Ambient Air Pollution with Chronic Obstructive Pulmonary Disease Hospitalization and Mortality*. Gan, W, Q., Fitzgerald, M., Carlesten, C., Sadatsafavi, M., Brauer, M. American Journal of Respiratory Critical Care Medicine. Vol. 187. Iss. 7. pp 721-727. April 1, 2013.
- *Risk of Nonaccidental and* Cardiovascular *Mortality in Relation to Long-term Exposure to Low Concentrations of Fine Particulate Matter: A Canadian National-Level Cohort Study*. Crouse, D. L., Peters, P. A., van Donkelaar, A., Goldberg, M. S., Vileneuve, P. J., Brion, O., Khan, S., Atari, D. O.,

[1] Although this report was not prepared by the BC Lung Association, the recommendations presented in the report were accepted by the Province for establishing Provincial $PM_{2.5}$ Air Quality Objectives, and was therefore included in the review.

Jerrett, M., Pope, C. A., Brauer, M., Brook, J. R., Martin, R. V., Steib, D., Burnett, R. T. Environmental Health Perspectives. Vol. 120. Iss. 5. pp. 708-714. 2012.

- *Association between Air Pollution and Multiple Respiratory Hospitalizations among the Elderly in Vancouver*, Canada. Fung, K. Y., Khan, S., Krewski, D., Chen, Y. Inhalation Toxicology. Vol. 18. pp. 1005-1011. 2006.
- *Association between Particulate Air Pollution and First Hospital Admission for Childhood Respiratory Illness in Vancouver, Canada.* Yang, Q., Chen, Y., Krewski, D., Shi, Y., Burnett, R. T., McGrail, K. M. Archives of Environmental Health: An International Journal. Vol. 59. Iss. 1. pp 14-21. 2004.
- *Influence of Relatively Low Level of Particulate Air Pollution on Hospitalization for COPD in Elderly People.* Chen, Y., Yang, Q., Krewski, D., Shi, Y., Burnett, R. T., McGrail, K. Inhalation Toxicology, Vol. 16. pp. 21-25. 2004.
- *A Time-Series Study of Air Pollution, Socioeconomic Status and Mortality in Vancouver, Canada.* Villeunue, P. J., Burnett, R. T., Shi, Y., Krewski, D., Goldberg, M.S., Hertzman, C., Chen, Y., Brook, J. Journal of Exposure Analysis and Environmental Epidemiology. Vol. 13. pp 427-435. 2003.
- *Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling.* Churg, A., Brauer, M., del Carmen Avila-Casado, M., Fortoul, T. L., Wright, J. L. Environmental Health Perspectives. Vol. 111 Iss. 5. pp 714-718. 2003.

IV.2. HEALTH STATUS IN THE REGION

The following provides a summary of studies referenced above with a focus on $PM_{2.5}$ and observed health effects within the Greater Vancouver region. It is stressed that this is not intended to be a comprehensive review of the pertinent literature, but rather is provided to establish baseline health data with respect to $PM_{2.5}$ exposure in the region and address uncertainty regarding potential for health effects within the region due to the Project; however, the quantitative results of the HHRA were not adjusted based on these findings.

***Associations of Ambient Air Pollution with Chronic Obstructive Pulmonary Disease Hospitalization and Mortality.* Gan, W, Q., Fitzgerald, M., Carlesten, C., Sadatsafavi, M., Brauer, M. American Journal of Respiratory Critical Care Medicine. Vol. 187., Iss. 7., pp 721-727. April 1, 2013.**

Gan et al. (2013) investigated the association of ambient air pollution and chronic obstructive pulmonary disease (COPD), hospitalization and mortality for residents between the ages of 45 and 85 with no previous physician diagnosis of COPD residing within Metropolitan Vancouver during a 5 year period (1994 to 1998). The objective of the study was to investigate the effects of elevated traffic-related pollution within the region; however, as the study examined $PM_{2.5}$ concentrations with a large population cohort (within Metropolitan Vancouver) and baseline COPD data were provided by the authors of the study, the study was considered appropriate for investigating regional health status with respect to $PM_{2.5}$. The following summarized the findings of Gan et al. (2013):

The baseline characteristics identified approximately 465,000 subjects (59.4 ± 10.7 years old, mean ± std deviation) without prior COPD within the study area. Approximately 2,300 cases of hospitalization were identified with the average age of those hospitalized being higher than that of the cohort (69.9 ± 9.1 years old). Mortality (average age of 74.6 ± 6.8 years) was observed in 540 cases. The percentage of individuals with co-morbid conditions (asthma, diabetes, coronary heart disease, hypertensive heart disease) were higher in cases of hospitalization (31%) and mortality (32%) compared to the general cohort (10%). The lowest income neighborhoods accounted for the majority of cases of hospitalization and mortality.

Relative risks for COPD outcomes for each pollutant were determined using biovariable and multivariable models with multivariable models adjusted for age, sex, asthma, diabetes, coronary heart disease, hypertensive heart disease, neighborhood income quintiles and co-pollutants. While the study identified increased risk of COPD due to higher levels of black carbon and wood-smoke related pollution (with higher mortality risks also identified for increase black carbon levels), the study did not find significant associations of COPD with $PM_{2.5}$ in Metropolitan Vancouver (no significant risks were observed for NO_2 and NO as well); however, it was indicated that the lack of spatial variability (i.e., homogeneous $PM_{2.5}$ distribution) in the study area may account for the null associations observed. The authors stated that although not observed in their study, traffic related fine particulate air pollution has been associated with increased COPD risk in previous studies (specific studies were not provided).

Risk of Nonaccidental and Cardiovascular Mortality in Relation to Long-term Exposure to Low Concentrations of Fine Particulate Matter: A Canadian National-Level Cohort Study. **Crouse, D., L., Peters, P. A., van Donkelaar, A., Goldberg, M. S., Vileneuve, P. J., Brion, O., Khan, S., Atari, D. O., Jerrett, M., Pope, C. A., Brauer, M., Brook, J. R., Martin, R. V., Steib, D., Burnett, R. T. Environmental Health Perspectives. Vol. 120 Iss. 5. 2012.**

Crouse et al. (2012) conducted a national level cohort study examining the relationship between $PM_{2.5}$ concentrations and cardiovascular mortality. Although results were not specifically reported for Greater Vancouver but the Canadian population in general, the study was reviewed in the HHRA based on the significance of the large cohort and the findings. The following provides a summary of Crouse et al. (2012):

The study investigated the relationship between $PM_{2.5}$ concentrations measured in 11 Canadian cities (including Vancouver) and those predicted for rural communities (excluding the northern territories) and mortality for the non-immigrant population. The study found that cardiovascular mortality was positively associated with $PM_{2.5}$ exposures at concentrations (average $PM_{2.5}$ concentration of 8.7 µg/m³) lower than those previously reported in large cohort studies conducted elsewhere. The study found that ischemic heart disease was strongly correlated with long-term $PM_{2.5}$ exposure. It was concluded that the associations found in the study were similar in magnitude to those reported in other large cohort studies.

Association between Air Pollution and Multiple Respiratory Hospitalizations among the Elderly in Vancouver, Canada. **Fung, K., Y., Khan, S., Krewski, D., Chen, Y. Inhalation Toxicology. Vol. 18. pp. 1005-1011. 2006.**

Fung et al. (2006) assessed the impact of particulate pollutants (including $PM_{2.5}$) on repeated respiratory hospital admissions among the 65 year and older population in Greater Vancouver based on data collected from 1995 to 1999 using an analysis method established by others. An objective of the study was to examine the autocorrelation among repeated respiratory hospitalization and the increased likelihood of re-admission for the same condition, whereas the authors indicated that other studies may treat hospitalization events as being independent of one another. The findings of the Fung et al. (2006) study are as follows:

The study identified approximately 26,000 individuals admitted to the hospital due to respiratory conditions. For persons admitted, an average 1.78 admissions were observed with 72% of individuals having no re-admission. Average $PM_{2.5}$ concentrations for the 5 year study period were 7.7 µg/m³ with concentrations ranging from 2 µg/m³ to 32 µg/m³. The study found a correlation between hospital admissions for individuals > 65 years old and air pollutants. While relative risks for elderly respiratory hospitalization was observed for $PM_{2.5}$, results were not statistically significant at the 5% level (statistically significant correlations between pollutants and respiratory hospitalization were observed for CO, SO_2, NO_2 and course particulate matter, $PM_{10-2.5}$) It was stated that average pollutant concentrations in Greater Vancouver were much lower than other Canadian cities (with 10 other cities evaluated). The authors concluded that although the health effects observed in Greater Vancouver were generally low, there were important implications to public health (specifics were not provided).

Association between Particulate Air Pollution and First Hospital Admission for Childhood Respiratory Illness in Vancouver, Canada. **Yang, Q., Chen, Y., Krewski, D., Shi, Y., Burnett, R., T., McGrail, K., M. Archives of Environmental Health: An International Journal. Vol. 59. Iss. 1., pp 14-21. 2004.**

Yang et al. (2004) assessed the impact of particulate air pollution on the first respiratory hospitalization for children under 3 years old in Greater Vancouver from a period of 1995 to 1999. The authors did not find a significant relationship between either maximum or mean $PM_{2.5}$ concentrations and respiratory hospitalization. The following presents a summary of the findings of Yang el al. (2004):

A positive relationship was observed between PM concentrations (daily mean and maximum) with a 3 day lag time for asthma and pneumonia (mean PM_{10} 13.3 µg/m³, $PM_{10-.5}$ 5.6 µg/m³, $PM_{2.5}$ 7.7 µg/m³; maximum PM_{10} 26.7 µg/m³, $PM_{10-.5}$ 13.0 µg/m³, $PM_{2.5}$ 14.0 µg/m³); however, it was concluded that the relationship was not statistically significant. While a correlation was not observed between $PM_{2.5}$ and early childhood respiratory hospitalization, the authors noted that work by the US EPA suggests that coarse particulate pollution may be more appropriate for determining asthmatic response due to the deposition of course particulates in the upper airways. The results of the study indicate that mean and maximum coarse particulate matter ($PM_{10-2.5}$) concentrations were positively correlated to respiratory hospitalization following a 3 day lag period.

Influence of Relatively Low Level of Particulate Air Pollution on Hospitalization for COPD in Elderly People. **Chen, Y., Yang, Q., Krewski, D., Shi, Y., Burnett, R., T., McGrail, K. Inhalation Toxicology, Vol. 16. pp. 21-25. 2004.**

Chen et al. (2004) investigated the association between particulate matter and COPD hospitalization among the 65 year and older population in Greater Vancouver based on data collected from 1995 to 1999. The authors noted that while there is considerable evidence for ambient particulate matter associated hospitalization for COPD and mortality, it is unclear whether particulate matter is an independent risk factor which exacerbates cardio-respiratory disease beyond attribution to climate and gaseous air pollution, particularly in areas with low level air pollution (Chen et al., 2004). The following presents a summary of the findings of Chen et al. (2004):

The study found that an average of 3.2 individuals (ranging from 0 to 15 admissions) were admitted to hospitals daily for COPD in Greater Vancouver. The daily average $PM_{2.5}$ concentrations during the 5 year study period were 7.7 µg/m³. A 6.4% increase of COPD was estimated to be associated with $PM_{2.5}$; however, when adjusted for gaseous co-pollutants, a significant association between $PM_{2.5}$ and COPD hospitalization was not observed. The authors indicated that it was likely that both $PM_{2.5}$ and $PM_{10-2.5}$ were important for COPD exacerbation. The

authors concluded that the effects of particulate matter on COPD hospitalization in the study were less certain as individual particulate matter effects could not be isolated from the combined toxicity of the atmospheric mixture.

***A Time-Series Study of Air Pollution, Socioeconomic Status and Mortality in Vancouver, Canada.* Villeunue, P. J., Burnett, R., T., Shi, Y., Krewski, D., Goldberg, M., S., Hertzman, C., Chen, Y., Brook, J. Journal of Exposure Analysis and Environmental Epidemiology. Vol. 13. pp 427-435. 2003.**

Villeneue et al. (2003) evaluated the association of daily particulate concentrations and mortality in individuals 65 years of age or older living within Greater Vancouver for a period from 1986 to 1998. The following provides a summary of the findings of Villeneue et al. (2003):

Two $PM_{2.5}$ datasets were assessed, with daily sampling from 1995 to 1998 (tapered element oscillating microbalance [TEOM]) being the primary focus of the conclusions of the study and additional data being provided from the results of dichotomous sampling with Teflon filters samples taken every 6th day from 1986 to 1995.

The study included investigating multiple pollutants with TEOM $PM_{2.5}$ data indicating an average daily concentration of 7.9 µg/m³ (ranging from 2 µg/m³ to 32 µg/m³). Mortality effects were not observed due to $PM_{2.5}$ (nor were effects observed for PM_{10} or $PM_{2.5-10}$). The authors stated that the lack of observed effects may potentially be due to the short time series with relatively few deaths and low $PM_{2.5}$ concentrations, with $PM_{2.5}$ concentrations in other Canadian cities being approximately twice that of Vancouver. The study also examined the effects of $PM_{2.5}$ concentrations measured every 6th day (dichotomous sampling) from a period of 1986 to 1995 where a 4.5% increase in all-caused mortality was observed; however, the authors concluded that $PM_{2.5}$ was not an important predictor of mortality based on the more recent 1995 to 1998 TEOM daily measurements and noted that $PM_{2.5}$ exposures have decreased dramatically from 1986 to 1995.

***Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling.* Churg, A., Brauer, M., del Carmen Avila-Casado, M., Fortoul, T. L., Wright, J. L. Environmental Health Perspectives. Vol. 111 Iss. 5., pp 714-718. 2003.**

Churg et al. (2003) investigated the effects of exposure to high level particulate concentrations on the morphometry of airways by conducting a histological examination of lung specimens. While the study focused on the effects of high level exposures to particulate matter in Mexico City, the control group consisted of lung specimens from non-smoking Vancouver residents with Vancouver considered to be a relatively low PM city. The following is a summary of the findings of Churg et al. (2003):

As the study was conducted in the early 2000's, residents were exposed to higher than current day ambient $PM_{2.5}$ concentrations (the authors reporting the 1984-1993 average $PM_{2.5}$ as 15 µg/m³ and PM_{10} as 25 µg/m³). The findings showed that the Vancouver residents had relatively normal membranous and terminal bronchioles. The study found that Mexico City (a city with chronically high PM concentrations with PM_{10} reported at 66 µg/m³, $PM_{2.5}$ concentrations were not reported) residents had significantly greater amounts of fibrous tissue and muscle in airway walls compared to the Vancouver residents. It was concluded that that living in an area with high PM levels could lead to chronic airflow obstruction.

IV-2.1. Summary Regional Health Status

With the exception of the national cohort study, the reviewed studies did not indicate a strong correlation between $PM_{2.5}$ concentrations and observed COPD and mortality in Greater Vancouver; however, studies indicating confounding factors including lack of spatial variability to observe effects based on study design and low $PM_{2.5}$ concentrations. As noted in Crouse et al (2012) and by other authors, it is possible that a large population size is needed to observe changes in mortality rates. It is stressed that $PM_{2.5}$ may be an important factor for COPD exacerbation (Chen et al., 2004) and although the health effects observed in Greater Vancouver were generally low, there were important implications to public health (Fung et al., 2006). While the region specific studies did not present a strong observed correlation, there is a strong possibility that the effect of $PM_{2.5}$ on these health indicators is non-measurable due to low daily and annual $PM_{2.5}$ concentrations and confounding factors associated with epidemiological studies. The findings of a larger cohort study, Crouse et al. (2012), which examined effects on the Canadian population, did present a correlation.

Based on the above, there remains a potential that the Greater Vancouver study area is not large enough for adverse $PM_{2.5}$ effects to be measurable at the present day concentrations. Based on the current body of science (further discussed below), a threshold has not been established for $PM_{2.5}$ exposures (i.e., there is not considered an ambient particulate matter has no effect on health). Consequently, it was considered appropriate to look at health studies conducted by regulatory agencies to establish a better understanding of $PM_{2.5}$ with respect to regional health.

IV-3. REGULATORY AGENCY STUDIES HUMAN HEALTH AND AIR QUALITY

Epidemiological studies conducted specifically for the Greater Vancouver area did not show a clear link between $PM_{2.5}$ concentrations and adverse health effects; however, as discussed, confounding factors such as small population size and low $PM_{2.5}$ concentrations may have resulted in a non-observed response. The Crouse et al. (2012) study, which was a large cohort study which included Greater Vancouver population showed an association between increased $PM_{2.5}$ concentrations and increased mortality.

As there is a strong body of literature supporting a link between $PM_{2.5}$ and adverse health effects, additional information which relies on large scale cohort studies and evaluation by regulatory health agencies responsible for establishing health protection objectives (e.g., WHO) was reviewed. The following provides a summary of reports commissioned by health agencies examining the effects of air pollution and health effects. The review focused on $PM_{2.5}$ concentrations and is presented in the context of establishing a baseline for the study area and is not intended to be a comprehensive review of all pertinent literature.

Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide, Global Updates 2005. Summary of Risk Assessment. **World Health Organization. Genevia, Switzerland. 2006.**

The Word Health Organization (WHO) established air quality guidelines with the objective of providing guidance in reducing the human health impacts resulting from air pollution. A summary of WHO (2006) is presented below:

Due to the absence of a threshold, $PM_{2.5}$ concentrations to protect all individuals from all possible health outcomes cannot be derived and, therefore, WHO (2006) developed guidelines for concentrations below those at which mortality was observed within populations. After conducting an extensive literature review, WHO used the following two studies as the key basis for establishing an annual $PM_{2.5}$ guideline: an American Cancer Society (ACS) study (Pope et al., 2002); and, the Harvard Six-cities study (Dockery et al., 1993, Pope et al., 1995, HEI, 2000, Pope et al., 2002 and Jerrett, 2005). The studies evaluated ambient air pollution and cardiopulmonary mortality and lung cancer effects (with adjustment for risk factors such as smoking). The mean $PM_{2.5}$ concentrations in the ACS study were 20 µg/m³ and 18 µg/m³ in the six-cities study. Based on their review of the studies, it was reported that measurable health effects can be expected when mean annual concentrations range from 11 µg/m³ to 15 µg/m³. Therefore, the WHO guideline was set at 10 µg/m³ as it was considered to be below the mean for the most likely effects. WHO state that the potential for adverse health effects due to $PM_{2.5}$ cannot be ruled out at concentrations of 10 µg/m³ or lower, but that the guideline presents an achievable level in urban environments which is expected to reduce significant health risk to acceptable levels. WHO states that there is little evidence to support the notion of a threshold response and that the low end of the range of concentrations at

which adverse health effects have been seen is not greatly different than background, ranging from 3 µg/m³ to 5 µg/m³.

WHO also recommended a short term (24 hour) guideline of 25 µg/m³ to protect against potential short term spikes in $PM_{2.5}$ concentrations. The short term guideline allows for immediate mitigation due to spikes in $PM_{2.5}$ concentrations.

***Development of Options for a New Provincial $PM_{2.5}$ Air Quality Objective.* SENES Consultants and Bates, D. V. Prepared for BC Lung Association. 2005.**

SENES and Bates (2005) conducted a review of the scientific literature on behalf of the BC Lung Association and provided various recommendations for a provincial air quality guideline for $PM_{2.5}$. The recommended $PM_{2.5}$ is air quality objective of 8-10 µg/m³ was later adopted by the province. The following provides a summary of SENES and Bates (2005):

Air quality guidelines were derived on the basis of mortality and reflect concentrations below which increased mortality outcomes due to exposure to PM air pollution are not expected based on the current body of scientific evidence. The objective of 8-10 µg/m³ is based on the WHO (2006) recommended value of 10 µg/m³ in conjunction with the advisory reporting standard of 8 µg/m³ in Australia where $PM_{2.5}$ concentrations range from 7.7-10.3 µg/m³. As air concentrations in BC, including the Lower Mainland, were considered to be similar to those in Australian cities, the inclusion of a more stringent value (8 µg/m³) of $PM_{2.5}$ was included as threshold effects have not been established for $PM_{2.5}$ exposures and adverse health effects.

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

The Canadian Council of Ministers of the Environment (CCME) have implemented a Canada-Wide Standard (CWS) of 30 µg/m³ for $PM_{2.5}$ under the Canadian Environmental Protection Act (CEPA). The CWS of 30 µg/m³ is based on a 24 hour average with achievement of the standard dependant on the 98th percentile ambient measurement annually averaged over 3 consecutive years. The CCME recognized that $PM_{2.5}$ was the PM fraction that had the greatest effect on human health. The CWS is considered to be an important step in achieving the long-term goal of reducing health effects related to PM exposures. While the CCME recognized that some jurisdictions in Canada have lower $PM_{2.5}$ concentrations and that efforts should be made to reduce PM emissions, the CCME established the CWS of 30 µg/m³ to represent a balance between the desire to achieve the best health and environmental protection possible and economic considerations pertaining to the feasibility and costs of reducing the pollutant emissions.

It is noted that the CWS for $PM_{2.5}$ is similar to the US EPA (2006) 24 hour standard of 35 µg/m³ and the WHO (2005) 24 hour guideline (based on the 99th percentile) of 25 µg/m³.

IV-3.1. Summary Regulatory Human Health Studies

As some health effects are expected with any increase of $PM_{2.5}$, $PM_{2.5}$ objectives have been set to consider the context of continually improving objectives and establishing guidelines at concentrations where significant adverse effects have not been demonstrated. Due to the absence of a threshold, $PM_{2.5}$ concentrations to protect all individuals from all possible health outcomes cannot be derived (WHO, 2006). The federal standard for $PM_{2.5}$ is 30 µg/m³ (24 hour average) and was established to balance reducing health effects related to PM exposures and economic feasibility. The current provincial $PM_{2.5}$ air quality objectives of 8-10 µg/m³ are among the lowest of the available guidelines across Canada and world-wide. WHO (2006) suggested that while a threshold for $PM_{2.5}$ has not been established, the lowest range of observed adverse health effects of 11 µg/m³ are quite similar to background concentrations of 3 µg/m³ to 5 µg/m³ and, consequently, there would be quite minor measureable health risks associated with air concentrations at either WHO (2006) guideline of 10 µg/m³ or the BQ AQO of 8 µg/m³.

IV-4 REFERENCES

A Time-Series Study of Air Pollution, Socioeconomic Status and Mortality in Vancouver, Canada. Villeunue, P.J., Burnett, R., T., Shi, Y., Krewski, D., Goldberg, M. S., Hertzman, C., Chen, Y., Brook, J. Journal of Exposure Analysis and Environmental Epidemiology. Vol. 13. pp 427-435. 2003.

Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide, Global Updates 2005. Summary of Risk Assessment. World Health Organization. Genevia, Switzerland. 2006.

Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide, Global Updates 2005. Summary of Risk Assessment. World Health Organization. Geneva, Switzerland. 2006.

Association between Air Pollution and Multiple Respiratory Hospitalizations among the Elderly in Vancouver, Canada. Fung, K. Y., Khan, S., Krewski, D., Chen, Y. Inhalation Toxicology. Vol. 18. pp. 1005-1011. 2006.

Association between Particulate Air Pollution and First Hospital Admission for Childhood Respiratory Illness in Vancouver, Canada. Yang, Q., Chen, Y., Krewski, D., Shi, Y., Burnett, R., T., McGrail, K., M. Archives of Environmental Health: An International Journal. Vol. 59. Iss. 1., pp 14-21. 2004.

Associations of Ambient Air Pollution with Chronic Obstructive Pulmonary Disease Hospitalization and Mortality. Gan, W, Q., Fitzgerald, M., Carlesten, C., Sadatsafavi, M., Brauer, M. American Journal of Respiratory Critical Care Medicine. Vol. 187., Iss. 7., pp 721-727. April 1, 2013.

Canada Wide Standards for Particulate Matter (PM) and Ozone. Canadian Council of Ministers of the Environment (CCME). 2000.

Chronic Exposure to High Levels of Particulate Air Pollution and Small Airway Remodeling. Churg, A., Brauer, M., del Carmen Avila-Casado, M., Fortoul, T. L., Wright, J. L. Environmental Health Perspectives. Vol. 111 Iss. 5., pp 714-718. 2003.

Development of Options for a New Provincial $PM_{2.5}$ Air Quality Objective. SENES Consultants and Bates, D. V. Prepared for BC Lung Association. 2005.

Development of Options for a New Provincial $PM_{2.5}$ Air Quality Objective. SENES Consultants and Bates, D. V. Prepared for BC Lung Association. 2005.

Influence of Relatively Low Level of Particulate Air Pollution on Hospitalization for COPD in Elderly People. Chen, Y., Yang, Q., Krewski, D., Shi, Y., Burnett, R. T., McGrail, K. Inhalation Toxicology, Vol. 16. pp. 21-25. 2004.

Risk of Nonaccidental and Cardiovascular Mortality in Relation to Long-term Exposure to Low Concentrations of Fine Particulate Matter: A Canadian National-Level Cohort Study. Crouse, D., L., Peters, P. A., van Donkelaar, A., Goldberg, M. S., Vileneuve, P. J., Brion, O., Khan, S., Atari, D. O., Jerrett, M., Pope, C. A., Brauer, M., Brook, J. R., Martin, R. V., Steib, D., Burnett, R. T. Environmental Health Perspectives. Vol. 120 Iss. 5. 2012.

US EPA (United States Environmental Protection Agency). 2006. Fact Sheet – Final Revisions to the National Ambient Air Quality Standards for Particle Pollution (Particulate Matter).

APPENDIX V

SNC·LAVALIN

Toxicity Assessment: Detailed Information

APPENDIX V: RATIONALE FOR TOXICITY REFERENCE VALUES

Toxicity reference values (TRVs) for the chemicals of potential concern (COPC) available from the agencies discussed in Section 5 of the report were compiled and reviewed in order to select the most appropriate TRV for use in the characterization of human health risks associated with exposures to the Project emissions. TRVs represent an acceptable dose or concentration of exposure for the COPCs, or more specifically, a dose/concentration without appreciable risk of causing harmful effects). As discussed in Section 5 of the report, given the Project falls under federal jurisdiction, where available and scientifically defensible, Health Canada inhalation and oral TRVs have been used. In cases where Health Canada TRVs were not available, or were determined not to be suitable (i.e., another agency recommended a TRV based on toxicological data that would not have been available to Health Canada at the time they derived their TRV), other international agency TRVs have been considered (see sources listed in Section 5), with preference given to US Environmental Protection Agency (EPA).

The TRVs were determined from responses to exposures observed in toxicity (animal) studies and epidemiology (human) studies. For non-carcinogens, inhalation TRVs were typically an air concentration (i.e., $\mu g/m^3$), associated with a No Observed Adverse Effect Concentration (NOAEC) or Lowest Observed Adverse Effect Concentration (LOAEC), which was then adjusted (i.e., reduced) by the application of uncertainty factors. Similarly, oral TRVs for non-carcinogens were typically a dose (i.e., µg/kg bw/day) associated with a No Observed Adverse Effect Level (NOAEL) or Lowest Observed Adverse Effect Level (LOAEL), adjusted by the application of uncertainty factors. Uncertainty factors are assigned to account for uncertainty of the response between species (e.g., interspecies uncertainty factor, typically 10-fold), the response within a species population (e.g., intraspecies uncertainty factor, typically 10-fold), the difference in response to sub-chronic versus chronic exposures (e.g., typically 10-fold), for use of a LOAEL instead of a NOAEL (e.g., typically 10-fold), and the quality of the database for observed effects (e.g., database deficiencies uncertainty factor, typically 3 to 10-fold). The overall uncertainty associated with an observed response is the product of the individual uncertainty factors and generally ranges from 10 to 1000. An acceptable air concentration (i.e., $\mu g/m^3$) is referred to as a Tolerable Concentration (TC), a Reference Concentration (RfC), an inhalation Minimum Risk Level (MRL) and/or an inhalation Reference Exposure Level (REL), depending on the agency recommending the TRV.

For carcinogens, it is assumed that there is no air concentration or dose to describe a NOAEL, that is, any exposure to a carcinogen could produce a tumourigenic response. For these chemicals the TRV represents an estimate of carcinogenic potency determined from the slope of the dose-response curve for exposure and the occurrence of cancer. The slope or cancer potency factor is expressed in units of $(\mu g/m^3)^{-1}$ for airborne exposures or as a cancer potency factor/slope factor $(\mu g/kg\ bw/day)^{-1}$. Alternatively, the TRV can also be expressed as a unit risk air concentration ($\mu g/m^3$) or dose (µg chemical/kg body weight/day) associated with an acceptable Incremental Lifetime Cancer Risk (ILCR) estimate (i.e., 1-in-100,000 as per Health Canada).

As discussed in Section 3.3 of the report, the physical chemical properties of the COPCs were reviewed, and the COPCs were divided into two groups: gaseous and non-gaseous. The gaseous COPCs will be present in air, with human exposure limited to inhalation. The non-gaseous COPCs have the potential to be deposited to from the atmosphere to soil or other surfaces in the Study Area, and were therefore retained in the multimedia assessment conducted as part of the HHRA. The bioaccumulation potential of the gaseous COPCs was also evaluated; the gaseous COPCs that were determined to be bioaccumulative, were also retained in the multi-media assessment.

The rationale for the various toxicity reference values selected for use in the human health risk assessment is provided below. For all COPCs, use of a TRV derived specifically for the route of exposure (i.e., inhalation, oral) was preferred. For COPCs where no inhalation TRVs were available, but oral TRVs were recommended by a reputable agency, the oral TRVs were used to assess risks associated with inhalation exposures. This approach was preferred over route-to-route extrapolation (i.e., estimating an inhalation TRV from an oral TRV).

V.1. CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS

Carcinogenic polycyclic aromatic hydrocarbons (PAHs) were identified as those listed in Table 7 of Health Canada (2012a). The following carcinogenic PAHs were identified; the Health Canada potency equivalence factors (PEF) for each of the PAHs (Health Canada, 2012a) are also provided:

Benzo(a)pyrene	1
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(g,h,i)perylene	0.01
Benzo(k)fluoranthene	0.1
Chrysene	0.01
Dibenzo(a,h)anthracene	1
Fluoranthene	0.001
Indeno(1,2,3-c,d)pyrene	0.1
Phenanthrene	0.001

The above listed PEFs were used to adjust the individual PAHs to their carcinogenic potency relative to benzo[a]pyrene. The below identified Health Canada inhalation unit risk and oral slope factor for benzo(a)pyrene was adjusted for each of the above PAHs using their respective PEFs. The resulting cancer risk estimates (potency equivalents) were then summed to estimate a total excess cancer risk for the carcinogenic PAHs for both inhalation and oral exposures.

In addition, for the evaluation of non-cancer effects of certain carcinogenic PAHs lacking an agency recommended Tolerable Daily Intake or Reference Dose, the Tolerable Daily Intake (TDI) for all such carcinogenic PAHs was assumed to be 30 µg/kg body weight/day. This value is equal to the US EPA Reference Doses (RfDs) for pyrene (available at: http://www.epa.gov/iris/subst/0445.htm) and represents the most conservative RfD available for any PAH with 3 or more aromatic rings. This TDI was used to estimate non-cancer effects from oral, dermal and inhalation routes of the carcinogenic PAHs unless otherwise specified.

The carcinogenic PAHs were classified as non-gaseous, and were therefore included in the multi-media assessment. The available inhalation and oral TRVs benzo(a)pyrene are summarized below.

V-1.1. Acute Inhalation TRVs for Benzo(a)pyrene

No acute inhalation exposure limits were identified for benzo(a)pyrene or any of the other carcinogenic PAHs.

V-1.2. Chronic Inhalation TRVs for Benzo(a)pyrene

Chronic inhalation TRVs for benzo(a)pyrene are summarized in the following table.

Table V-1. Chronic inhalation TRVs for Benzo(a)pyrene

Source	Exposure Limit	Value	Reference
Health Canada	***Unit Risk***	***3.1 x 10^{-5} (µg/m^3)$^{-1}$***	***Health Canada, 2010***
California Office of Environmental Health Hazards Assessment (OEHHA)	Unit Risk	1.1 x 10^{-3} (µg/m^3)$^{-1}$	OEHHA, 2008

Health Canada's (2010) recommends an inhalation unit risk of 3.1 x 10^{-5} (µg/m^3)$^{-1}$. The inhalation unit risk was based on inhalation exposure to benzo(a)pyrene via multi-stage modelling of respiratory tract tumours in Syrian golden hamsters in a study conducted in 1981. The hamsters were exposed to benzo(a)pyrene concentrations between 0 and 45.6 mg/m^3 for a duration of 4.5 hours per day, 7 days a week for the first 10 weeks, followed by 3 hours per day, 7 days a week for the remaining exposure period (up to 96 weeks).

The OEHHA (2009) presents an inhalation unit risk estimate of 1.1 x 10^{-3} $(\mu g/m^3)^{-1}$. This value was derived based on the same study used by Health Canada. Linearized multistage modelling was used to evaluate the respiratory tumour incidence data.

Because both Health Canada and the OEHHA unit risk estimates are based on the same study, Health Canada's (2010) inhalation unit risk of 3.1 x 10^{-5} $(\mu g/m^3)^{-1}$ was used, and adjusted by the above PEFs to determine inhalation unit risk estimates (relative to benzo(a)pyrene) for the other carcinogenic PAH COPCs.

V.1.3. Oral TRVs for Benzo(a)pyrene

The oral TRVs for benzo(a)pyrene are summarized in the following table.

Table V-2. Oral TRVs for Benzo(a)pyrene

Source	Exposure Limit	Value	Reference
Health Canada	***Oral Slope Factor (SF)***	***2.3 x 10^{-3} (µg/kg bw/d)$^{-1}$***	***Health Canada, 2010***
Health Canada	Oral SF	1.6 x 10^{-3} (µg/kg bw/d)$^{-1}$	Health Canada, 2013
US EPA	Oral SF	7.3 x 10^{-3} (µg/kg bw/d)$^{-1}$	US EPA, 1994
RIVM	Oral SF	2.0 x 10^{-4} (µg/kg bw/d)$^{-1}$	RIVM, 2001

Health Canada (2010) recommended an oral slope factor (SF) for benzo(a)pyrene of 2.3 x 10^{-3} (µg/kg bw/d)$^{-1}$. The oral slope factor for benzo(a)pyrene was based on a study by Neal and Rigdon (1967) and the observance of gastric tumours (mostly squamous cell papillomas, with a few carcinomas) in mice following dietary exposure to between 0 and 0.25 mg benzo(a)pyrene per gram of food for a duration of 110 days. The SF was derived using multistage modelling and an allometric scaling factor.

Through communication with Chemical Health Hazard Assessment Division (CHHAD) of Health Canada (under the Health Products and Food Branch), an oral SF of 2.3 x 10^{-3} (µg/kg bw/d)$^{-1}$ for benzo(a)pyrene was provided for consideration (Health Canada, 2013). Details of the derivation of the SF were not available for review.

US EPA (1994) recommended an oral SF of 7.3 x 10^{-3} (µg/kg bw/d)$^{-1}$ for benzo(a)pyrene. The SF was based on the mean of four slope factors obtained from different modelling procedures. The data were combined from multiple data sets from multiple studies with mice and rats completed in 1967, 1973 and 1981 using more than one sex and species.

RIVM (2001) recommended an excess carcinogenic risk via oral intake (CRoral) of 0.5 µg/kg bw/d for an acceptable excess lifetime cancer risk of 1:104 (converted to a slope factor of 2 x 10^{-4} (µg/kg bw/d)$^{-1}$ by dividing the acceptable risk level by the CRoral). The CRoral was based on a 1999 study where rats were administered benzo(a)pyrene via gavage for 5 days per week for 2 years. The study noted dose-dependent tumour development in a variety of organs and tissues, most notably, in the liver and stomach. Linear non-threshold modelling was applied to the data to derive the CRoral

The Health Canada (2010) oral SF for benzo(a)pyrene was used in the HHRA. The studies considered by the US EPA were available to Health Canada at the time of derivation of their oral SF, however, Health Canada determined a 1967 study as the most appropriate for use.

For the evaluation of non-cancer effects of the carcinogenic PAHs evaluated in the HHRA, neither Health Canada (2010), US EPA (1994), nor RIVM (2001) provided toxicity reference values as a TDI or RfD. Consequently, the TDI for all such carcinogenic PAHs without a non-cancer TRV was assumed to be 30 µg/kg bw/d. This value is equal to the Health Canada (2010) TDI for pyrene and represents the more conservative TDI available for any PAH with three or more aromatic rings. The Health Canada TDI of 30 µg/kg bw/d was used to estimate non-cancer effects from oral, dermal and inhalation routes for all PAHs, unless otherwise specified.

References for Benzo(a)pyrene:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2012. Federal Contaminated Site Risk Assessment in Canada – Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). Version 2.0. September 2010, revised 2012. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013. Toxicological Reference Doses for Organic Contaminants. Obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. 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.

US EPA, 1994. IRIS Summary of Benzo(a)pyrene (CASRN 50-32-8). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0136.htm

V.2. NON-CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS

The non-carcinogenic PAHs, with the exception of naphthalene and 2-methylnaphthalene, were classified as non-gaseous, and were therefore included in the multi-media assessment. Naphthalene and 2-methylnaphthalene were considered to be semi-volatile, and therefore were also included in the multi-media assessment. The available inhalation and oral TRVs for the non-carcinogenic PAHs are summarized below.

V.2.1. Acenaphthene

V.2.1.1. Inhalation TRVs

No acute or chronic inhalation exposure limits were identified for acenaphthene.

The oral TRVs for acenaphthene are summarized in the following table.

V.2.1.2. Oral TRVs

Table V-3. Oral TRVs for Acenaphthene

Source	Exposure Limit	Value	Reference
Health Canada	***TDI***	***60 µg/kg bw/d***	***Health Canada, 2013***
US EPA	***RfD***	***60 µg/kg bw/d***	***US EPA, 1994***

Communication with the CHHAD of Health Canada (under the Health Products and Food Branch) recommended a TDI of 60 µg/kg bw/d for acenaphthene (Health Canada, 2010). The RfD recommended by Health Canada was based on the RfD recommended by US EPA, which was last reviewed in 1994. US EPA (1994) recommended an oral RfD of 60 µg/kg bw/d, based on study conducted by the US EPA in 1989. From this study, a NOAEL of 175 mg/kg bw/d for hepatotoxicity in CD-1 mice exposed to acenaphthene via gavage administration for 90 days was obtained. A 3,000-fold uncertainty factor was applied to the NOAEL (10-fold for interspecies variability, 10-fold for intraspecies variability, 10-fold for the use of a sub-chronic study, and 3-fold for lack of adequate data).

The Health Canada recommended TDI of 60 µg/kg bw/d, which is based on and equivalent to the US EPA RfD, was used to estimate potential health risks from oral, dermal and inhalation exposures to acenaphthene.

References for Acenaphthene:

Health Canada, 2013. Toxicological Reference Doses for Organic Contaminants. Obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

US EPA, 1994. IRIS Summary of Acenaphthene (CASRN 83-32-9). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0442.htm.

V.2.2. Acenaphthylene

V.2.2.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for acenaphthylene.

V.2.2.2. Oral TRVs

No oral TRVs were identified for acenaphthylene.

V.2.3. Acridine

V.2.3.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for acridine.

V.2.3.2. Oral TRVs

No oral TRVs were identified for acridine.

V.2.4. Anthracene

V.2.4.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for anthracene.

V.2.4.2. Oral TRVs

The oral TRVs for anthracene are summarized in the following table.

Table V-4. Oral TRVs for Anthracene.

Source	Exposure Limit	Value	Reference
Health Canada	***TDI***	***300 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	300 µg/kg bw/d	US EPA, 1993
RIVM	TDI	40 µg/kg bw/d	RIVM, 2001

Through communication documented as Health Canada (2013), an oral TDI of 300 µg/kg bw/d for evaluation of exposure to anthracene was identified from CHHAD. The Health Canada TDI was based on the US EPA RfD for anthracene, which was last reviewed in 1993. The US EPA RfD was based on a NOAEL of 1,000 mg/kg bw/d obtained from a study by the US EPA conducted in 1989 where male and female CD-1 (ICR)BR mice were exposed to anthracene via oral gavage for a duration of 90 days. The NOAEL was established as the highest dose tested (1,000 mg/kg bw/d) and a 3,000-fold uncertainty factor was applied (10-fold for interspecies variation, 10-fold for intraspecies variation, 10-fold for subchronic to chronic extrapolation, and 3-fold for the limitations of the database).

RIVM (2001) recommended an oral TDI of 40 µg/kg bw/d for exposure to anthracene. The TDI was based on the Total Petroleum Hydrocarbons Criteria Working Group (TPHCWG) (1997) evaluation of total petroleum hydrocarbons, where it was concluded that an overall TDI of 40 µg/kg bw/d was applicable to non-carcinogenic aromatic compounds with equivalent carbon numbers of >9 to 16, which included anthracene.

The Health Canada (2013) identified TDI, which is based on and equivalent to the US EPA RfD, was derived specifically for anthracene and was used to estimate potential health risks from oral, dermal and inhalation exposures to anthracene.

<u>References for Anthracene:</u>

Health Canada, 2013. Toxicological Reference Doses for Organic Contaminants. Obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

TPHCWG. 1997. Development of fraction specific reference doses (RfDs) and reference concentrations (RfCs) for total petroleum hydrocarbons. TPHCWG Series Volume 4. Amherst Scientific Publishers, Amherst, MA.

US EPA, 1994. IRIS Summary of Anthracene (CASRN 120-12-7). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0434.htm.

V.2.5. Fluorene

V.2.5.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for fluorene.

V.2.5.2. Oral TRVs

The oral TRVs for fluorene are summarized in the following table.

Table V-5. Oral TRVs for Fluorene.

Source	Exposure Limit	Value	Reference
Health Canada	***TDI***	***40 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	40 µg/kg bw/d	US EPA, 1990
RIVM	TDI	40 µg/kg bw/d	RIVM, 2001

Through communication referenced as Health Canada (2013), an oral TDI of 40 µg/kg bw/d for evaluation of exposure to fluorine was identified from CHHAD. The Health Canada TDI was based on the US EPA RfD for fluorene which was last reviewed in 1990. US EPA (1990) recommended an oral RfD of 40 µg/kg bw/d based on a NOAEL of 125 mg/kg bw/d for decreased red blood cells, packed cell volume and haemoglobin in CD-1 mice exposed to fluorine via gavage for 13 weeks (study conducted by the US EPA, 1989). A 3,000-fold uncertainty factor was applied for inter- and intra-species variability, use of a sub-chronic study, and lack of data in another species.

As was the case with anthracene, RIVM (2001) recommended an oral TDI of 40 µg/kg bw/d for evaluation of exposure to fluorene. The TDI was based on the TPHCWG (1997) evaluation of total petroleum hydrocarbons, where it was concluded that an overall TDI of 40 µg/kg bw/d was applicable to non-carcinogenic aromatic compounds with equivalent carbon numbers of >9 to 16, which included fluorene.

Based on the previously described TRV selection hierarchy, the Health Canada recommended TDI of 40 µg/kg bw/d was used to estimate potential health risks from oral, dermal and inhalation exposures to fluorene.

References for Fluorene:

Health Canada, 2013. Toxicological Reference Doses for Organic Contaminants. Obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

TPHCWG, 1997. Development of fraction specific reference doses (RfDs) and reference concentrations (RfCs) for total petroleum hydrocarbons. TPHCWG Series Volume 4. Amherst Scientific Publishers, Amherst, MA.

US EPA, 1990. IRIS Summary of Fluorene (CASRN 86-73-7). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0435.htm.

V.2.6. Fluoranthene

V.2.6.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for fluoranthene.

V.2.6.2. Oral TRVs

The oral TRVs for fluoranthene are summarized in the following table.

Table V-6. Oral TRVs for Fluoranthene.

Source	Exposure Limit	Value	Reference
Health Canada	***TDI***	***40 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	40 µg/kg bw/d	US EPA, 1993

Through communication referenced as Health Canada (2013), an oral TDI of 40 µg/kg bw/d for evaluation of exposure to fluoranthene was identified from CHHAD. The Health Canada TDI was based on the US EPA RfD, which was last reviewed in 1993. US EPA (1993) recommended an oral RfD of 40 µg/kg bw/d, based on a NOAEL of 125 mg/kg bw/d for nephropathy, increased liver weights, haematological alterations, and clinical

effects in CD-1 mice exposed to fluoranthene via gavage for 13 weeks (US EPA, 1988). A 3,000-fold uncertainty factor was applied to account for inter- and intra-species variability, use of a sub-chronic study, and lack of reproductive/developmental toxicity data and toxicity data in a second species.

Based on the previously described TRV selection hierarchy, the Health Canada recommended TDI of 40 µg/kg bw/d was used to estimate potential health risks from oral, dermal and inhalation exposures to fluoranthene.

References for Fluoranthene:

Health Canada, 2013. Toxicological Reference Doses for Organic Contaminants. Obtained through communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

US EPA, 1993. IRIS Summary of Fluoranthene (CASRN 206-44-0). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0444.htm.

V.2.7. Naphthalene

V.2.7.1. Inhalation TRVs

No acute inhalation TRVs were identified for naphthalene.

The chronic inhalation TRVs identified for naphthalene are presented in the following table.

Table V-7: Chronic Inhalation TRVs for Naphthalene.

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	***TC***	***10 µg/m^3***	***Health Canada, 2013***
World Health Organization (WHO)	Air Quality Guideline	10 µg/m^3	WHO, 2010
US EPA	RfC	3 µg/m^3	US EPA, 1998
OEHHA	Chronic REL	3 µg/m^3	OEHHA, 2008
Agency of Toxic Substances and Disease Registry (ATSDR)	Chronic MRL	3 µg/m^3	ATSDR, 2005

Health Canada (2013) recommends a long-term exposure limit for naphthalene in indoor air of 10 µg/m^3 based on the WHO (2010) air quality guideline for naphthalene. The exposure limit was derived based on the LOAEL of 53 mg/m^3 from studies of rats exposed to naphthalene and screened for nasal lesions. This value was then adjusted for continuous exposure (from 6 hours/day × 5 days/week to 24 hours/day × 7 days/week). A total

uncertainty factor of 1000 was applied to the LOAEL; 10 for interspecies variability, 10 for intraspecies variability and 10 for database deficiencies.

The US EPA (1998) derived a chronic inhalation RfC of 3 µg/m³ for naphthalene. The RfC is based on hyperplasia and metaplasia in respiratory and olfactory epithelium in the nasal cavity of mice following inhalation exposures (based on a study conducted in 1992). A Lowest Observed Adverse Effect Level, Human Equivalent Dose (LOAELHEC) of 9.3 mg/m³ was determined for the study. An uncertainty factor of 3,000 was applied to the LOAELHEC to account for interspecies variability (10), intraspecies variability (10), use of a LOAEL (10), and for database uncertainties (3).

The OEHHA (2008) derived a chronic REL of 9 µg/m³ (0.002 parts per million [ppm]) based the same 1992 study identified by the US EPA as the key study, however, the OEHHA identified a LOAEL of 10 ppm.

The LOAEL was adjusted for continuous exposure to 1.8 ppm and an uncertainty factor of 1,000 (10 for use of a LOAEL, 10 for interspecies variability and 10 for intraspecies variability) was applied to the adjusted LOAEL.

The ATSDR derived a chronic inhalation MRL of 3 µg/m³ (0.0007 ppm) for naphthalene based on the same 1992 study identified by the US EPA and the OEHHA, and the nasal lesions observed in rats. An uncertainty factor of 300 (10 for the use of a LOAEL, 3 for extrapolation from animals to humans using dosimetric adjustment, and 10 for human variability) was applied to the LOAELHEC of 0.002 ppm.

The recently derived Health Canada (2013) RfC of 10 µg/m^3 was selected for use in the HHRA.

V.2.7.2. Oral TRVs

The oral TRVs for naphthalene are presented in the following table.

Table V-8. Oral TRVs for Naphthalene.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***20 µg/kg bw/d***	***Health Canada, 2010***
US EPA	RfD	20 µg/kg bw/d	US EPA, 1998
RIVM	TDI	40 µg/kg bw/d	RIVM, 2001

Health Canada (2010) recommended an oral TDI of 20 µg/kg bw/d for naphthalene. Health Canada's TDI was based on the US EPA RfD for naphthalene, which was last reviewed in 1998. US EPA (1998) reported a NOAEL, adjusted for continuous exposure, of 71 mg/kg bw/d for decreased body weight in Fischer 344 rats exposed to naphthalene for 5 days per week for 13 weeks reported in a study completed in 1980. A 3,000-fold uncertainty

factor was applied to the NOAEL to account for extrapolation from rats to humans, protection of sensitive humans, extrapolation from subchronic to chronic exposure, and database deficiencies, including the lack of chronic oral exposure studies and two generation reproduction toxicity studies.

As with other non-carcinogenic PAHs presented, RIVM (2001) recommended an oral TDI of 40 µg/kg bw/d for exposure to naphthalene. The TDI was based on the TPHCWG (1997) evaluation of total petroleum hydrocarbons, where it was concluded that an overall TDI of 40 µg/kg bw/d was applicable to non-carcinogenic aromatic compounds with equivalent carbon numbers of >9 to 16, which included naphthalene.

Based on the previously described TRV selection hierarchy, the Health Canada TDI of 20 µg/kg bw/d was used to estimate potential health risks from oral and dermal exposures to naphthalene.

References for Naphthalene:

ATSDR, 2005. Toxicological Profile for Naphthalene. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013. Residential Indoor Air Quality Guideline: Naphthalene. Health Canada Environmental and Workplace Health. Available on-line at: http://www.hc-sc.gc.ca/ewh-semt/pubs/air/naphthalene/index-eng.php

OEHHA (California Office of Environmental Health Hazard Assessment). 2008. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

TPHCWG, 1997. Development of fraction specific reference doses (RfDs) and reference concentrations (RfCs) for total petroleum hydrocarbons. TPHCWG Series Volume 4. Amherst Scientific Publishers, Amherst, MA.

US EPA, 1998. IRIS Summary of Naphthalene (CASRN 91-20-3). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0436.htm.

WHO, 2010. World Health Organization Guidelines for Indoor Air Quality, Selected Pollutants. The WHO European Centre for Environment and Health, Bonn Office, ISBN 978 92 890 0213 4. 2010. Available at http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf.

V.2.8. 2-Methylnaphthalene

V.2.8.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for 2-methylnaphthalene.

V.2.8.2. Oral TRVs

The oral TRVs for 2-methylnaphthalene are summarized in the following table.

Table V-9. Oral TRVs for 2-Methylnaphthalene.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***4 µg/kg bw/d***	***Health Canada, 2010***
US EPA	RfD	4 µg/kg bw/d	US EPA, 2003

Health Canada (2010) has recommended an oral TDI of 4 µg/kg bw/d for 2-methylnaphthalene. The Health Canada TDI was based on the US EPA RfD, which was last reviewed in 2003. Using a study completed in 1997, US EPA (2003) identified a benchmark response level of 5% (BMD05) extra risk of the critical effect, pulmonary alveolar proteinosis, which was observed in mice exposed to 2-methylnaphthalene in their diet for 81 weeks. This effect was similar to a disorder of unknown etiology that has been identified in humans, and that if this disorder were to occur in humans following exposure to 2-methylnaphthalene, children may be more susceptible (US EPA, 2003); therefore, it was judged that a 5% extra risk of pulmonary alveolar proteinosis was an appropriate level of extra risk for this critical effect. The BMD05 from the quantal-linear model was 4.7 mg/kg-d for pulmonary alveolar proteinosis in male and female mice exposed to 2-methylnaphthalene in the diet for 81 weeks (US EPA, 2003).

Based on the previously described TRV selection hierarchy, the Health Canada recommended TDI of 4 µg/kg bw/d was used to estimate potential health risks from oral, dermal and inhalation exposures to 2-methylnaphthalene.

References for 2-Methylnaphthalene:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

US EPA, 2003. IRIS Summary of 2-Methylnaphthalene (CASRN 91-57-6). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/1006.htm.

V.2.9. Pyrene

V.2.9.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for pyrene.

V.2.9.2. Oral TRVs

The oral TRVs for pyrene are summarized in the following table.

Table V-10. Oral TRVs for Pyrene.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***30 µg/kg bw/d***	***Health Canada, 2010***
US EPA	RfD	30 µg/kg bw/d	US EPA, 1993

Health Canada (2010) recommended an oral TDI of 30 µg/kg bw/d for the evaluation of exposures to pyrene, based on the recommended RfD from US EPA, which was last reviewed in 1993. The US EPA (1993) RfD was based on a NOAEL of 75,000 µg/kg bw/d for kidney effects, including renal tubular pathology and decreased kidney weights, in mice following 13 weeks of exposure to pyrene via gavage in a study conducted in 1989. A 3,000-fold uncertainty factor was applied to this NOAEL to account for intra- and interspecies variability, conversion from sub-chronic to chronic exposures, lack of toxicity studies in a second species, and lack of developmental/reproductive studies.

Based on the previously described TRV selection hierarchy, the Health Canada TDI of 30 µg/kg bw/d was used to estimate potential health risks from oral, dermal and inhalation exposures to pyrene.

References for Pyrene:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

US EPA, 1993. IRIS Summary of Pyrene (CASRN 129-00-0). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0445.htm.

V.2.10. Quinoline

V.2.10.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for quinoline.

V.2.10.2. Oral TRVs

No oral TRVs were identified for quinoline.

V.3. METALS

The metals/metalloids were classified as non-gaseous, and were therefore included in the multi-media assessment. The available inhalation and oral TRVs for the metal/metalloid COPCs are summarized below.

V.3.1. Aluminum

V.3.1.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for aluminum.

V.3.1.2. Oral TRVs

The oral TRVs for aluminum are summarized in the following table.

Table V-11. Oral TRVs for Aluminum.

Source	Exposure Limit	Value	Reference
Health Canada	***pTDI***	***300 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	0.4 µg/kg bw/d (aluminum phosphide) 0.2 µg/kg bw/d (converted to aluminum)	US EPA, 1988
US EPA	RfD	1,000 µg/kg bw/d	US EPA, 2006
ATSDR	Chronic oral MRL	1,000 µg/kg bw/d	ATSDR, 2008

Communication with Health Canada CHHAD (2013) provided a provisional TWI of 2,000 µg/kg bw/d for aluminum. This is considered to be equivalent to a TDI of 300µg/kg bw/d. Further details of the derivation of the TDI were not available for review.

US EPA (1988) derived a RfD of 0.4 µg/kg bw/d for aluminum phosphide, which is approximately 0.2 µg/kg bw/d for aluminum. The RfD was last reviewed by the agency in 1988. The RfD was based on a chronic oral study conducted in 1972 where rats were exposed to aluminum phosphide-fumigated feed for 2 years. Phosphine gas is liberated from aluminum phosphide and the toxicity of aluminum phosphide is largely attributed to phosphine rather than the aluminum cation. A NOAEL of 0.043 mg/kg bw/d was derived for body weight changes and clinical parameters. An uncertainty factor of 100 was applied to account for inter and intraspecies variability. Since this RfD was based on the toxicity of phosphine, rather than aluminum, and aluminum phosphide, which is synthesized for use as a pesticide, would not likely be found in the coal samples/combustion sources, use of the RfD is this assessment was not considered appropriate.

US EPA (2006) recommended a Provisional Peer Reviewed Toxicity Value (PPRTV) oral RfD of 1,000 µg/kg bw/d. The RfD was based on studies in 1989 and 1995 where a LOAEL of 100 mg/kg bw/d was estimated for minimal neurotoxicity in the offspring of mice exposed to dietary aluminum lactate (soluble aluminum) during gestation and lactation. An uncertainty factor of 100 was applied to account for use of a LOAEL, inter and intraspecies variability.

ATSDR (2008) provided a chronic oral MRL of 1,000 µg/kg bw/d for aluminum. The MRL was based on a LOAEL of 100 mg/kg bw/d for decreased forelimb and hindlimb grip strength and decreased thermal sensitivity in 2 year mouse study in 2000 where the mice were exposed to aluminum lactate in a purified diet. An uncertainty factor of 300 was applied to the LOAEL to account for the use of a LOAEL rather than a NOAEL and inter and intraspecies variability. A modifying factor of 0.3 was applied to account for possible differences in aluminum bioavailability of between aluminum lactate and the aluminum found in drinking water and a typical US diet.

Based on the previously described TRV selection hierarchy, the Health Canada (2013) provisional TDI of 300 µg/kg bw/d was used to assess risks following oral, dermal and inhalation exposures to aluminum.

References for Aluminum:

ATSDR, 2008. Toxicological Profile for Aluminum. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.US EPA, 1988. IRIS Summary of Aluminum Phosphide (CASRN: 20859-73-8). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0005.htm.

US EPA. 2006. Provisional Peer Reviewed Toxicity Values for Aluminum. National Center for Environmental Assessment. Office of Research and Development, United States Environmental Protection Agency, Cincinnati, OH.

V.3.2. Antimony

V.3.2.1. Inhalation TRVs

No acute or chronic inhalation exposure limits were identified for antimony.

V.3.2.2. Oral TRVs

The oral TRVs identified for antimony are summarized in the following table.

Table V-12. Oral TRVs for Antimony.

Source	Exposure Limit	Value	Reference
Health Canada	TDI	0.2 µg/kg bw/d	Health Canada, 1999
Health Canada	***RfD***	***3 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	0.4 µg/kg bw/d	US EPA, 1991
RIVM	TDI	6 µg/kg bw/d	RIVM, 2009

Health Canada (1999) recommended a TDI of 0.2 µg/kg bw/d for antimony, which was used to develop the interim maximum acceptable concentration for antimony in drinking water. The TDI was based on a 1998 study where Sprague-Dawley rats were exposed to soluble trivalent antimony salt in tap water for 13 weeks. A NOAEL of 0.5 mg/L of antimony in drinking water (equivalent to an average intake of 0.06 mg/kg bw/d) was established based on histological changes in the thyroid, liver, pituitary gland, spleen, and thymus. An uncertainty factor of 300 was applied to the NOAEL to account for inter- and intraspecies variation and the use of short-term study.

Through communication identified as Health Canada (2013), a RfD of 3 µg/kg bw/d for antimony was identified from CHHAD. This RfD was based on a study by that reported a significant decrease in thymus to body weight ratios in female mice exposed for 13 weeks to 50 mg antimony (potassium antimony tartrate) per litre of drinking water (6.0 mg/kg bw/day). An uncertainty factor of 2000 was applied to this lowest observed effect level (LOEL) to derive the TDI of 3 µg/kg bw/d (J. Eastwood, personal communication, Health Canada, July 22, 2009). Further details of the study, including the date of the study, were not available for review.

US EPA recommended a RfD of 0.4 µg/kg bw/d for antimony, which was last reviewed in 1991. The RfD was based on a study by Schroeder, et al. (1970) where a LOAEL of 0.35 mg/kg bw/d was established based on the effects to longevity, blood glucose levels, and cholesterol levels of rats exposed to potassium antimony tartrate in water for a chronic duration. An uncertainty factor of 1000 was applied to account for interspecies conversion, protection of sensitive individuals, and the use of a LOAEL instead of a NOAEL.

RIVM (2009) derived the TDI of 6 µg/kg bw/d for antimony from the 1998 study used by Health Canada described above; however, RIVM (2009) used a NOAEL of 6 mg/kg bw/d, as proposed in a 1999 interpretation of the study rather than the NOAEL of 0.06 mg/kg bw/d, as reported in the original study. The 1999 interpretation recommended the NOAEL of 6 mg/kg bw/d because it was concluded that the original NOAEL was based on subtle physiological histological changes in the thyroid gland rather than toxicological changes. RIVM (2009) applied an uncertainty factor of 1,000 to the proposed NOAEL to account for intra and interspecies variation and for the use of a subchronic study.

Based on the previously described TRV selection hierarchy, the Health Canada (2011) RfD was used in the assessment of potential health risks from oral, dermal and inhalation exposures to antimony. The Health Canada (1999) TDI was not selected for use, as the data used in the 1999 derivation were available to Health Canada during their derivation of the RfD recommended in 2011; however, they opted to use a different approach.

References for Antimony:

Health Canada, 1999. Guidelines for Canadian Drinking Water Quality: Supporting Documentation. Antimony. May 1997, edited August 1999, archived June 24, 2013.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1991. IRIS Summary of Antimony (CASRN 7440-36-0). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0006.htm.

V.3.3. Arsenic

V.3.3.1. Inhalation TRVs

The acute inhalation TRVs identified for arsenic are presented in the following table.

Table V-13. Acute Inhalation TRVs for Arsenic.

Source	Exposure Limit	Value (µg/m^3)	Reference
OEHHA	***Acute REL (1 hour)***	***0.2 µg/m³***	***OEHHA, 2008***
OEHHA	8 hour REL	0.015 µg/m³	OEHHA, 2008

The OEHHA (2008) derived an acute REL of 0.2 µg/m³ for arsenic and inorganic arsenic compounds based on reproductive and developmental effects in mice (study conducted in 1985). The study involved the exposure of pregnant mice to As2O3 for 4 hours a day on gestation days 9, 10, 11, and 12. A LOAEL of 0.19 mg/m³ for arsenic (0.26 mg/m³ for As2O3) was estimated for the critical effect of decreased fetal weight. An uncertainty factor of 1,000 was applied to the LOAEL for arsenic to account for interspecies variability in toxicokinetics (3) and toxicodynamics (3), intraspecies variability in toxicodynamics (3) and toxicokinetics (3), and a for use of a LOAEL (10).

In addition, the OEHHA (2008) recommend an 8-hour REL of 0.015 µg/m³, which has been set equivalent to the OEHHA chronic REL for arsenic. The OEHHA (2008) indicates that the value is equivalent to the chronic REL due to arsenic's slow clearance from the body, noting that a single exposure to arsenic would take several days to be cleared, mainly via urinary metabolites.

The OEHHA acute (1 hour) REL was selected for the characterization of risks associated with acute inhalation exposures to arsenic. The OEHHA 8-hour REL was not used in the HHRA as it was not derived using acute exposure data.

The chronic inhalation TRVs for arsenic are summarized in the following table.

Table V-14. Chronic Inhalation TRVs for Arsenic.

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	***Unit Risk***	***6.4 x 10^{-3} (µg/m^3)$^{-1}$***	***Health Canada, 2010***
OEHHA	Unit Risk	3.3 x 10^{-3} (µg/m^3)$^{-1}$	OEHHA, 2009
OEHHA	Chronic REL	0.015 µg/m³	OEHHA, 2008a
RIVM	***tolerable concentration in air (TCA)***	***1 µg/m^3***	***RIVM, 2001***

Health Canada (2010) recommended a unit risk of 6.4 x 10^{-3} $(\mu g/m^3)^{-1}$ based on the increased incidence of lung cancer determined for male workers in copper smelters. The inhalation unit risk was derived from a tumourigenic concentration found to induce a 5% increase in the incidence of, or deaths due to, tumours considered to be associated with exposure (TC05) of 7.83 $\mu g/m^3$ for inhaled arsenic and application of a relative risk model (Health Canada, 2010).

The OEHHA (2009) derived an inhalation unit risk for arsenic of 3.3 x 10^{-3} $(\mu g/m^3)^{-1}$. The OEHHA based their unit risk on the results of a study conducted in 1987. The study calculated standard mortality ratios in respiratory tumours from 582 exposed workers in a Tacoma smelter.

The OEHHA also derived a chronic inhalation REL for arsenic of 0.015 $\mu g/m^3$. The REL is based on the results of two drinking water studies in children (studies conducted in 2003 and 2004) were neurological effects were observed. The OEHHA derived the chronic inhalation REL based on a LOAEL from the drinking water studies of 0.23 $\mu g/m^3$ based on an estimated LOAEL of 2.27 µg/L. An uncertainty factor of 30 was applied based on the use of a LOAEL (3), for toxicodynamic variability (3) and for toxicokinetic variability (3).

RIVM (2001) recommends a chronic TCA of 1 $\mu g/m^3$, based on a LOAEL for lung cancer of 10 $\mu g/m^3$ and a 10-fold uncertainty factor for variation in human susceptibility. RIVM (2001) indicates that the mechanism for the lung tumours was not directly genotoxic, so a threshold exists for this effect.

The Health Canada unit risk of 6.4 x 10^{-3} $(\mu g/m^3)^{-1}$ was used in the HHRA. The RIVM TCA of 1 $\mu g/m^3$ was selected as the most appropriate non-cancer inhalation TRV as it is based on human inhalation data, whereas the OEHHA REL was derived based on the results of drinking water studies (i.e., oral exposure data).

V.3.3.2. Oral TRVs

The oral TRVs for arsenic are summarized in the following table.

Table V-15. Oral TRVs for Arsenic.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral SF***	***1.8 x 10^{-3} (µg/kg bw/d)$^{-1}$***	***Health Canada, 2010***
US EPA	Oral SF	1.5 x 10^{-3} (µg/kg bw/d)$^{-1}$	US EPA, 1998
US EPA	***RfD***	***0.3 µg/kg bw/d***	***US EPA, 1993***
RIVM	TDI	1 µg/kg bw/d	RIVM, 2001
ATSDR	Chronic Oral MRL	0.3 µg/kg bw/d	ATSDR, 2007
OEHHA	Chronic Oral REL	0.0035 µg/kg bw/d	OEHHA, 2008b

Arsenic was recently added to Institute of Medicine of the National Academies (IOM) list of probable essential trace elements, indicating arsenic may play an essential metabolic role in some species (IOM, 2001).

For carcinogenic health effects, Health Canada (2010) recommended an oral SF of 1.8 x 10^{-3} (µg/kg bw/d)$^{-1}$. The slope factor was derived from a natural exposure epidemiological study completed in 2000 and was based on the occurrence of bladder, liver, and lung cancer in humans drinking groundwater with naturally occurring arsenic concentrations ranging from 10 to greater than 600 µg/L (mean arsenic concentration of 300 µg/L to 590 µg/L) for a duration of less than or equal to 60 years. The SF was derived using Poisson model and was based on the upper end of a range of mean unit risks.

US EPA established an oral SF of 1.5 x 10^{-3} (µg/kg bw/day)$^{-1}$ for arsenic, which was last reviewed in 1998. The SF was based on the studies conducted in 1968 and 1977 described below and the occurrence of skin cancer in humans exposed to arsenic in drinking water. US EPA (1988) assessed the data and established that the maximum likelihood estimate of skin cancer risk for a 70 kg person drinking 2 L of water per day ranged from 1 x 10^{-3} to 2 x 10^{-3} for an arsenic intake of 1 µg/kg bw/d. The data were extrapolated to a slope factor using time and dose related formulation of the multistage model (US EPA, 1988).

US EPA (1993) established an oral RfD of 0.3 µg/kg bw/d for arsenic, which was last reviewed in 1993. The RfD was based on studies in 1968 and 1977 of a Taiwanese population (n = 40,000) chronically exposed to arsenic in drinking water, where the occurrence of non-cancerous skin lesions (e.g., hyper-pigmentation, keratosis) and vascular effects was investigated. The average concentration of arsenic in drinking water from the control group (9 µg/L) was considered the NOAEL. After adjusting for water consumption and other sources of arsenic (adjusted NOAEL 0.8 µg/kg bw/d), an uncertainty factor of 3 was applied to account for limitations of the database (i.e., lack of reproductive studies).

RIVM (2001) recommended a TDI of 1 µg/kg bw/d for arsenic exposure. The TDI was based on recommendations by the Health Council of The Netherlands (1993) to apply an uncertainty factor of 2 to the TDI previously recommended by Vermeire et al. (1991) of 2.1 mg/kg bw/d for arsenic. The TDI of 2.1 mg/kg bw/d was based on a human drinking water epidemiological study. The uncertainty factor of 2 was recommended to account for the inevitable observation errors in epidemiological studies.

ATSDR (2007) recommended a chronic oral MRL of 0.3 µg/kg bw/d for arsenic. The MRL was based on the 1968 and 1977 studies described above.

OEHHA (2008) recommended a chronic oral REL of 0.0035 µg/kg bw/d for arsenic. The REL was based on studies in 2003 and 2004 where 10 year old children were exposed to arsenic in drinking water for 9.5 to 10.5 years. A LOAEL of 2.27 µg/L was established based on decreased intellectual function and adverse effects on neurobehavioral development. A 30-fold uncertainty factor was applied to account for use of a LOAEL, inter and intraspecies variability.

For the purposes of the HHRA, the US EPA (1993) RfD and ATSDR (2007) MRL value of 0.3 µg/kg bw/d was used to evaluate risks from arsenic. Although US EPA (1993) did not have access to the 2003 and 2004 toxicological data considered by OEHHA (2008), it is noted that ATSDR (2007) affirmed the US EPA value as appropriate. Nevertheless, the chronic oral REL of 0.0035 µg/kg bw/d recommended by OEHHA (2008) was considered in the sensitivity analysis. In addition, the Health Canada oral slope factor of 1.8×10^{-3} $(\mu g/kg\text{-}d)^{-1}$ was used. The TRVs were used to estimate potential health risks from oral and dermal exposures to arsenic.

References for Arsenic:

ATSDR, 2007. Toxicological Profile for Arsenic. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Council of The Netherlands. 1993. Arseen, toetsing van een basis document. Report no 1993/02, Den Haad, The Netherlands.

Institute of Medicine of the National Academies (IOM). 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of DRIs. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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. 2008b. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

OEHHA, 2009. California Office of Environmental Health Hazard Assessment. 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.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA. 1988. Quantitative Toxicological Evaluation of Ingested Arsenic. Office of Drinking Water, Washington, DC. (Draft)

US EPA, 1993 and 1998. IRIS Summary of Arsenic (CASRN 7440-38-2). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0278.htm.

V.3.4. Barium

V.3.4.1. Inhalation TRVs

No acute inhalation TRVs were identified for barium.

The chronic inhalation TRVs identified for barium are presented in the following table.

Table V-16: Chronic Inhalation TRVs for Barium.

Source	Exposure Limit	Value (µg/m^3)	Reference
RIVM	***TCA***	***1 µg/m^3***	***RIVM, 2001***

RIVM (2001) derived a TCA of 1 µg/m^3 for chronic exposure. The TCA was based on cardiovascular effects observed in male rats exposed to insoluble barium carbonate dust for 4 hours per day, 6 days per week for a duration of 4 months. The NOAEL was adjusted for continuous exposure, resulting in a value of 0.16 mg/m^3 barium carbonate, which is equivalent to a NOAEL of 0.11 mg/m^3 of barium. An uncertainty factor of 100 was applied to account for interspecies variability and intraspecies variability. The TCA of 1 µg/m^3 is recommended for both soluble and insoluble salts of barium, due to similar absorptions of the salts. The TCA was used to estimate potential health risks from inhalation exposures to barium.

V.3.4.2. Oral TRVs

The oral TRVs for barium are summarized in the following table.

Table V-17. Oral TRVs for Barium.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***200 µg/kg bw/d***	***Health Canada, 2010***
US EPA	RfD	200 µg/kg bw/d	US EPA, 2005
RIVM	TDI	20 µg/kg bw/d (for soluble barium compounds)	RIVM, 2001
ATSDR	Chronic oral MRL	200 µg/kg bw/d (for soluble salts)	ATSDR, 2007

Health Canada (2010) recommended a TDI of 200 µg/kg bw/d for barium. The TDI was based on the recommended US EPA RfD, which was last reviewed by the agency in 2005. The RfD was based on a Benchmark Dose, Lower Confidence Limit (BMDL) of 63,000 µg/kg bw/d estimated from a chronic drinking water study by National Toxicology Program (NTP) (1994) where kidney toxicity (renal lesions) were observed in mice exposed to barium chloride dehydrate in drinking water for 2 years. A 300-fold uncertainty factor was applied to the BMDL for inter and intraspecies variation and deficiencies in the database.

RIVM (2001) stated that only soluble barium-salts are orally bioavailable and thus, insoluble salts are not toxicologically significant via the oral exposure route. RIVM (2001) recommended a TDI of 20 µg/kg bw/d for soluble barium compounds, which was based on the previously recommended TDI of 20 µg/kg bw/d by Vermeire et al. (1991) following the evaluation of human data and cardiovascular effects.

ATSDR (2007) derived the MRL 200 µg/kg bw/d for barium using the same NTP (1994) study and uncertainty factors that were referenced by Health Canada and US EPA.

Based on the previously described TRV selection hierarchy, the Health Canada TDI of 200 µg/kg bw/d was used to estimate potential health risks from oral and dermal exposures to barium.

<u>References for Barium:</u>

ATSDR, 2007. Toxicological Profile for Barium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

NTP. 2004. Nonneoplastic Lesions by Individual Animal - Barium Chloride Dihydrate. Available on-line: http://ntp-server.niehs.nih.gov/index.cfm?objectid=037BBD0D-F9EB-7773-1E4ECB464EC0DF30.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 2005. IRIS Summary of Barium (CASRN 7440-39-3). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0010.htm.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands.

V.3.5. Beryllium

V.3.5.1. Inhalation TRVs

No acute inhalation TRVs were identified for barium.

The chronic inhalation TRVs identified for beryllium are presented in the following table.

Table V-18. Chronic Inhalation TRVs for Beryllium

Source	Exposure Limit	Value (µg/m^3)	Reference
US EPA	***RfC***	***0.02 µg/m^3***	***US EPA, 1998***
US EPA	***Unit Risk***	***2.4 x 10^{-3} (µg/m^3)$^{-1}$***	***US EPA, 1998***
OEHHA	Chronic REL	0.007 µg/m³	OEHHA, 2001, 2008a
OEHHA	Unit Risk	2.4 x 10^{-3} (µg/m^3)$^{-1}$	OEHHA, 2009

The US EPA (1998) recommended a RfC of 0.02 µg/m^3 for beryllium, based on a LOAEL for beryllium sensitization (and potential for progression to chronic beryllium disease [CBD]) in an occupational exposure study where workers were exposed to 0.55 µg beryllium/m^3 (median of average concentrations) (study conducted in 1996). An uncertainty factor of 10 (3 for use of a LOAEL and 3 for database deficiencies) was applied to the adjusted LOAEL for the study of 0.2 µg/m³. The US EPA provided detailed rationale for their selection of an uncertainty factor of 10, noting that only a small percentage of the population (1% to 5%) appears to be susceptible to CBD, and therefore, because individuals developing beryllium sensitization and CBD are the most sensitive subpopulation, an uncertainty factor of 1 was used to account for human variability (i.e., intraspecies variability). Additionally, an uncertainty factor of 1 was also used to adjust for the less-than-chronic exposure duration of the Kreiss et al. (1996) study as the available data suggests that the occurrence of CBD does not appear to be related to exposure duration.

For assessing the carcinogenic effects of inhalation exposure to beryllium, the US EPA (1998) recommended an inhalation unit risk of 2.4 x 10^{-3} $(\mu g/m^3)^{-1}$. The unit risk was based on a lung cancer in workers following occupational exposure, using a retrospective cohort study of 3,055 adult males who worked at a beryllium extraction, processing and fabrication facility. A statistically significant increase in the incidences of deaths attributable to malignant tumours of the trachea, bronchus, and lung were observed in the study group. The US EPA unit risk of 2.4 x 10^{-3} $(\mu g/m^3)^{-1}$ was used to evaluate carcinogenic risks associated with the inhalation of beryllium particulates.

The OEHHA (2008) has developed a chronic inhalation REL of 0.007 µg/m³ based on the same 1996 occupational study used by the US EPA. An uncertainty factor of 30 (10 for use of a LOAEL, 3 for intraspecies variability) was applied to the adjusted LOAEL of 0.2 µg/m³ to derive the REL.

The OEHHA (2009) also presents a chronic inhalation unit risk estimate of 2.4E-03 per µg/m³ based on lung cancer incidence and the results of the same study identified as the key study by the US EPA.

The US EPA RfC of 0.02 $\mu g/m^3$ was selected as the most appropriate value and was used in the HHRA. In addition, a unit risk of 2.4 x 10^{-3} $(\mu g/m^3)^{-1}$ was used to characterize carcinogenic risks associated with inhalation exposures to beryllium. It is noted that both the US EPA and the OEHHA recommended the same value.

V.3.5.2. Oral TRVs

The oral TRVs for beryllium are summarized in the following table.

Table V-19. Oral TRVs for Beryllium.

Source	Exposure Limit	Value	Reference
Health Canada	***TDI***	***2 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	2 µg/kg bw/d	US EPA, 1998
ATSDR	Chronic Oral MRL	2 µg/kg bw/d	ATSDR, 2002
California OEHHA	Chronic Oral REL	2 µg/kg bw/d	OEHHA, 2008b

Health Canada (2011) CHHAD recommended an oral RfD of 2 µg/kg bw/d based on US EPA's RfD, which was last reviewed in 1998, and ATSDR (2002). US EPA (1998), ATSDR (2002), and OEHHA (2008) established a RfD for beryllium of 2 µg/kg bw/d based on a 1976 study. In the study, beagle dogs were administered beryllium and beryllium sulphate tetrahydrate in their diet for 172 weeks. Using dose-response model, a BMD10 of 0.46 mg/kg bw/d was derived, corresponding to a 10% increase in small intestine lesions. An uncertainty factor of 300 was

applied to the BMD10 to account for interspecies variability, intraspecies variability, and database deficiencies (i.e., limitations of human oral data, reproductive/developmental and immunotoxicologic studies).

The Health Canada recommended RfD of 2 µg/kg bw/d was used to assess potential human health risks from oral and dermal exposure to beryllium; the RfD is equivalent to the oral TRVs recommended by the US EPA, ATSDR and the OEHHA.

References for Beryllium:

ATSDR, 2002. Toxicological Profile for Beryllium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta Georgia.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

OEHHA, 2001. California Office of Environmental Health Hazard Assessment. Determination of Non-Cancer Chronic Reference Levels, Chronic Toxicity Summary, Beryllium and Beryllium Compounds. December, 2001. Available at http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=35.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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. 2008b. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

OEHHA, 2009. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 1998. United States Environmental Protection Agency IRIS Summary of Beryllium (CASRN 7440-41-7). Available at: http://www.epa.gov/iris/subst/0012.htm.

V.3.6. Boron

V.3.6.1. Inhalation TRVs

The acute inhalation TRVs identified for boron are presented in the following table.

Table V-20: Acute Inhalation TRVs for Boron.

Source	Exposure Limit	Value (µg/m^3)	Reference
ATSDR	***Acute MRL***	***300 µg/m³***	***ATSDR, 2010***

The ATSDR (2010) recommended an acute inhalation MRL for boron of 300 µg/m^3. The acute MRL is based on a NOAEL of 800 µg/m^3 for mild nasal and throat irritation and significantly increased nasal secretions in volunteers exposed to sodium borate for 20 minutes. An uncertainty factor of 3 for human variability in the pharmacodynamic response to boron. The acute MRL was used in the HHRA.

No chronic inhalation exposure limits were identified for boron, however, the ATSDR has recommended that the acute inhalation MRL for boron of 300 µg/m³ is protective of chronic exposures (ATSDR, 2010). The ATSDR (2010) indicate that the available data indicate that there is no temporal increase in effect intensity, as would be expected for a local irritant, and therefore, the assessment of acute exposures and associated risks in the HHRA is protective of chronic exposures/risks.

V.3.6.2. Oral TRVs

The oral TRVs for boron are summarized in the following table.

Table V-21. Oral TRVs for Boron.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral acceptable daily intake (ADI)***	***17.5 µg/kg bw/d***	***Health Canada, 2010***
Health Canada	TDI	400 µg/kg bw/d	Health Canada, 2013
US EPA	RfD	200 µg/kg bw/d	US EPA, 2004

Health Canada (2010) provided an acceptable daily intake (ADI) of 17.5 µg/kg bw/d for oral exposure to boron. The ADI was based on chronic study in 1972 where dogs were administered boron in their diet for either 2 years or 38 weeks. A NOAEL of 8.75 mg/kg bw/d was established based on testicular atrophy resulting in infertility and

spermatogenic arrest. An uncertainty factor of 500 was applied to the NOAEL to account for inter- and intraspecies variability and study limitations.

Health Canada (2013) CHHAD communications provided a TDI of 400 µg/kg bw/d based on International Programme on Chemical Safety (IPCS) from 1998. The TDI was derived from studies from 1992 and 1996 where rats were administered boric acid in their diets from gestation day 0 to 20. A NOAEL of 9.6 mg/kg bw/d was established from these studies based on decreased fetal body weight. An uncertainty factor of 25 was applied to the NOAEL to account for inter and intraspecies variability.

US EPA provided a RfD of 200 µg/kg bw/d for boron, which was last reviewed by the agency in 2004. The RfD was based on the studies cited by IPCS above. A BMDL05 of 10.3 mg boron/kg bw/d was calculated from the combined observations of developmental effects (decreased fetal weights) from the primary studies. An uncertainty factor of 66 was applied to the BMDL05 to account for toxicodynamic uncertainty for inter and intraspecies variation.

Based on the previously described TRV selection hierarchy, the Health Canada (2010) ADI was selected for use in the HHRA to assess the risks associated with oral and dermal exposures to boron.

References for Boron:

ATSDR, 2010. Toxicological Profile for Boron. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

US EPA. 2004. IRIS Summary of Boron (CASRN: 7440-42-8). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0410.htm.

V.3.7. Cadmium

V.3.7.1. Inhalation TRVs

The acute inhalation TRVs identified for cadmium are presented in the following table.

Table V-22: Acute Inhalation TRVs for Cadmium

Source	Exposure Limit	Value (µg/m^3)	Reference
ATSDR	***Acute MRL***	***0.03 µg/m^3***	***ATSDR, 2012***

The only acute inhalation exposure limit identified for cadmium is an acute MRL recommended by the ATSDR. The ATSDR has derived an acute inhalation MRL of 0.03 µg/m^3 based on a LOAEL of 0.088 mg/m^3 (LOAELHEC of 0.01 mg/m^3) for respiratory effects in rats exposed to cadmium oxide 6.2 hours/day, 5 days/week for 2 weeks (study conducted in 1995). An uncertainty factor of 300, based on use of a LOAEL (10), extrapolation to humans using dosimetric adjustments (3) and intraspecies variability (10), was applied to the LOAELHEC to derive the acute MRL.

Although the ATSDR acute MRL is based on repeat exposure that can occur continuously for up to 14 days (i.e., may be more accurately termed sub-acute), it was conservatively used to assess risks associated with acute inhalation exposures to the 24 hour maximum air concentrations of cadmium.

The chronic inhalation TRVs identified for cadmium are presented in the following table.

Table V-23: Chronic Inhalation TRVs for Cadmium

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	***Unit Risk***	***9.8 x 10^{-3} (µg/m^3)$^{-1}$***	***Health Canada, 2010***
US EPA	Unit Risk	1.8 x 10^{-3} (µg/m^3)$^{-1}$	US EPA, 1992
ATSDR	***Chronic MRL***	***0.01 µg/m³***	***ATSDR, 2012***

Health Canada (2010) recommended an inhalation unit risk of 9.8 x 10^{-3} (µg/m^3)$^{-1}$ based on a tumourigenic concentration (TC05) of 5.1 µg/m^3, which was associated with the 5% increase in incidence of lung cancer in rats chronically exposed to cadmium chloride aerosols for 23 hours per day, 7 days a week for a duration of 18 months (studies conducted in 1983 and 1984).

The US EPA (1992) derived an inhalation unit risk of 1.8 x 10^{-3} (µg/m^3)$^{-1}$ based on lung, trachea and bronchus cancer deaths reported in a 1985 study for occupationally exposed workers. The US EPA also considered the results of the 1983 study used by Health Canada; however, US EPA concluded that the use of available human data were more reliable because of the species variations in response and the type of exposure (cadmium salt vs. cadmium and cadmium oxide).

For non-carcinogenic effects, ATSDR (2012) recommended a chronic inhalation MRL of 0.01 µg/m^3 based on meta-analysis of several studies. An air concentration associated with a urinary cadmium level that would result in a 10% increase in renal damage was established based on a 95% lower confidence limit of 0.5 µg cadmium per g urinary creatinine, which corresponds to inhalation exposure of 0.1 µg/m^3. An uncertainty factor of 9 was applied to account for intraspecies variability and database limitations, including a lack of human data relative to sensitivities of the kidneys and respiratory tract.

The Health Canada unit risk of 9.8 x 10^{-3} (µg/m^3)$^{-1}$ was selected for use in the HHRA for evaluation of cancer risks. It is noted that Health Canada would have had access to the 1985 study identified by the US EPA as the key study. In addition, the ATSDR chronic inhalation MRL of 0.01 µg/m^3 was used to evaluate non-cancer risks via inhalation.

V.3.7.2. Oral TRVs

The identified oral TRVs for cadmium are summarized in the following table.

Table V-24: Oral TRVs for Cadmium

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	PTDI	1 µg/kg bw/d	Health Canada, 2010
WHO Joint FAO/WHO Expert Committee on Food Additives (JECFA)	***TDI (estimated from provisional tolerable monthly intake [PTMI])***	***0.83 µg/kg bw/d***	***WHO JECFA, 2011***
US EPA	RfD	0.5 µg/kg bw/d (for water) 1 µg/kg bw/d (for food)	US EPA, 1994
RIVM	TDI	0.5 µg/kg bw/d	RIVM, 2001
ATSDR	Chronic Oral MRL	0.1 µg/kg bw/d	ATSDR, 2012
OEHHA	Chronic Oral REL	0.5 µg/kg bw/d	OEHHA, 2008
Health Canada	PTDI	1 µg/kg bw/d	Health Canada, 2010

Health Canada (2010) recommended a provisional oral TDI of 1 µg/kg bw/d for cadmium. This TDI was based on renal tubular dysfunction (proximal tubule epithelial cell damage) manifested by low molecular weight proteinuria observed in chronically exposed humans in an occupational exposure epidemiological study conducted in1971. Various routes were examined but inhalation exposure to cadmium oxide dusts and fumes was the primary route (Health Canada, 2010). A NOAEL of 2.5 µg cadmium per gram of creatinine in urine associated with chronic oral intake of 0.5 to 2.0 µg/kg bw/d was established. A provisional tolerable weekly intake of 7 µg/kg bw/week was derived, which is equivalent to 1 µg/kg bw/d (Health Canada, 2010).

WHO JECFA (2011) established a provisional tolerable monthly intake (PTMI) of 25 µg/kg bw/month for dietary exposure to cadmium. Reviewing the recent literature, WHO JECFA (2011) concluded that increased urinary excretion of β2-microglobulin was the most sensitive effect of cadmium exposure. Based on epidemiological data, WHO JECFA (2011) concluded that urinary concentration of cadmium of 5.24 µg/g of creatinine (confidence interval of 4.94 to 5.57 µg/g of creatinine) was not associated with increased urinary excretion of β2-microglobulin. Using a one-compartment toxicokinetic model, the lower bound of this interval (i.e., 4.94 µg/g of creatinine) was then estimated by WHO JECFA (2010) to be associated with an exposure rate of 0.8 µg/kg bw/d or 25 µg/kg bw/month. Although the WHO JECFA (2011) PTMI was developed for food, it can reasonably be considered to be analogous to soil ingestion exposure (i.e., there is no reason that cadmium in soil would have a greater oral bioavailability than cadmium in food);however, for water borne exposures which are not addressed by WHO JECFA (2011), a modifying factor of 2 may be appropriate (i.e., PTMI of 12.5 µg/kg bw/month) since the relatively bioavailability of cadmium in water has been reported to be twice as high by US EPA (1994). Nevertheless, it is stressed that the WHO JECFA (2011) has not made this distinction; however, waterborne exposure are not considered to be appreciable for the current HHRA. Dividing by 30 days, the PTMI of 25 µg/kg bw/month is equivalent to a TDI of 0.83 µg/kg bw/d for cadmium.

US EPA (1994) recommended an oral RfD of 1 µg/kg bw/day for cadmium in food and an oral RfD of 0.5 µg/kg bw/day for cadmium in water. The RfDs were last reviewed in 1994 and were derived assuming 2.5% absorption of cadmium from food and 5% from water. The RfDs were based on a study reported by the US EPA (1985) where it was determined from a number of human chronic exposure studies that a concentration of 200 µg cadmium/g wet human renal cortex is the highest renal level not associated with significant proteinuria. The RfD for cadmium in food (i.e., 1 µg/kg bw/day) was considered most appropriate for the characterization of exposures to soil.

RIVM (2001) recommended a TDI of 0.5 µg/kg bw/d for cadmium but preferentially referenced a TWI of 3.5 µg/kg bw/w, due to cadmium's long biological half-life. The TDI and TWI were based on recommendations by Vermeire et al. (1991) to maintain cadmium levels in the renal cortex below 50 mg/kg and 2.6 µg/g creatinine to prevent renal tubular damage. A study by Nogawa et al. (1989) suggested that the lowest cadmium levels at which adverse renal effects can be detected in approximately 4% of the population is approximately 50 mg/kg. The study suggested that this cadmium level is likely to be reached after approximately 40 to 50 years of receiving a cadmium dose of 1 µg/kg bw/d. As a result, RIVM (2001) applied an uncertainty factor of 2 to the existing recommended TDI of 1 µg/kg bw/d.

ATSDR (2012) recommended a chronic MRL of 0.1 µg/kg bw/d for oral exposure to cadmium. The MRL was based on meta-analysis of several large-scale environmental exposure studies that likely included sensitive subpopulations. From the dose-response analysis, the cadmium point of departure used as the basis of the MRL was determined to be 0.5 µg/g creatinine. The dietary cadmium intake which would result in the urinary cadmium levels of 0.5 µg/g creatinine was determined to be 0.33 µg/kg bw/d. To account for individuals who may be particularly sensitive to cadmium, an uncertainty factor of 3 was applied.

The OEHHA (2008) recommended a chronic oral REL of 0.5 µg/kg bw/d, which was based on US EPA (1985) as described above.

Based on the previously described TRV selection hierarchy, WHO JECFA (2011) was considered to be the most appropriate value. Consequently, the TDI of 0.83 µg/kg bw/d estimated from the PTMI from WHO/JECFA (2011) was selected as the most appropriate oral TRV and was used in the HHRA to estimate potential health non-cancer risks from oral and dermal exposures to cadmium.

References for Cadmium:

ATSDR, 2012. Toxicological Profile for Cadmium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA. 2008. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

RIVM. 2001. Re-evaluation of human-toxicological maximum permissible risk levels. Bilthoven, Netherlands

US EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, United States Environmental Protection Agency, Washington, DC. (Final draft)

US EPA. 1994. IRIS Summary of Cadmium (CASRN 7440-43-9). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0141.htm.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands

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

V.3.8. Chromium (III)

V.3.8.1. Inhalation TRVs

No acute inhalation exposure limits were identified for chromium III.

The chronic inhalation TRVs identified for chromium III are presented in the following table.

Table V-25: Chronic Inhalation TRVs for Chromium III.

Source	Exposure Limit	Value (µg/m^3)	Reference
RIVM	TCA	60 µg/m³	RIVM, 2001
ATSDR	***Intermediate MRL***	***5 µg/m³***	***ATSDR, 2012***

RIVM (2001) provided a TCA of 60 µg/m^3 for metallic and insoluble forms of chromium (III). This TCA was based on a NOAEL of 0.6 mg/m^3 from studies with humans occupationally exposed to metallic chromium; however, the NOAEL was obtained from a review of ATSDR MRL data, which has since been updated. ATSDR (2013) recommended a MRL of 5 µg/m^3 for insoluble chromium (III) and of 0.1 µg/m^3 for soluble chromium (III) particulate for intermediate exposure durations (i.e., 15-364 days). The MRL for insoluble particulates is considered to be most appropriate to characterize chromium present in coal dust generated from the Project.

The ATSDR intermediate MRL of 5 µg/m³ has been derived for intermediate-duration inhalation exposure (i.e., for use in evaluation of exposure periods from 14 to 365 days) to insoluble trivalent chromium particulate compounds. The MRL is based on a minimal LOAEL of 3 mg/m^3 for trace-to-mild septal cell hyperplasia and chronic interstitial inflammation of the lung in rats in a study conducted in 1999.

In addition to the above, Health Canada has derived an inhalation unit risk for total chromium based on occupational exposures to total chromium, including soluble (principally hexavalent) and insoluble (principally trivalent) chromium. Because hexavalent chromium will be assessed separately, and trivalent chromium is not classified as a carcinogen, the Health Canada unit risk was not used to characterize risks associated with exposures to chromium III.

The intermediate inhalation MRL for insoluble chromium (III) particulate was conservatively selected for use in the characterization risks associated with chronic inhalation exposures.

V.3.8.2. Oral TRVs

The oral TRVs for chromium (III) are summarized in the following table.

Table V-26. Oral TRVs for Chromium (III).

Source	Exposure Limit	Value	Reference
Health Canada	***RfD***	***1,500 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	1,500 µg/kg bw/d	US EPA, 1998
RIVM	TDI	5 µg/kg bw/d (soluble) 5,000 µg/kg bw/d (insoluble and metallic)	RIVM, 2001

Communication with Health Canada (2013) CHHAD provided a RfD of 1,500 µg/kg bw/d for chromium III. The RfD was based on the US EPA RfD for insoluble salts of chromium (III), which was last reviewed in 1998. The US EPA (1998) RfD was based on a 1975 study where male and female rats were exposed to chromium (III) oxide in their diets for 5 days per week, for a total of 600 feedings (840 days). The NOAEL, adjusted for continuous exposure, was 1,468 mg/kg bw/d. A modifying factor of 10 was applied for database deficiencies and a 100 fold uncertainly factor (for intra and interspecies variation) was applied.

RIVM (2001) provided a TDI of 5 µg/kg bw/d for soluble chromium (III) compounds and 5000 µg/kg bw/d insoluble and metallic chromium (III). The TDIs were based on previous recommendations by Vermeire et. Al. (1991) and data referenced from the 1998 draft ATSDR toxicological profile for Chromium (III), which has be updated in 2012 (ATSDR, 2012). The current ATSDR (2012) profile concludes that there is insufficient data to derive a chronic oral MRL for chromium (III). RIVM (2001) based the TDI for soluble chromium (III) compounds on a chronic study where rats were exposed to chromium (III) acetate and a NOAEL of 0.46 mg/kg bw/d was derived. An uncertainty factor of 100 was applied to account for inter and intraspecies variability. The TDI for insoluble compounds and metallic chromium (III) was based on the chronic NOAELs for insoluble chromium (III) compounds being approximately 1000 times less toxic than those for insoluble compounds.

The Health Canada TDI was selected for use in the HHRA.

<u>References for Chromium (III):</u>

ATSDR, 2012. Toxicological Profile for Chromium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia. .

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1998. IRIS Summary of Chromium (III) Insoluble Salts (CASRN 16065-83-1). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0028.htm.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands.

V.3.9. Chromium (VI)

V.3.9.1. Inhalation TRVs

No acute inhalation exposure limits were identified for chromium VI.

The chronic inhalation TRVs identified for chromium VI are presented in the following table.

Table V-27: Chronic Inhalation TRVs for Chromium VI.

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	***Unit Risk***	***7.6 x 10^{-2} (µg/m^3)$^{-1}$***	***Health Canada, 2010***
US EPA	***RfC***	***0.1 µg/m³***	***US EPA, 1998***
US EPA	Unit Risk	1.2 x 10^{-2} (µg/m^3)$^{-1}$	US EPA, 1998
ATSDR	Intermediate MRL	0.3 µg/m³	ATSDR, 2012
OEHHA	Unit Risk	1.5 x 10^{-1} (µg/m^3)$^{-1}$	OEHHA, 2009
OEHHA	Chronic REL	0.2 µg/m³	OEHHA, 2008a
RIVM	Cancer risk (CR) (converted to a unit risk)	2.5 x 10^{-2} (µg/m^3)$^{-1}$	RIVM, 2001

The US EPA (1998) recommended a RfC of 0.1 µg/m³ for hexavalent chromium. The RfC was based on a sub chronic rat inhalation study, using the endpoint of lactate dehydrogenase in bronchioalveolar lavage fluid; the RfC has 300 fold uncertainty factory applied to the benchmark concentration (adjusted for pharmacokinetic differences between species) (study conducted in 1990).

Using the same study as the US EPA, the ATSDR and the OEHHA have derived an intermediate inhalation MRL and a chronic REL for hexavalent chromium particulate of 0.3 µg/m³ and 0.2 µg/m³, respectively. The US EPA chronic RfC is more conservative and will be used over the ATSDR intermediate duration MRL for hexavalent chromium and the OEHHA chronic REL.

For assessing the carcinogenic effects of inhalation exposure to chromium (VI), Health Canada (2010) recommended an inhalation unit risk of 7.6×10^{-2} $(\mu g/m^3)^{-1}$. The unit risk was based on a TC05 of 0.66 $\mu g/m^3$ associated with 5% increase in lung cancer among plant workers from a cohort of 332 men employed at a chromate production plant for a duration of between 1 and 8 years (1975 study).

The US EPA and the OEHHA have also derived an inhalation unit risks for hexavalent chromium of 1.2×10^{-2} $(\mu g/m^3)^{-1}$ and 1.5×10^{-1} $(\mu g/m^3)^{-1}$, respectively. Both unit risk estimates were derived from the same occupational study (from 1975) used by Health Canada in the derivation of their unit risk.

RIVM adopted an inhalation cancer risk (CR) of 0.0000025 mg/m^3 (converted to a unit risk of 2.5×10^{-2} $(\mu g/m^3)^{-1}$) that was derived by the World Health Organization (WHO) in 2000 (WHO, 2000) on the basis of four human-epidemiological studies (Langard et al., 1980, 1990; Hayes et al., 1979; Braver et al., 1985). Few details on the derivation of the CR were available from RIVM (2001).

As indicated above, the US EPA RfC of 0.1 µg/m³ was used to characterize non-cancer hazards associated with the inhalation of chromium (VI). In addition, the Health Canada unit risk of 7.6×10^{-2} $(\mu g/m^3)^{-1}$ was used to characterize cancer risks.

V.3.9.2. Oral TRVs

The oral TRVs for chromium (VI) are summarized in the following table.

Table V-28. Oral TRVs for Chromium (VI).

Source	Exposure Limit	Value	Reference
Health Canada	RfD	3 µg/kg bw/d	Health Canada, 2011
US EPA	RfD	3 µg/kg bw/d	US EPA, 1998
RIVM	pTDI	5 µg/kg bw/d	RIVM, 2001
ATSDR	***Chronic Oral MRL***	***0.9 µg/kg bw/d***	***ATSDR, 2012***
California OEHHA	Chronic Oral REL	20 µg/kg bw/d	OEHHA, 2008b
California OEHHA	***Cancer Slope Factor***	***4.2×10^{-4} (µg/kg bw/day)$^{-1}$***	***OEHHA, 2009***

Through communication with Health Canada (2011) CHHAD, a RfD of 3 µg/kg bw/d for chromium (VI) was identified. The RfD was based on the US EPA RfD, which was last reviewed in 1998. The US EPA (1998) RfD was from a one year drinking water study completed in 1958 using rats ingesting chromium (VI) as potassium chromate. A NOAEL of 25 mg/L, or 2.5 mg/kg bw/d, was established with no critical effect reported. A 300-fold uncertainly factor and a 3-fold modifying factor applied to account for inter and intraspecies variability, less-than-lifetime exposure, and database deficiencies.

RIVM (2001) provided a provisional TDI of 5 µg/kg bw/d for chromium (VI). The TDI was based on a one-year drinking water study in rats) (same study as used by US EPA, 1998). Using the study NOAEL of 2.5 mg/kg bw/d and applying an uncertainty factor of 500 to account for inter-individual and interspecies variation and less-than-lifetime exposure, the provisional TDI was derived.

ASTDR (2012) provided a chronic oral MRL of 0.9 µg/kg bw/d for chromium (VI). The MRL was based on a study by NTP in 2008 where non-neoplastic lesions of the duodenum were reported in mice following a chronic, 2 year, sodium dichromate dihydrate drinking water study. A composite uncertainty factor of 100 was applied to account for inter and intraspecies variability.

OEHHA (2008) recommended a chronic oral REL of 20 µg/kg bw/d for chromium (VI). The REL was based on MacKenzie et al. (1958), as described above, with a 300 fold uncertainty factor applied to the NOAEL to account for inter and intraspecies variability.

OEHHA (2009) estimated an oral cancer slope factor of 4.2×10^{-4} $(\mu g/kg\ bw/day)^{-1}$ for chromium (VI). OEHHA (2009) is the only major health agency to estimate an oral cancer potency factor for chromium (VI). OEHHA (2009) did not use the results of the NTP (2008) study which suggested oral cancer risks in mice and rats and instead relied on 1991 mouse study where gastric tumours were noted. Using a linearized multistage model, OEHHA (2009) estimated a cancer slope factor of 4.2×10^{-4} $(\mu g/kg\ bw/day)^{-1}$.

Since the ASTDR TRV was based on the data that were not available to the US EPA at the time of their last update (1998), the ATSDR chronic oral MRL of 0.9 µg/kg bw/d was used to assess potential human health risks from oral and dermal exposure to chromium (VI). In addition, the OEHHA (2009) slope factor of 4.2×10^{-4} $(\mu g/kg\ bw/day)^{-1}$ was used since recent information has been suggestive of cancer potency via the oral route.

References for Chromium (VI):

ATSDR, 2012. Toxicological Profile for Chromium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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. 2008b. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

OEHHA, 2009. California Office of Environmental Health Hazard Assessment. 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.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA. 1998. United States Environmental Protection Agency IRIS Summary of Chromium VI (CASRN 18540-29-9). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0144.htm.

V.3.10. Cobalt

V.3.10.1. Inhalation TRVs

No acute inhalation exposure limits were identified for cobalt.

The chronic inhalation TRVs identified for cobalt are presented in the following table.

Table V-29: Chronic Inhalation TRVs for Cobalt.

Source	Exposure Limit	Value (µg/m^3)	Reference
RIVM	TCA	0.5 µg/m^3	RIVM, 2001
ATSDR	***Chronic MRL***	***0.1 µg/m^3***	***ATSDR, 2004***

RIVM (2001) derived a TCA of 0.5 µg/m^3 for chronic exposure to cobalt. The TCA was based on a LOAEL of 0.05 mg/m^3 for interstitial lung disease in humans (1988 study). An uncertainty factor of 10 was applied for extrapolation from a LOAEL and a factor of 10 was applied for variability in humans (total uncertainty factor of 100).

ATSDR (2004) derived a chronic inhalation MRL for cobalt of 0.1 µg/m^3 based on a NOAEL of 0.0053 mg/m^3 for decreases in several measures of respiratory function in diamond polishers (1992 study). An uncertainty factor of 10 (for human variability) was applied to an adjusted NOAEC of 0.0013 mg/m^3.

The ATSDR (2004) inhalation exposure limit of 0.1 µg/m^3 was used in the HHRA. This value is more recent and conservative than the RIVM value and is also more thoroughly documented.

V.3.10.2. Oral TRVs

The oral TRVs for cobalt are summarized in the following table.

Table V-30. Oral TRVs for Cobalt.

Source	Exposure Limit	Value	Reference
Health Canada	***MRL***	***10 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	0.3 µg/kg bw/d	US EPA, 2008
ATSDR	MRL	10 µg/kg bw/d	ATSDR, 2004
RIVM	TDI	1.4 µg/kg bw/d	RIVM, 2001

Health Canada (2013) communications indicated a MRL of 10 µg/kg bw/d, based on ATSDR. The ATSDR (2004) MRL was derived for intermediate-duration (≤ 365 days) oral exposure and was based on two studies. In a 1958 study b), human males were exposed to cobalt chloride at doses approximately equal to 1 mg cobalt/kg bw/d for 22 days, which results in the development of polycythemia (increased proportion of red blood cells in the blood) which subsided after 9 to 15 days following cessation of cobalt administration. In a 1947 study, rats were exposed to cobalt for eight weeks, resulting in increased erythrocyte numbers and a LOEL of 1 mg/kg bw/d. An uncertainty factor of 100 was applied to account for use of a LOAEL and for human variability.

US EPA (2008) provided a PPRTV RfD of 0.3 µg/kg bw/d for cobalt. The RfD was based on a 1956 study where it was shown that oral exposure to cobalt (1 mg/kg bw/d) for 2 weeks markedly inhibited radioactive iodine uptake in the human thyroid. The LOAEL of 1 mg/kg bw/d was established for decreased iodine uptake in humans. An uncertainty factor of 3,000 was applied to account for extrapolation from a LOAEL to a NOAEL, intraspecies sensitivity, the lack of multi-generation toxicity studies, and extrapolation from subchronic to chronic exposure.

RIVM (2001) recommended a TDI of 1.4 µg/kg bw/d for cobalt. The TDI was previously recommended by Vermeire et al. (1991) and was based on the migration limit of 100 µg cobalt per day for humans from packaging material. The TDI was also supported by a LOAEL of 0.04 mg/kg bw/d reported from a study were cardiomyopathy was observed in humans following intermediate exposure. An uncertainty factor of 30 was applied to account for use of LOAEL rather than a NOAEL and intraspecies variation.

Based on the previously described TRV selection hierarchy, the Health Canada (2011) MRL of 10 µg/kg bw/d was used to assess potential human health risks from oral and dermal exposure to cobalt.

References for Cobalt:

ATSDR, 2004. Toxicological Profile for Cobalt. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA. 2008. Provisional Peer Reviewed Toxicity Values for Cobalt. National Center for Environmental Assessment. Office of Research and Development. United States Environmental Protection Agency, Cincinnati, OH.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands.

V.3.11. Copper

V.3.11.1. Inhalation TRVs

The acute inhalation TRVs identified for copper are presented in the following table.

Table V-31: Acute Inhalation TRVs for Copper.

Source	Exposure Limit	Value (µg/m^3)	Reference
OEHHA	***1 hour REL***	***100 µg/m³***	***OEHHA, 2008***

The OEHHA (2008) recommended an acute (1-hour) REL for copper of 100 µg/m³. The REL was based on mild respiratory effects in workers exposed to 1 to 3 mg/m³ of copper dust for an unknown duration. The exposure resulted in the workers reporting a sweet taste, which is consistent with the onset of metal fume fever (OEHHA, 2008). An uncertainty factor of 10 for human variability was applied to the NOAEL of 1 mg/m³.

The OEHHA 1 hour REL for copper of 100 µg/m³ was used in the characterization of acute inhalation risks for copper.

The chronic inhalation TRVs identified for copper are presented in the following table.

Table V-32: Chronic Inhalation TRVs for Copper

Source	Exposure Limit	Value (µg/m^3)	Reference
RIVM	TCA	1 µg/m³	RIVM, 2001

RIVM (2001) recommended a TCA of 1 µg/m^3, based on a NOAEL of 0.6 mg/m^3 for respiratory and immunological effects. The NOAEL was derived from rabbits exposed to copper chloride for 6 hours per day, 5 days per week for 6 weeks. The NOAEL was adjusted for continuous exposure, and an uncertainty factor of 100 to the NOAEL to account for inter- and intra-species variability.

The RIVM (2001) TCA was used in the assessment of potential risks due to chronic inhalation of copper.

V.3.11.2. Oral TRVs

The oral TRVs for copper are summarized in the following table.

Table V-33. Oral TRVs for Copper.

Source	Exposure Limit	Value	Reference
Health Canada	***Upper Limit (UL)***	***91 µg/kg bw/d for ages 0-4 years; 110 µg/kg bw/d for ages 5-11 years; 126 µg/kg bw/d for ages 12-19; and, 141 µg/kg bw/d for ages 20 and older***	***Health Canada, 2010***
RIVM	TDI	140 µg/kg bw/d	RIVM, 2001

Copper is an essential trace element (ETE) for normal physiological function and disease can result from deficient or excessive intake of copper. Health Canada (2010) provided a UL of 91 µg/kg bw/d for ages less than 4 years, 110 µg/kg bw/d for ages 5-11 years, 126 µg/kg bw/d for ages 12-19, and 141 µg/kg bw/d for ages 20 and older. The ULs were based IOM (2001) and were adjusted for life stage and body weight. The IOM (2001) ULs were

based on two epidemiological studies reported in 1985 and 1993 of humans ingesting copper tablets for the duration of 12 weeks in, or 2 years, resulting in a NOAEL of 10 mg/d based on the critical effect of hepatotoxicity and gastrointestinal effects. No uncertainty factors were applied.

RIVM (2001) provided a TDI of 140 µg/kg bw/d for copper. The TDI was based on the recommended TDI for copper of 140 µg/kg bw/d proposed by Vermeire et al. (1991), which is above the minimum requirements of daily intake of this essential element (i.e., 20 to 80 µg/kg bw/d). A LOAEL of 4.2 mg/kg bw/d was obtained from a study for decreased body weight in mice following chronic oral exposure to copper. An uncertainty factor of 1,000 was applied to account for the use of a LOAEL rather than a NOAEL, as well as inter- and intraspecies variability, which resulted in a TDI that was beyond the limit value for copper deficiency and therefore was not used.

Based on the previously described TRV selection hierarchy, the Health Canada (2010) ULs were used in the assessment of potential risks due to ingestion and dermal exposures to copper.

References for Copper:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

IOM. 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and of the Interpretation and Uses of Dietary Intakes, and Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands.

V.3.12. Indium

V.3.12.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for indium.

V.3.12.2. Oral TRVs

No oral TRVs were identified for indium.

V.3.13. Iron

V.3.13.1. Inhalation TRVs

No chronic inhalation exposure limits were identified for iron.

V.3.13.2. Oral TRVs

The oral TRVs for iron are summarized in the following table.

Table V-34. Oral TRVs for Iron.

Source	Exposure Limit	Value	Reference
Health Canada	***PMTDI***	***800 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	700 µg/kg bw/d	US EPA, 2006

Health Canada (2013) CHHAD communications provided a provisional maximum TDI of 800 µg/kg bw/d, based on the JECFA (1983). The primary study or studies used to derive the provisional maximum TDI was not specified by JECFA.

The US EPA (2006) recommended a PPRTV RfD of 700 µg/kg bw/day for iron. Iron is essential for normal physiologic functioning and disease can result from deficient or excessive intake of iron. The PPRTV was based on a LOAEL of 60 mg/day for gastrointestinal effects in humans exposed for 1 month to iron (ferrous fumarate) in the diet in a study reported in 1994. The estimated mean dietary intake of iron reported for six European countries

by National Academy of Science in 2001 (11 mg/day) was added to this LOAEL, which was then divided by an uncertainty factor of 1.5 and by a reference body weight of 70 kg to determine a PPRTV of 700 µg/kg bw/day.

Based on the previously described TRV selection hierarchy, the Health Canada TDI of 800 µg/kg bw/d was used to assess risks associated with oral, dermal and inhalation exposures to iron.

References for Iron:

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.JECFA. 1983. Toxicological Evaluation of Certain Food Additives and Food Contaminants. WHO Food Additives Series 18. Geneva, April 11-20, 1983.

US EPA. 2006. Provisional Peer Reviewed Toxicity Values for Iron and Compounds. National Center for Environmental Assessment. Office of Research and Development, United States Environmental Protection Agency, Cincinnati, OH.

V.3.14. Lanthanum

V.3.14.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for lathanum.

V.3.14.2. Oral TRVs

The oral TRVs for lanthanum are summarized in the following table.

Table X-35. Oral TRVs for Lanthanum

Source	Exposure Limit	Value	Reference
National Sanitation Foundation (NSF) International	***RfD***	***500 µg/kg bw/d***	***NSF International, 2010***

NSF International (2010) provided an oral RfD of 500 µg/kg bw/d for lanthanum carbonate. The RfD was based on the lowest therapeutic dose of lanthanum carbonate (375 mg/d lanthanum, which is approximately 5.4 mg lanthanum/kg bw/d based on a 70 kg adult) associated with pharmacological activity (reduced serum or urine phosphate) for multiple clinical trials. The point of departure of 5.4 mg lanthanum/kg bw/d was divided by an

uncertainty factor of 10 to account for extrapolation for a LOAEL and use of a subchronic study. The RfD is applicable to the trivalent lanthanum ion from lanthanum carbonate; however, it is not applicable to nitrate or chloride salts of lanthanum (NSF International, 2010).

The NSF International (2010) RfD of 500 µg/kg bw/d was used to asses oral and dermal risks following exposure to lanthanum.

References for Lanthanum:

National Sanitation Foundation (NSF) International. 2010. NSF Lanthanum Carbonate – 2010. Oral Risk Assessment Document. Published November 1, 2010. Available on-line at: http://www.techstreet.com/nsf/products/1754626?product_id=1754626#jumps.

V.3.15. Lead

V.3.15.1. Inhalation TRVs

No acute inhalation exposure limits were identified for lead.

At the time of this report, Health Canada was conducting a review of the toxicology of lead and will be revising the TRV for application to federal contaminated site (Health Canada 2010b). The toxicology of lead was also being re-evaluated by the US EPA, as well as internationally in the European Union and elsewhere. Developmental neurotoxicity was the key effect of lead in children (manifested as decrements in intelligence quotient (IQ) with increasing blood lead levels) and cardiovascular toxicity was the key effect in adults (manifested as increasing systolic blood pressure with increasing blood lead levels).

The chronic inhalation TRVs identified for lead are presented in the following table.

Table V-36: Chronic Inhalation TRVs for Lead.

Source	Exposure Limit	Value (µg/m^3)	Reference
USEPA	***Air Quality Standard***	***0.15 µg/m³***	***US EPA, 2008***
WHO	Air Quality Guideline	0.5 µg/m³	WHO, 2000
OEHHA	Unit Risk	1.2 x 10^{-5} (µg/m³)$^{-1}$	OEHHA, 2009

No inhalation TRVs were identified for lead from Health Canada or the US EPA's IRIS; however, the US EPA, under their National Ambient Air Quality Standards (NAAQS) recommended a standard for lead of 0.15 µg/m^3 for protection of a variety of health effects including considerations on IQ decrements (US EPA, 2008).

The WHO (2000) recommended an air quality guideline for lead of 0.5 µg/m³ based on the recommendation that the annual average air concentration of lead not exceed 0.5 µg/m³. The guideline is based on an air concentration of 0.5 µg/m³ being associated with an upper limit blood lead level of 10 to 30 µg/L (or 1 to 3 µg/dL), and the critical blood lead level proposed by WHO (2000) of 100 µg/L (10 µg/dL). WHO indicated that haematological and nervous system effects have been observed in adults and children at blood lead levels of 100 µg/L. Since that time, additional toxicological information has become available and it is unclear whether the WHO would consider this air quality guideline protective of all concerns.

The OEHHA (2009) recommend an inhalation unit risk for lead of 1.2×10^{-5} $(\mu g/m^3)^{-1}$ based on rat oral exposure studies and that assumption that approximately 50% of inhaled lead is absorbed compared to approximately 10% of ingested lead (OEHHA, 2009). OEHHA is the only major health agency to develop an inhalation unit risk for lead whereas most other agencies consider there not to be sufficient dose-response data to support such a value. Consequently, the OEHHA (2009) unit risk value was not used. Nevertheless, it is noted that the US EPA (2008) of 0.15 $\mu g/m^3$ would be associated with a cancer risk much lower than 1×10^{-5} if the OEHHA (2009) value were considered.

Given the current toxicological literature for lead, the USEPA NAAQS air quality standard of 0.15 µg/m³ was used to characterize risks associated with inhalation exposures to lead.

V.3.15.2. Oral TRVs

The oral TRVs for lead are summarized in the following table.

Table V-37. Oral TRVs for Lead.

Source	Exposure Limit	Value	Reference
Based on WHO JEFCA	***TRV***	***1.3 µg/kg bw/d for adults*** ***0.6 µg/kg bw/d for infants, toddlers and older children***	***Based on WHO JECFA, 2011 potency estimate***

Health Canada does not currently provide a potency estimate for lead (Pb). Although US EPA and OEHHA provide potency estimates for lead in various documents, these are not provided as dose rates and instead are estimates of blood lead levels associated with various effects.

Using a variety of epidemiological data, WHO JECFA (2011) estimated the potency of lead to be 0.6 µg/kg bw/day for toddlers for an upper bound IQ point determent of 1. Furthermore, WHO JECFA (2011) estimated that 1.3 µg/kg bw/day would be associated with a 1 mmHg increase in systolic blood pressure in adults. Although

WHO JECFA (2011) did not specify the acceptable IQ point decrement or blood pressure increase, these potency estimates have recently been used in Wilson and Richardson (2013) and the BC Ministry of Environment (MoE) established an interim lead soil standard using the Wilson and Richardson (2013) approach.

Although OEHHA (2009) recommended a reference blood Pb level of 1 µg/dL for children, which they predict is associated with a 1 IQ point decrement, they do not provide a dose rate estimate for this blood Pb level. Furthermore, the WHO JECFA (2011) analysis has indicated that this is likely an overestimate of the relationship between IQ and blood Pb level.

For the purpose of this assessment, the TRVs of 1.3 µg/kg bw/d and 0.6 µg/kg bw/d based on WHO JECFA (2011) were selected to estimate potential non-carcinogenic health risks from oral and dermal exposures to adults and toddlers, respectively, from lead in soil and dust.

References for Lead:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2009. California Office of Environmental Health Hazard Assessment. 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.

Wilson, R, Richardson, GM. 2013. Lead (Pb) is now a non-threshold substance: how does this affect soil quality guidelines? Human and Ecological Risk Assessment: An International Journal. 19(5):1152-1171. DOI: 10.1080/10807039.2013.771534. WHO JECFA. 2011. WHO Food Additive Series: 64 – Safety Evaluation of Certain Food Additives and Contaminants. Prepared by the seventy-third meeting of JECFA. Joint FAO/WHO Expert Committee on Food Additives.

US EPA, 2008. United States Environmental Protection Agency. National Ambient Air Quality Standards for Lead. Available at http://www.epa.gov/air/criteria.html.

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.

V.3.16. Manganese

V.3.16.1. Inhalation TRVs

The acute inhalation TRVs identified for manganese are presented in the following table.

Table V-38: Acute Inhalation TRVs for Manganese

Source	Exposure Limit	Value (µg/m^3)	Reference
OEHHA	***8 hour REL***	***0.17 µg/m³***	***OEHHA, 2008***

The OEHHA derived an 8-hour REL of 0.17 µg/m³ based nervous system effects in workers exposed to manganese dust for approximately 0.2 to 17.7 years. Although listed as an 8-hour exposure limit, the REL is based on chronic exposure data, and therefore is not considered appropriate for the characterization of acute exposures.

No other acute inhalation exposure limits were identified for manganese. It is noted that as manganese is a systemic toxicant, the assessment of chronic exposures and associated risks for manganese is protective of acute exposures/risks.

The chronic inhalation TRVs identified for manganese are presented in the following table.

Table V-39: Chronic Inhalation TRVs for Manganese.

Source	Exposure Limit	Value (µg/m^3)	Reference
US EPA	**RfC**	**0.05 µg/m³**	**US EPA, 1993**
ATSDR	Chronic MRL	0.3 µg/m³	ATSDR, 2012
OEHHA	Chronic REL	0.09 µg/m³	OEHHA, 2008

The US EPA, ATSDR and OEHHA each derived chronic inhalation exposure limits based on neurotoxic effects observed in workers in a Belgian alkaline battery plant (1992 study). The US EPA based their RfC of 0.05 µg/m³ on the LOAELHEC for the study, and applied an uncertainty factor of 1000 (10 for human variability, 10 for use of a LOAEL, and 10 for database deficiencies). The ATSDR determined a BMCL10 (benchmark concentration at the lower 95 percent confidence limit for the level of manganese exposure expected to result in a 10 percent response rate) of 142 µg/m^3 for the study, and adjusted it for intermittent exposure (5 days/week, 8 hours/day) and divided by an uncertainty factor of 100 (10 each for human variability and for limitations/uncertainties in the database). The OEHHA derived a Benchmark Concentration, 95% lower confidence limit at the 5% response rate (BMCL05) of 72 µg/m³, which was adjusted for intermittent exposures to 26 µg/m³. An uncertainty factor of 300

was then applied (a factor of 3 for use of a sub-chronic study, and factors of 10 for each of intra- and inter-species variability).

As all three TRVs are based on the same study, the US EPA RfC was selected for use in the HHRA (i.e., according to the toxicological hierarchy used in risk assessment in BC and Canada, US EPA values were generally given precedence over ATSDR and OEHHA values unless there is a rationale for why the US EPA values are not protective).

V.3.16.2. Oral TRVs

The oral TRVs for manganese are summarized in the following table.

Table V-40. Oral TRVs for Manganese.

Source	Exposure Limit	Value	Reference
Health Canada	***UL***	***136 µg/kg bw/d for ages 0-4 years; 122 µg/kg bw/d for ages 5-11 years; 142 µg/kg bw/d for ages 12-19; and, 156 µg/kg bw/d for ages 20 and older.***	***Health Canada, 2010***
US EPA	RfD	46.7 µg/kg bw/d	US EPA, 1996

Manganese is an ETE for normal physiological function and disease can result from deficient or excessive intake of manganese. Health Canada (2010) provided a UL for manganese of 136 µg/kg bw/d for ages 0 to 4 years, 122 µg/kg bw/d for ages 5 to 11 years, 142 µg/kg bw/d for ages 12 to 19, and 156 µg/kg bw/d for ages 20 and older. The ULs were based IOM (2001) and were adjusted for life stage and body weight. The IOM (2001) ULs were based on a 1989 study, which used a weight of evidence from epidemiological studies of humans ingesting manganese in food and water for an unspecified duration. A NOAEL of 11 mg/kg bw/d was established from food ingestion and the critical health effect was Parkinsonian-like neurotoxicity. Uncertainty factors were deemed unnecessary.

The US EPA (1996) derived a RfD of 140 µg/kg bw/day for the evaluation of oral exposures to manganese in food. This RfD was based on a composite of data from several human studies reported from 1973 to 1989 and WHO (1973), providing information on the essential intake of manganese and is not based on a specific study identifying an adverse critical effect (US EPA, 1996). The US EPA (1996) recommended a modifying factor of 3 be applied to the RfD for manganese in food when assessing non-dietary exposure to manganese (e.g., water or

soil), based on an epidemiology study which suggested adverse neurological effects at dose levels above an essential level. This results in an oral RfD of 46.7 µg/kg bw/day.

Based on the previously described TRV selection hierarchy, the Health Canada (2010) ULs were used in the assessment of potential risks due to ingestion and dermal exposures to manganese.

References for Manganese:

ATSDR, 2012. Toxicological Profile for Manganese. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

IOM. 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and of the Interpretation and Uses of Dietary Intakes, and Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

National Research Council. 1989. Recommended Dietary Allowances, 10th ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, DC. p. 230-235.

US EPA. 1993 and 1996. IRIS Summary of Manganese (CASRN 7439-96-5). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0144.htm.

WHO. 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532. World Health Organization, Geneva, Switzerland. p. 34-36.

V.3.17. Mercury

V.3.17.1. Inhalation TRVs

The acute inhalation TRVs identified for mercury are presented in the following table.

Table V-41: Acute Inhalation TRVs for Mercury

Source	Exposure Limit	Value (µg/m^3)	Reference
OEHHA	***1 hour REL***	***0.6 µg/m³***	***OEHHA, 2008a***
OEHHA	8 hour REL	0.06 µg/m³	OEHHA, 2008a

The OEHHA (2008) derived an acute 1-hour REL for elemental mercury of 0.6 µg/m³ based on developmental neurotoxicity in rats. In a study conducted in 1993 groups of 12 pregnant rats were exposed by inhalation to 1.8 mg/m³ of metallic mercury vapour for 1 or 3 hours/day on gestation days 11 to 14 and 17 to 21. Dose-dependent CNS disturbances were observed in offspring. An uncertainty factor of 3000 was applied to the LOAEL of the study (10 for use of a LOAEL, 10 for interspecies variability, 10 for intraspecies variability).

The OEHHA also recommended an 8-hour based on occupational exposure studies that were of chronic duration (i.e., 13-15 years). Because the 8-hour REL is based on chronic exposure data, it will not be used in the HHRA.

The OEHHA 1-hour REL will be used to characterize acute inhalation exposure to mercury.

The chronic inhalation TRVs identified for mercury are presented in the following table. It is recognized that much of the mercury present would be particulate bound rather than present as mercury vapour.

Table V-42: Chronic Inhalation TRVs for Mercury Vapour.

Source	Exposure Limit	Value (µg/m3)	Reference
Health Canada	TC	0.06 µg/m³	Health Canada, 2011
US EPA	***RfC***	***0.3 µg/m³***	***US EPA, 1995***
ATSDR	Chronic MRL	0.2 µg/m³	ATSDR, 1999
OEHHA	Chronic REL	0.03 µg/m³	OEHHA, 2008a
RIVM	TCA	0.2 µg/m³	RIVM, 2001

Health Canada (2010) does not provide an inhalation TRV for mercury. Although the Health Canada (2011) DQRA spreadsheet tool provides a TC of 0.06 µg/m^3 for mercury vapour, Health Canada has indicated that the 2010 document generally provides their formal recommendation. Nevertheless, the TC of 0.06 µg/m^3 was still considered as part of the sensitivity analysis.

The US EPA (2014) recommended a RfC of 0.3 µg/m^3 for elemental mercury based on a LOAEL of 0.025 mg/m^3 for neurobehavioral effects in occupationally exposed males in various studies. Observed critical effects included hand tremor, increased memory disturbance and slight subjective and objective evidence of autonomic dysfunction. A 30-fold (10-fold for protection of sensitive subpopulations and use of LOAEL, as well as 3-fold for database deficiencies, including lack of developmental and reproductive studies) uncertainty factor was applied to the LOAEL, which was adjusted for continuous exposure.

The ATSDR derived a chronic inhalation MRL of 0.2 µg/m^3 for metallic mercury vapor. The MRL is based on hand tremors in a group of 26 mercury-exposed workers from three industries exposed to low levels of mercury for an average of 15.3 years (range, 1–41 years) (Fawer et al. 1983).

RIVM derived a TCA for elemental mercury based on the LOAEC of 26 µg/m^3 identified in Fawer et al. (1983). The LOAEC was adjusted to a continuous exposure concentration of 6.2 µg/m^3, and an uncertainty factor of 30 was applied to derive the TCA of 0.2 µg/m^3.

The OEHHA also derived a chronic inhalation exposure limit (REL) for elemental mercury of 0.03 µg/m^3 based on the results of the Danielsson et al. (1993) study discussed under the acute TRVs. An uncertainty factor of 3,000 (30 for interspecies differences, 10 for intraspecies differences, and 10 for use of a LOAEL) was applied to the LOAEL for the study (1.8 mg/m³).

A US EPA RfC of 0.3 µg/m^3 was selected for use in the characterization of chronic inhalation exposures to mercury. The US EPA RfC is based on data from several human studies, and is considered to be the most robust of the available TRVs. Nevertheless, the Health Canada TC of 0.06 µg/m^3 was also considered in the sensitivity analysis to determine if substitution of this value would have a major effect on the results and conclusions.

V.3.17.2. Oral TRVs

The oral TRVs for mercury are summarized in the following table.

Table V-43. Oral TRVs for Mercury.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***0.3 µg/kg bw/d***	***Health Canada, 2010***
RIVM	TDI	2 µg/kg bw/d	RIVM, 2001
California OEHHA	Chronic Oral REL	0.16 µg/kg bw/d	OEHHA, 2008b

Health Canada (2010) provided a TDI for inorganic mercury of 0.3 µg/kg bw/d. The TDI was based on three sub-chronic studies reported from 1978 to 1984 where rats were administered mercury through either sub-cutaneous injections (results converted from sub-cutaneous to oral exposure) for up to 8 weeks or by gavage for 2 months with nephrotoxicity identified as the critical health effect. A LOAEL of 0.3 mg/kg bw/d was derived and an uncertainty factor of 1000 (10-fold for use of subchronic studies, 10-fold for interspecies variability and 10-fold for use of a LOAEL) was applied to the LOAEL.

RIVM (2001) provided a TDI of 2 µg/kg bw/d for exposure to inorganic mercury. The TDI was based on a chronic study by NTP in 1993 where rats and mice were chronically exposed to mercury via gavage. A NOAEL of 0.23 mg/kg bw/d was established based on renal effects, such as changes in kidney weight. An uncertainty factor of 100 was applied to the NOAEL to account for inter and intraspecies variability.

OEHHA (2008) recommended a chronic oral REL of 0.16 µg/kg bw/d for inorganic mercury. The details of the derivation of the REL were not available at the time of this assessment.

Based on the described toxicological hierarchy, the Health Canada (2010) TDI was used to assess potential human health risks from oral and dermal exposure to inorganic mercury.

References for Mercury:

ATSDR, 1999. Toxicological Profile for Mercury. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia. .

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2011. Federal Contaminated Site Risk Assessment in Canada – Spreadsheet Tool for Human Health Detailed Quantitative Risk Assessment (DQRA). Version: July 13, 2011. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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. 2008b. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1995. United States Environmental Protection Agency. IRIS Summary of Mercury, elemental (CASRN 7439-97-6). US EPA, Washington, DC. Available at: http://www.epa.gov/iris/subst/0370.htm.

V.3.18. Molybdenum

V.3.18.1. Inhalation TRVs

No acute inhalation exposure limits were identified for molybdenum.

The chronic inhalation TRVs identified for molybdenum are presented in the following table.

Table V-44: Chronic Inhalation TRVs for Molybenum.

Source	Exposure Limit	Value (µg/m^3)	Reference
RIVM	TCA	12 µg/m³	RIVM, 2001

RIVM (2001) derived a TCA of 12 µg/m^3 for chronic exposure. The TCA was based on a subchronic study where rats and mice were exposed to molybdenum trioxide via inhalation. A NOAEL, based on body weight effects and adjusted for continuous exposure, of 12 mg/m^3 was identified. An uncertainty factor of 1,000 was applied to account for interspecies variability, intraspecies variability, and extrapolation for sub-chronic to chronic exposure.

No other inhalation exposure limits were identified for molybdenum. The TCA of 12 µg/m^3 was selected for use in the HHRA.

V.3.18.2. Oral TRVs

The oral TRVs for molybdenum are summarized in the following table.

Table V-45. Oral TRVs for Molybdenum.

Source	Exposure Limit	Value	Reference
Health Canada	***UL***	***23 µg/kg bw/d for ages 0 months to 11 years; 27 µg/kg bw/d for ages 12 to 19 years; and, 28 µg/kg bw/d for ages 20 years and older.***	***Health Canada, 2010***
US EPA	RfD	5 µg/kg bw/d	US EPA, 1993
RIVM	TDI	10 µg/kg bw/d	RIVM, 2001

Molybdenum is an ETE for normal physiological function and disease can result from deficient or excessive intake of this element. Health Canada (2010) provided a UL of 23 µg/kg bw/d for ages 0 months to 11 years, 27 µg/kg bw/d for ages 12 to 19 years, and 28 µg/kg bw/d for ages 20 years and older based on IOM (2001). The IOM (2001) ULs were derived from a subchronic, developmental/reproductive study conducted in 1990 where rats were administered molybdenum in drinking water for a duration of 9 weeks (including 3 weeks of gestation). A NOAEL of 0.9 mg/kg bw/d was derived based on reproductive effects. An uncertainty factor of 30 was applied to the NOAEL to account for inter and intraspecies variability. The IOM (2001) ULs were adjusted for age group and body weight.

US EPA (1993) provided a RfD of 5 µg/kg bw/d for molybdenum, which was last reviewed in 1993. The RfD was based on a cross-sectional epidemiology study conducted in 1961 where dietary intake of molybdenum was correlated with serum uric acid levels. A molybdenum intake of 0.14 mg/kg bw/d was established as the LOAEL based on elevated serum uric acid levels. An uncertainty factor of 30 was applied to the LOAEL for protection of sensitive human populations and for use of a LOAEL rather than a NOAEL.

RIVM (2001) recommended a TDI of 10 µg/kg bw/d for molybdenum based on Vermeire et al. (1991). The TDI was based on a NOAEL of 1 mg/kg bw/d in rats. Further details of the derivation of the TDI were not available for review.

Based on the previously described TRV selection hierarchy, the Health Canada (2010) recommended ULs, were used to assess potential human health risks from oral and dermal exposure to molybdenum.

References for Molybdenum:

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

IOM. 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of DRIs. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1993. IRIS Summary of Molybdenum (CASRN 7439-98-7). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0425.htm.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands

V.3.19. Nickel

V.3.19.1. Inhalation TRVs

The toxicity of nickel is highly dependent on its chemical form; however, speciation of nickel is difficult. Because the form of nickel in the coal samples/combustion sources is not known, the most stringent (i.e., lowest TC/REL/RfC and greatest unit risk) of the TRVs recommended for the various forms of nickel have conservatively been used.

The acute inhalation TRVs identified for nickel are presented in the following table.

Table V-46: Acute Inhalation TRVs for Nickel.

Source	Exposure Limit	Value (µg/m^3)	Reference
OEHHA	***1 hour REL***	***0.2 µg/m³***	***OEHHA, 2012***
OEHHA	8 hour REL	0.06 µg/m³	OEHHA, 2012

The OEHHA derived an acute REL of 0.2 µg/m³ for immunotoxic effects in mice following a 2-hour inhalation exposure (1978 study) to nickel chloride (NiCl2). An overall uncertainty factor of 1,000 was applied to the time adjusted BMCL for the study to derive the 1 hour REL.

The OEHHA also derived an 8 hour REL based on respiratory effects (alveolar macrophage hyperplasia, alveolar proteinosis and chronic active inflammation) observed in male and female rats following inhalation exposures to nickel sulfate (NiSO4) for 6.2 hours/day, 5 days/week for 16 days to 24 months. The 8 hour REL is based on chronic exposure data, and therefore will not be used in the acute effects assessment.

The OEHHA 1 hour REL of 0.2 µg/m³ was used in the assessment of risks associated with acute exposures to nickel.

The chronic inhalation TRVs identified for nickel are presented in the following table.

Table V-47: Chronic Inhalation TRVs for Nickel.

Source	Exposure Limit	Value (µg/m^3)	Reference
Health Canada	TC (for nickel oxide)	0.02 µg/m³	Health Canada, 2010
Health Canada	TC (for nickel subsulphide)	0.018 µg/m³	Health Canada, 2010
Health Canada	***TC (for nickel sulphate)***	***0.0035 µg/m³***	***Health Canada, 2010***
Health Canada	TC (for metallic nickel)	0.018 µg/m³	Health Canada, 2010
Health Canada	***Unit Risk (for oxidic, sulphidic and soluble nickel)***	***1.3 x 10^{-3} (µg/m^3)$^{-1}$***	***Health Canada, 2010***
Health Canada	Unit Risk (for soluble nickel)	7.1 x 10^{-4} (µg/m^3)$^{-1}$	Health Canada, 2010
US EPA	Unit Risk (for nickel subsulphide)	4.8x10^{-4} (µg/m^3)$^{-1}$	US EPA, 1991
US EPA	Unit Risk (for refinery dust)	2.4 x 10^{-4} (µg/m^3)$^{-1}$	US EPA, 1992
ATSDR	Chronic MRL	0.09 µg/m^3	ATSDR, 2005
RIVM	TCA	0.05 µg/m^3	RIVM, 2001
OEHHA	Chronic REL	0.014 µg/m^3	OEHHA, 2008a and 2012

Health Canada (2010) has derived inhalation unit risk estimates for oxidic, sulphidic and soluble forms of nickel (combined) (1.3 x 10^{-3} (µg/m^3)$^{-1}$), and for soluble forms of nickel (7.1 x 10^{-3} (µg/m^3)$^{-1}$). The unit risk estimates are based on lung cancer mortality data determined from occupational exposure studies from two mines/refineries in Canada and in Norway.

Health Canada also recommends threshold inhalation exposure limits is for nickel oxide, nickel subsulphide, nickel sulphate and metallic nickel. The lowest TC of 0.0035 µg/m^3 is for nickel sulphate, and is based on respiratory effects and lesions in the lung and nasal epithelium observed in rats following subchronic inhalation exposures. An uncertainty factor of 1000 was applied to the NOAEL for the study to account for inter- and intra-species variability and a less than chronic exposure duration.

The US EPA has derived inhalation unit risk estimates for refinery dust (2.4 x 10^{-4} (µg/m^3)$^{-1}$) and for nickel subsulfide (4.8x10^{-4} (µg/m^3)$^{-1}$) based on excess lung cancer mortality observed in four studies of workers exposed to nickel compounds (1981 and 1982 studies). The unit risk estimate for nickel refinery dust (2.4 x 10^{-4} (µg/m^3)$^{-1}$) was used with a multiplication factor of 2 to account for the roughly 50% nickel subsulfide composition to derive the nickel subsulfide unit risk estimate.

The ATSDR derived a chronic MRL for nickel based on nickel sulfate. This MRL is for the soluble nickel compounds (i.e., nickel chloride, nickel sulfate, and nickel nitrite), but ATSDR (2005) indicates that the MRL would also be protective against the toxicity of other nickel compounds (i.e., the less-soluble compounds, including

nickel oxide, nickel subsulfide, and metallic nickel). The chronic inhalation MRL for nickel of 0.09 µg/m^3 is based on a NOAELHEC of 0.0027 mg/m^3 for chronic active inflammation and lung fibrosis in rats exposed to nickel sulfate (1996 study). ATSDR used an uncertainty factor of 30 (3 for extrapolation from animals to humans with dosimetric adjustment and 10 for human variability).

RIVM derived a TCA of 0.05 µg/m^3 based on a duration-adjusted NOAEC for respiratory effects in rats (same study used by the ATSDR), with an uncertainty factor of 100 (10 each for intra- and interspecies variability).

The OEHHA derived a chronic inhalation REL of 0.014 µg/m^3 for nickel and nickel compounds (except nickel oxide) based on pathological changes in the lung, lymph nodes and nasal epithelium in rats following discontinuous inhalation exposures to nickel (1994 study). An uncertainty factor of 100 was applied to the HEC for the study of 1.4 µg/m^3 for the NOAEL.

The Health Canada unit risk estimate for oxidic, sulphidic and soluble nickel of 1.3 x 10^{-3} (µg/m^3)$^{-1}$ and the Health Canada TC for nickel sulphate of 0.0035 µg/m³ was selected for use in the HHRA.

V.3.19.2. Oral TRVs

The oral TRVs for nickel are summarized in the following table.

Table V-48. Oral TRVs for Nickel.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***11 µg/kg bw/d for soluble nickel (nickel chloride and nickel sulphate)***	***Health Canada, 2010***
Health Canada	TDI	25 µg/kg bw/d	Health Canada, 2013
US EPA	RfD	20 µg/kg bw/d (soluble salts)	US EPA, 1996
RIVM	TDI	50 µg/kg bw/d	RIVM 2001
OEHHA	Chronic oral REL	11 µg/kg bw/d	OEHHA, 2008b

Health Canada (2010) recommended a TDI for soluble nickel (primarily nickel chloride or nickel sulphate) of 11 µg/kg bw/d. The TDI was based on a study reported in 2000 where a NOAEL of 1.1 mg/kg bw/d was found from a two-generation reproductive toxicity study in rats, with post-implantation perinatal lethality noted as the critical effect. An uncertainty factor of 100 was applied to the NOAEL to account for inter- and intraspecies variability.

Through communications with Health Canada (2013) CHHAD, a TDI of 25 µg/kg bw/d for nickel was identified. Further details of the derivation of the TDI were not available for review.

US EPA (1996) recommended a RfD of 20 µg/kg bw/d for soluble salts of nickel, which was last reviewed in 1996. The RfD was based on a study in 1976 where rats were fed nickel in their diets for 2 years. A NOAEL of 5 mg/kg bw/d was established based on decreased body and organ weights. An uncertainty factor of 300 was applied to account for inter- and intraspecies variability and inadequacies of reproductive studies.

RIVM (2001) recommended a TDI of 50 µg/kg bw/d for nickel. The value was originally recommended by Vermeire et al. (1991) and was derived from a study where rats were exposed to nickel sulphate in their diet for a semi-chronic exposure duration. A NOAEL of 5 mg/kg bw/d was established and a 100-fold uncertainty factor was applied to the NOAEL.

OEHHA (2008) recommended a chronic oral REL of 11 µg/kg bw/d for nickel. The REL was based on studies by Nickel Producers Environmental Research Association (NiPERA) (2000a,b) and supported by Smith el al (1993). In the studies, rats were exposed to nickel through gavage administration for 70 weeks. A NOAEL of 1.12 mg/kg bw/d was established based on perinatal mortality in a two generation study. An uncertainty factor of 100 was applied to account for inter and intraspecies variability.

The form of nickel in the coal samples/combustion sources is not known, and therefore, the lowest of the TRVs recommended by Health Canada (2010) (11 µg/kg bw/d) for the various forms of nickel was used to assess risks associated with oral and dermal exposures.

References for Nickel:

ATSDR, 2005. Toxicological Profile for Nickel. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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, 2012. California Office of Environmental Health Hazard Assessment. Nickel Reference Exposure Levels, Nickel and Nickel Compounds, Nickel Oxide. Reference Exposure Levels. Office of Environmental Health Hazard Assessment. February 2012. Available at: http://www.oehha.ca.gov/air/chronic_rels/pdf/032312NiREL_Final.pdf.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1991. United States Environmental Protection Agency IRIS Summary of Nickel Subsulphide (CASRN: 12035-72-2). Available at http://www.epa.gov/iris/subst/0273.htm.

US EPA, 1992. IRIS Summary of Nickel refinery dust (no CASRN). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0272.htm.

US EPA, 1996. IRIS Summary of Nickel soluble salts (no CASRN). United States Environmental Protection Agency, Washington, DC. Available at http://cfpub.epa.gov/ncea/iris/index.cfm?fuseaction=iris.showQuickView&substance_nmbr=0271.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands

V.3.20. Selenium

V.3.20.1. Inhalation TRVs

No acute inhalation exposure limits were identified for selenium.

The chronic inhalation TRVs identified for selenium are presented in the following table.

Table V-49: Chronic Inhalation TRVs for Selenium

Source	Exposure Limit	Value	Reference
OEHHA	Chronic REL	20 µg/m³	OEHHA, 2008a and 2001

OEHHA (2008) recommended a chronic inhalation REL of 20 µg/m^3 based on the US EPA (2014) oral RfD of 5 µg/kg bw/d. The oral RfD was based on NOAEL for clinical selenosis (liver, blood, skin, and central nervous system effects) observed in a Chinese epidemiological study of 400 individuals. OEHHA established the REL through route-to-route extrapolation from oral to inhalation exposure. Although route-to-route extrapolation does

create some uncertainty with TRV derivation, selenosis was noted by OEHHA (2001) as a relevant toxicological effect for both oral and inhalation routes and basing the REL on a RfD was reported to be acceptable.

Although the OEHHA (2014) REL is based on an oral study, based on the rationale provided above, it was selected for use in the HHRA.

V.3.20.2. Oral TRVs

The oral TRVs for selenium are summarized in the following table.

Table V-50. Oral TRVs for Selenium.

Source	Exposure Limit	Value	Reference
Health Canada	***UL***	***5.5 µg/kg bw/d for ages 0-6 months; 6.2 µg/kg bw/d for ages 7 months-4 years; 6.3 µg/kg bw/d for ages 5-11 years; 6.2 µg/kg bw/d for ages 12-19; and, 5.7 µg/kg bw/d for ages 20 and older.***	***Health Canada, 2010***
US EPA	RfD	5 µg/kg bw/d	US EPA, 1991
ATSDR	Chronic Oral MRL	5 µg/kg bw/d	ATSDR, 2003
California OEHHA	Chronic Oral REL	5 µg/kg bw/d	OEHHA, 2008b

Selenium is an essential trace element for normal physiological function and disease can result from deficient or excessive intake of selenium. Health Canada (2010) provided a UL of 5.5 µg/kg bw/d for ages 0 to 6 months, 6.2 µg/kg bw/d for ages 7 months to 4 years, 6.3 µg/kg bw/d for ages 5 to 11 years, 6.2 µg/kg bw/d for ages 12 to 19, and 5.7 µg/kg bw/d for ages 20 and older, based on IOM (2000). For adults, the UL was derived from a human epidemiological study conducted in 1994 where selenium was administered through diet and selenosis was identified as the critical health effect. A NOAEL of 800 µg/d was established and an uncertainty factor of 2 was applied to account for the severity of irreversible results. The UL for infants and children was based on a chronic epidemiological study on infants conducted in 1975 where selenium was administered through diet and selenosis was the critical effect. A NOAEL of 7 µg/kg bw/d was established with no uncertainty factor applied. The IOM (2000) ULs were adjusted for age group and body weight.

US EPA (1991) recommended an oral RfD of 5 µg/kg bw/d for selenium, which was last reviewed in 1991. The oral RfD was based on NOAEL of 0.015 mg/kg bw/d for clinical selenosis (liver, blood, skin, and central nervous

system effects) observed in a Chinese epidemiological study of 400 individuals conducted in 1989. An uncertainty factor of 3 was applied to the NOAEL to account for sensitive individuals.

ATSDR (2003) recommended a chronic oral MRL of 5 µg/kg bw/d for selenium. The MRL was based on the 1994 study used by IOM (2000) discussed above. A NOAEL of 819 µg/d (or 0.015 mg/kg bw/d) was established based on the disappearance of symptoms of selenosis. An uncertainty factor of 3 was applied to account for sensitive individuals.

Based on the previously described TRV selection hierarchy, the Health Canada (2010) ULs, which considered dietary requirements of selenium in the sub-adult population, were used in the assessment of potential risks due to inhalation, ingestion and dermal exposures to selenium.

References for Selenium:

ATSDR, 2003. Toxicological Profile for Selenium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Institute of Medicine of the National Academies (IOM). 2000. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Panel on Dietary Antioxidants and Related Compounds, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of DRIs. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

OEHHA, 2001. California Office of Environmental Health Hazard Assessment. Chronic Toxicity Summary, Selenium and Selenium Compounds (other than hydrogen selenide). December 2001. Available at: http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD3_final.pdf#page=476.

OEHHA, 2008a. California Office of Environmental Health Hazard Assessment. 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. 2008b. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

US EPA, 1991. IRIS Summary of Selenium (CASRN: 7782-49-2). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0472.htm.

V.3.21. Strontium

V.3.21.1. Inhalation TRVs

No acute or chronic inhalation exposure limits were identified for strontium.

V.3.21.2. Oral TRVs

The oral TRVs for strontium are summarized in the following table.

Table V-51. Oral TRVs for Strontium.

Source	Exposure Limit	Value	Reference
Health Canada	***RfD***	***600 µg/kg bw/d***	***Health Canada, 2013***
US EPA	RfD	600 µg/kg bw/d	US EPA, 1996

Health Canada (2013) CHHAD provided a RfD of 600 µg/kg bw/d for strontium. The basis for the recommended RfD was not reported by Health Canada (2013).

The US EPA recommended a RfD of 600 µg/kg bw/d for strontium, which was last reviewed in 1996. The RfD was based on three rat studies. In a study conducted by Storey (1961) young and adult rats were administered strontium carbonate in the diets for 20 days. A NOAEL of 190 mg/kg bw/d was established for young rats based on changes in bone mineralization (rachitic bone). In a study conducted by Marie et al. (1985) Sprague-Dawley rats were administered strontium for 9 weeks to determine the effect of low doses of strontium on mineral homeostasis and bone histology. A NOAEL of 525 mg/kg bw/d was established. In the third study, by Skoryna (1981), the oral toxicity of strontium was investigated in adult male RVH hooded rats who were given strontium chloride in drinking water for 3 years. A NOAEL of 263 mg/kg bw/d was established from this study. An uncertainty factor of 300 was applied to the lowest study NOAEL of 190 mg/kg bw/d to account for inter species extrapolation, an incomplete database, and sensitive subpopulations.

The Health Canada (2011) RfD, which is equivalent to the US EPA RfD of 600 µg/kg bw/d, was used to assess oral, dermal and inhalation exposures to strontium.

References for Strontium:

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.US EPA,

US EPA, 1996. IRIS Summary of Strontium (CASRN: 7440-24-6). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0550.htm.

V.3.22. Tin

V.3.22.1. Inhalation TRVs

No acute or chronic inhalation exposure limits were identified for tin.

V.3.22.2. Oral TRVs

Table V-52. Oral TRVs for Tin (Inorganic).

Source	Exposure Limit	Value	Reference
Health Canada	pTWI pTDI	14,000 µg/kg bw/wk 2,000 µg/kg bw/d	Health Canada, 2013
RIVM	TDI	200 µg/kg bw/d	RIVM, 2009

Through communications with Health Canada (2013) CHHAD, a provisional TWI of 14,000 µg/kg bw/wk (equivalent TDI of 2000 µg/kg bw/d) for inorganic tin. The TWI was based on JECFA (1988) provisional TWI, which was last reviewed in 2005. The TWI was based on the observation that inorganic tin at concentrations greater than 150 mg/kg in canned beverages or 250 mg/kg in canned foods may produce acute manifestations of gastric irritation in certain individuals. The specific details of the derivation of the provisional TWI were not available.

RIVM (2009) provided a TDI of 200 µg/kg bw/d for tin. The TDI was based on a 1973 study where Wistar rats were administered tin (as stannous chloride) in their diet for 13 weeks, which resulted in hematological effects. An uncertainty factor of 200 for inter- and intraspecies variability and exposure duration was applied to the NOAEL of 32 mg/kg bw/d.

Based on the previously described TRV selection hierarchy, the Health Canada (2011) provisional TWI of 2,000 µg/kg bw/d was selected for use in the HHRA.

References for Tin:

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.JECFA. 1988. Toxicological Evaluation of Certain Food Additives and Food Contaminants. WHO Food Additives Series 24: Tin. 1988.

RIVM. 2009. Re-evaluation of some human-toxicological maximum permissible risk levels earlier evaluated in the period 1991-2001. Bilthoven, Netherlands.

Vermeire TG, Apeldoorn ME van, Fouw JC de & Janssen PJCM.1991. Voorstel voor de human-toxicologische onderbouwing van C-toetsingswaarden. National Institute of Public Health and the Environment. RIVM report no. 725201005, February 1991, Bilthoven, The Netherlands

V.3.23. Titanium

V.3.23.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for titanium.

V.3.23.2. Oral TRVs

The oral TRVs for titanium are summarized in the following table.

Table V-53. Oral TRVs for Titanium.

Source	Exposure Limit	Value	Reference
NSF International	***RfD***	***3,000 µg/kd bw/d***	***NSF International, 2005***

Oral TRVs for titanium were not available from Health Canada, the US EPA, ATSDR, OEHHA or the WHO. The NSF International (2005) recommended an oral RfD of 3,000 µg/kd bw/d. The RfD was based on a two-year titanium dioxide feeding study in rats conducted by the National Cancer Institute in 1978 where no treatment related effects were observed. A NOAEL of 2,680 mg/kg bw/d was derived from the study and an uncertainty factor of 1,000 was applied to account for inter- and intraspecies extrapolation, as well as database deficiencies.

The RfD of 3,000 µg/kd bw/d was used to assess oral, dermal and inhalation risks from exposure to titanium.

References for Titanium:

National Sanitation Foundation (NSF) International. 2005. NSF Titanium and Titanium Dioxide Standard. Published September 6, 2005. Available on-line at: http://www.techstreet.com/nsf/products/1227185#jumps.

V.3.24. Uranium (non-radiological)

V.3.24.1. Inhalation TRVs

No acute inhalation TRVs were identified for titanium.

The chronic inhalation TRVs identified for uranium are presented in the following table.

Table V-54: Chronic Inhalation TRVs for Uranium.

Source	Exposure Limit	Value (µg/m^3)	Reference
ATSDR	Chronic MRL (insoluble uranium compounds)	0.8 µg/m³	ATSDR, 2013
ATSDR	***Chronic MRL (soluble uranium compounds)***	***0.04 µg/m³***	***ATSDR, 2013***

The ATSDR has derived a chronic inhalation MRL of 0.8 µg/m³ for insoluble uranium compounds based on a LOAEL of 5.1 mg/m³ for lung fibrosis in monkeys exposed to uranium dioxide for 5.4 hours/day, 5 days/week for 5 years (in studies conducted in 1970, 1973). An application of 1000 was applied to the LOAEL (10 for the use of a LOAEL, 10 for interspecies variability, and 10 for intraspecies variability).

The ATSDR also derived a chronic inhalation MRL of 0.04 µg/m³ for soluble uranium compounds based on a BMCL10 19 µg/m³ for renal effects in dogs exposed to uranium tetrachloride 33 hours/week for 1 year (study conducted in 1953) and an uncertainty factor of 100 (10 each for inter- and intraspecies variablity).

The ATSDR chronic inhalation MRL of 0.04 µg/m³ for soluble uranium compounds was used to assess chronic inhalation exposures to uranium. It is noted that the form of the uranium present in the coal is not known and therefore the more conservative TRV for soluble uranium compounds has been used.

V.3.24.2. Oral TRVs

The oral TRVs for uranium are summarized in the following table.

Table V-55. Oral TRVs for Uranium.

Source	Exposure Limit	Value	Reference
Health Canada	***Oral TDI***	***0.6 µg/kg bw/d***	***Health Canada, 2010***
US EPA	RfD	3 µg/kg bw/d (soluble salts)	US EPA, 1989

Health Canada (2010) recommended a TDI of 0.6 µg/kg bw/d for uranium. The TDI was based on a 1998 study where rats were exposed to varying concentrations of uranyl nitrate hexahydrate in the drinking water for 91 days. The critical endpoint was dose-dependent kidney effects and a LOAEL of 0.6 µg/kg bw/d was identified for degenerative lesions in the proximal convoluted tubule of the kidney in male rats. An uncertainty factor of 100 (for intra- and interspecies variation) was applied. Based on the estimated half-life of uranium in the kidneys of 15 days, an additional uncertainty factor to account for the use of a LOAEL instead of a NOAEL was not applied.

US EPA provided a RfD of 3 µg/kg bw/d for soluble uranium salts, which was last reviewed in 1989. The RfD was based on a study conducted by Maynard and Hodge (1949) where rabbits, rats and dogs were administered uranium compounds in their diets for 30 days. Rabbits showed greater sensitivity to the toxic effects of uranium. A LOAEL of 2.8 mg/kg bw/d was established based on body weight loss and moderate nephrotoxicity. An uncertainty factor of 1000 was applied to the LOAEL to account for inter and intraspecies variability and use of a LOAEL rather than a NOAEL.

Based on the previously described TRV selection hierarchy, the Health Canada TDI of 0.6 µg/kg bw/d, was used to assess oral and dermal exposures to uranium.

References for Uranium:

ATSDR, 2013. Toxicological Profile for Uranium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

US EPA, 1989. IRIS Summary of Uranium Soluble Salts (no CASRN). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0421.htm.

V.3.25. Vanadium

V.3.24.1. Inhalation TRVs

The acute inhalation TRVs identified for vanadium are presented in the following table.

Table V-56: Acute Inhalation TRVs for Vanadium.

Source	Exposure Limit	Value (µg/m^3)	Reference
ATSDR	Acute MRL	0.8 µg/m³	ATSDR, 2012
OEHHA	***1 hour REL***	***30 µg/m³***	***OEHHA, 2008***

The ATSDR has derived an acute inhalation MRL 0.8 µg/m³ based on a LOAEL of 560 µg/m³for lung inflammation in rats exposed to vanadium pentoxide for 6 hours/day, 5 days/week for 13 days (study conducted in 2002). The MRL was derived by dividing the LOAELHEC (73 µg/m³) by an uncertainty factor of 90 (3 for use of a minimal LOAEL, 3 for animal to human extrapolation with dosimetric adjustments, and 10 for intraspecies variability).

The OEHHA (2008) presented an acute (1-hour) REL of 30 µg/m³ based on the incidence of bronchial irritation in human volunteers exposed to vanadium pentoxide for eight hours. The lowest LOAEL of 0.1 mg/m³ for subjective reports of increased respiratory mucous production that was cleared by coughing was adjusted from an 8-hour to a 1-hour exposure. An uncertainty factor of 10 was applied to the duration-adjusted LOAEL to account for intraspecies variability. The OEHHA noted that no uncertainty factor was applied for a LOAEL based on the minor nature of the subjective effects.

The OEHHA 1-hour REL of 30 µg/m³ was used in the HHRA. The 1-hour REL is based on human exposure data adjusted for a 1 hour exposure, and the ATSDR MRL is based on subchronic animal exposure data.

The chronic inhalation TRVs identified for vanadium are presented in the following table.

Table V-57: Chronic Inhalation TRVs for Vanadium.

Source	Exposure Limit	Value (µg/m^3)	Reference
ATSDR	***Chronic MRL***	***0.1 µg/m³***	***ATSDR, 2012***
RIVM	Chronic REL	1 µg/m³	RIVM, 2009

ATSDR (2013) provided a chronic inhalation MRL for vanadium of 0.1 µg/m^3. The inhalation MRL for vanadium is based on a chronic inhalation study in rats exposed to vanadium pentoxide 6 hours/day, 5 days/week for 2 years (ATSDR, 2012). The MRL is based on a BMCL10 of 0.04 mg vanadium/m^3 for degeneration of epiglottis

respiratory epithelium observed in the rats. An uncertainty factor of 30 (3 for animal to human extrapolation with dosimetric adjustments and 10 for human variability) was applied.

The RIVM (2009) provides a provisional TCA of 1 µg/m³ for vanadium based on a chronic inhalation study involving rats and mice exposed to vanadium pentoxide (NTP study used by the ATSDR). A LOAEL of 0.5 mg/m³ was identified, and an uncertainty factor of 1000 was applied to account for extrapolation of a LOAEL to NOAEL (10), interspecies variation (10), and intraspecies variation (10).

Since there was not a clear rationale for the difference between these TRVs, the more conservative ATSDR chronic MRL of 0.1 µg/m^3 was selected for use in the HHRA.

V.3.25.2. Oral TRVs

The oral TRVs for vanadium are summarized in the following table.

Table V-58. Oral TRVs for Vanadium.

Source	Exposure Limit	Value	Reference
Health Canada	pTDI	15 µg/kg bw/d	Health Canada, 2013
US EPA	RfD	9 µg/kg bw/d (vanadium pentoxide) 5 µg/kg bw/d (converted to vanadium (V))	US EPA, 1996
US EPA	***RfD***	***0.07 µg/kg bw/d***	***US EPA, 2009***
RIVM	pTDI	2 µg/kg bw/d	RIVM, 2009

Health Canada (2013) CHHAD communications recommended a provisional TDI of 15 µg/kg bw/d for exposure to vanadium. Further details regarding the derivation of the provisional TDI were not available for review.

The US EPA (1996) recommended a RfD of 9 µg/kg bw/d for vanadium pentoxide, which approximates to 5 µg/kg bw/d for vanadium. The RfD, which was last reviewed in 1996, was based on a chronic rat oral study conducted by Stokinger et al. (1953) where rats were exposed to dietary vanadium pentoxide for 2.5 years. A NOAEL of 0.89 mg/kg bw/d was established based on decreased hair cystine. An uncertainty factor of 100 was applied to the NOAEL to account for inter- and intraspecies variability.

US EPA (2009) recommended a PPRTV RfD of 0.07 µg/kg bw/d for vanadium and its soluble compounds. The RfD was based on a study by Boscolo et al. (1994) where rats were exposed to sodium metavanadate in drinking water for 6 months. A NOAEL of 0.12 mg/kg bw/d was established based on kidney toxicity. The NOAEL was adjusted upwards by 0.1 mg/kg bw/d due to dietary exposure, resulting in a NOAEL of 0.22 mg/kg bw/d. An

uncertainty factor of 3000 was applied to account for inter and intraspecies variability, database deficiencies, and extrapolation from subchronic to chronic exposure.

RIVM (2009) presented a provisional TDI of 2 µg/kg bw/d for vanadium. The TDI was based on a reproduction study in rats by Domingo et al. (1986) where a LOAEL of 2.1 mg/kg bw/d was established based on developmental effects. An uncertainty factor of 1000 was applied to the LOAEL. The TDI was considered appropriate for vanadium from salts including sodium metavanadate, sodium orthovanadate, and vanadyl sulphate.

The Health Canada pTDI for vanadium of 15 µg/kg bw/d was selected for use in the HHRA.

References for Vanadium:

ATSDR, 2012. Toxicological Profile for Vanadium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Health Canada, 2013. Toxicological Reference Values, Estimated Daily Intakes, or Dietary Reference Values for Trace Elements. Obtained through Communication with Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. July 2013.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

RIVM, 2009. Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701092/2009. Available at: http://www.rivm.nl/bibliotheek/rapporten/711701092.pdf.

US EPA. 2009. Provisional Peer Reviewed Toxicity Values for Vanadium and Its Soluble Inorganic Compounds Other Than Vanadium Pentoxide. National Center for Environmental Assessment. Office of Research and Development United States Environmental Protection Agency, Cincinnati, OH

US EPA. 1996. IRIS Summary of Vanadium (CASRN:1314-62-1). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0125.htm.

V.3.26. Zinc

V.3.26.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for zinc.

V.3.26.2. Oral TRVs

The oral TRVs for zinc are summarized in the following table.

Table V-59. Oral TRVs for Zinc.

Source	Exposure Limit	Value	Reference
Health Canada	***UL***	***500 µg/kg bw/d for ages 0-19 years; and, 600 µg/kg bw/d for ages 20 and older.***	***Health Canada, 2010***
US EPA	RfD	300 µg/kg bw/d	US EPA, 2005
ATSDR	Chronic oral MRL	300 µg/kg bw/d	ATSDR, 2005

Zinc is an ETE for normal physiological function and disease can result from deficient or excessive intake of zinc. Health Canada (2010) provided a UL of 500 µg/kg bw/d for ages 0-19 years and 600 µg/kg bw/d for ages 20 and older, based on IOM (2001). For adults, the UL was based on a sub-chronic prospective supplementation trial from 1989 where human adults were administered 10 mg/d through dietary intake plus 50 mg/d supplementary intake of zinc for the duration of 10 weeks. The critical health effect was reduced iron and copper status. An uncertainty factor of 1.5 was applied to the LOAEL of 60 mg/d to account for intra-species variability and extrapolation of LOAEL to NOAEL. The UL for infants and children was based on a sub-chronic prospective supplementation trial in 1976 where human infants were given 5.8 mg/L of zinc in formula plus a 4 mg/L supplement for 6 months. A NOAEL of 4.5 mg/d was established based on the critical health effect of increased growth of the infant, specifically, length, weight, and head circumference. No uncertainty factors were applied to the NOAEL. Health Canada (2010) adjusted the ULs for age group and body weight.

The US EPA (2014) recommended a RfD of 300 µg/kg bw/day for zinc, which was last reviewed in 2005. This RfD was based on four principle studies conducted in adult male and female human volunteers reported from 1989 to 2001. A LOAEL of 910 µg/kg bw/d was calculated as the average effect level from the four co-principal studies for decreases in erythrocyte copper, zinc superoxide dismutase activity. A 3-fold uncertainty factor was applied to the LOAEL to account for variability in the sensitivity of the population.

ATSDR (2005) provided a chronic oral MRL of 300 µg/kg bw/d for zinc. The MRL was based on a 1989 study where 0.83 mg supplemental zinc/kg bw/d was designated as the NOAEL. A 3-fold uncertainty factor was applied to account for intraspecies variability.

The Health Canada ULs were selected for use in the HHRA and will be used in the assessment of risks associated with oral, dermal and inhalation exposures to zinc.

References for Zinc:

ATSDR, 2005. Toxicological Profile for Zinc. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Institute of Medicine of the National Academies (IOM). 2001. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and Interpretation and Uses of DRIs, Standing Committee on the Scientific Evaluation of DRIs. Food and Nutrition Board of the Institute of Medicine of the National Academies. National Academy Press, Washington, DC.

US EPA, 2005. IRIS Summary of Zinc (CASRN: 7440-66-6). United States Environmental Protection Agency, Washington, DC. Available at http://www.epa.gov/iris/subst/0426.htm.

V.4 CRITERIA AIR CONTAMINANTS

Air quality objectives/guidelines (AQO) from various regulatory agencies, including Health Canada, Metro Vancouver (2011), BC Ambient Air Quality Objectives (BC MoE, 2013), Canadian Council of Ministers of the Environment (CCME) Canada Wide Standards (CWS) for Particulate Matter (2000) and National Ambient Air Quality Objectives (NAAQO) (1999), World Health Organization Air Quality Guidelines (2000, 2006), US EPA (2014) and OEHHA (2014), were selected for the assessment of potential health risks associated with exposure to CAC.

The CCME has developed up to three objective values using the categories "maximum desirable", "maximum acceptable", and "maximum tolerable". The "maximum desirable" objective is the most stringent standard. British Columbia has established a similar set of objective values, designated as levels A, B and C, with level A being the most stringent. Level A is typically applied to new and proposed discharges to the environment, and is usually the same as the federal "maximum desirable" objective. Metro Vancouver's regional ambient air quality objectives are health-based objectives.

Since particulate matter less than 2.5 microns ($PM_{2.5}$), carbon monoxide (CO), nitrogen dioxide (NO_2), and sulphur dioxide (SO_2) are gaseous or primarily air-suspended contaminants, human exposure through non-inhalation pathways was not assessed. For diesel particulate matter (DPM) and particulate matter between 2.5 and 10 microns (PM_{10}), exposure from non-inhalation routes was characterized through the assessment of individual PAH and metal/metalloid constituents. The inhalation exposure limits selected for the characterization of risks associated with exposures to the CAC are summarized below.

V.4.1. Particulate Matter

The acute and chronic inhalation exposure limits identified for $PM_{2.5}$ and PM_{10} are summarized in the following tables. A summary of the rationale used to develop the exposure limits follows the tables.

Table V-60. Acute Inhalation Exposure Limits for $PM_{2.5}$ and PM_{10}

Source	Exposure Limit	Value	Reference
$PM_{2.5}$			
Metro Vancouver	24 hour AQO	25 µg/m³	Metro Vancouver, 2011
BC MoE	24 hour AQO	25 µg/m³	BC MoE, 2013
CCME	24 hour AQO	30 µg/m³ (2015: 28 µg/m³)a (2020: 27 µg/m³)a	CCME, 2000
WHO	24 hour AQO	25 µg/m³	WHO, 2006
PM_{10}			
Metro Vancouver	***24 hour AQO***	***50 µg/m³***	***Metro Vancouver, 2011***
BC MoE	24 hour AQO	50 µg/m³	BC MoE, 2013
WHO	24 hour AQO	50 µg/m³	WHO, 2006

Notes:

[a] CCME Proposed Air Quality Standards for $PM_{2.5}$ for 2015 and 2020

Table V-61. Chronic Inhalation Exposure Limits for $PM_{2.5}$ and PM_{10}

Source	Exposure Limit	Value	Reference
$PM_{2.5}$			
Metro Vancouver	***Annual Average AQO***	***8 (6) µg/m³***	***Metro Vancouver, 2011***
BC MoE	Annual Average AQO	8 (6) µg/m³	BC MoE, 2013
CCME	Annual Average AQO	(2015: 10 µg/m³)a (2020: 8.8 µg/m³)a	CCME, 2000 CCME, 2004
WHO	Annual Average AQO	10 µg/m³	WHO, 2006
PM_{10}			
Metro Vancouver	***Annual Average AQO***	***20 µg/m³***	***Metro Vancouver, 2011***
BC MoE	Annual Average AQO	20 µg/m³	BC MoE, 2013
WHO	Annual Average AQO	20 µg/m³	WHO, 2006

Notes:

[a] CCME Proposed Air Quality Standards for $PM_{2.5}$ for 2015 and 2020

The Metro Vancouver (2011) $PM_{2.5}$ 24-hour and PM_{10} 24-hour and annual average air quality objectives were based on WHO (2006), which represented the most stringent AQOs from the regulatory agencies searched. WHO (2006) recommended a 24-hour average of 25 µg/m^3 for $PM_{2.5}$, a 24-hour average of 50 µg/m3 and an annual average of 20 µg/m3 for PM_{10}. The Metro Vancouver ambient AQO (annual average) for $PM_{2.5}$ is based on the BC AQO for this parameter. The BC AQO for $PM_{2.5}$ was revised to 8 µg/m^3 (annual average) in 2009 following a review of the scientific literature by SENES Consultants Limited (SENES), on behalf of the BC Lung Association (SENES, 2005). Additionally, the Metro Vancouver AAQO references a planning goal (i.e., future desirable level) of 6 µg/m^3 for $PM_{2.5}$. A review of guidelines from other jurisdictions for $PM_{2.5}$ (annual averages) was conducted by SENES (2005); the AAQO for $PM_{2.5}$ is among the lowest of the available guidelines across Canada and world-wide.

The available data on particulate matter and associated health impacts were compiled and reviewed by SENES (2005), on behalf of the BC Lung Association (report is the basis of the BC AQO), and was largely based on the review of health aspects of air pollution in Europe completed by the WHO in 2004 and formed the basis of the WHO (2006) update of their Air Quality Guidelines for $PM_{2.5}$ and PM_{10}. WHO (2006) summarized that long-term exposure to elevated particulate matter concentrations had the potential to lead to a marked reduction in life expectancy, primarily due to increased cardio-pulmonary and lung cancer mortality. While mortality was the basis on which WHO considered that ambient air quality objectives should be set, increases in lower respiratory symptoms and reduced lung function in children, and chronic obstructive pulmonary disease and reduced lung function in adults, were likely long-term health outcomes associated with exposures to elevated $PM_{2.5}$ concentrations at or near background levels (SENES, 2005; WHO, 2006). WHO noted that epidemiological studies on large populations have not identified a threshold concentration for non-mortality endpoints below which ambient PM has no effect on health (SENES, 2005; WHO, 2006; CCME, 2004). It is important to be aware that a range of thresholds may exist within the population, depending on the type of health effect and the susceptibility of subgroups; noting, however, that no threshold for effects at the population level, other than mortality (as noted above), and for the most sensitive subgroups, has been identified (SENES, 2005). Both WHO (2006) and SENES (2005) have indicated that as threshold levels for effects other than mortality have not been identified, the air quality guidelines have been derived on the basis of mortality and reflect concentrations below which increased mortality outcomes due to exposure to PM air pollution are not expected based on the current body of scientific evidence.

The lowest 24-hour and annual average AQOs for $PM_{2.5}$ (25 µg/m^3 and 8 µg/m^3, respectively) and PM_{10} (50 µg/m^3 and 20 µg/m^3, respectively) were used in the assessment of risks associated with inhalation exposures to particulate matter.

References for Particulate Matter:

BC MoE, 2013. BC Ministry of Environment. Provincial Air Quality Objectives Information Sheet. British Columbia Ambient Air Quality Objectives, Updated August 2013. Available at http://www.bcairquality.ca/reports/pdfs/aqotable.pdf.

CCME. 2000. Canada-Wide Standards for Particulate Matter and Ozone. 2000.

CCME. 2004. Human Health Effects of Find Particulate Matter: Update in Support of the Canada-Wide Standards for Particulate Matter and Ozone. Revised in July 2004.

Metro Vancouver. 2011. 2011 Lower Fraser Valley Air Quality Monitoring Report Summary.

WHO. 2006. WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulphur Dioxide. Global update 2005. Summary of Risk Assessment. WHO Press, World Health Organization, Geneva.

SENES. 2005. Development of Options for a New Provincial $PM_{2.5}$ Air Quality Objective, Summary Report. SENES Consultants Ltd. Prepared for British Columbia Lung Association. December 2005.

V.4.2. Carbon Monoxide

The acute inhalation exposure limits for carbon monoxide are summarized in the following table.

Table V-62. Acute Inhalation Exposure Limits for Carbon Monoxide

Source	Exposure Limit	Value	Reference
Metro Vancouver	1-hour AQO	30,000 µg/m³	Metro Vancouver, 2011
Metro Vancouver	8-hour AQO	10,000 µg/m³	Metro Vancouver, 2011
BC MoE	1-hour AQO	14,300 µg/m³	BC MoE, 2013
BC MoE	8-hour AQO	5,500 µg/m³	BC MoE, 2013
CCME	1-hour AQO	15,000 µg/m³ (Max Desirable) 35,000 µg/m³ (Max Acceptable)	CCME, 1999
CCME	8-hour AQO	6,000 µg/m³ (Max Desirable) 15,000 µg/m³ (Max Acceptable) 20,000 µg/m³ (Max Tolerable)	CCME, 1999
WHO	1-hour AQO	30,000 µg/m³	WHO, 2010
WHO	8-hour AQO	10,000 µg/m^3	WHO, 2010

The WHO has derived one hour and eight hour average exposure guidelines for CO of 30,000 µg/m^3 and 10,000 µg/m^3, respectively. Metro Vancouver (2011) has adopted these values; however, the BC MoE (2013) provided more stringent objectives of 14,300 µg/m^3 and 5,500 µg/m^3 for 1-hour and 8-hour averaging times, respectively.

Following exposure, carbon monoxide can readily diffuse across membranes (e.g., alveolar, capillary, and placental) and absorbed CO binds with haemoglobin in the blood to form carboxyhaemoglobin (COHb) (WHO, 2000). Environmental exposure and endogenous production of CO results in COHb concentrations of approximately 0.5% to 1.5%, while pregnant women can experience COHb levels of up to 2.5%, due to increased endogenous CO production (WHO, 2000). Guidelines for a one hour average exposure of 30,000 µg/m^3 and an eight hour average exposure of 10,000 µg/m^3 were selected by WHO (2000) to ensure a COHb level of 2.5% is not exceeded in sensitive populations (i.e., non-smoking groups with coronary artery disease or foetuses of non-smoking women).

The most conservative and recent BC MoE 1-hour and 8-hour AQOs were used in the HHRA.

The chronic inhalation exposure limits for carbon monoxide are summarized below.

Table V-63. Acute Inhalation Exposure Limits for Carbon Monoxide

Source	Exposure Limit	Value	Reference
WHO	24-hour AQO	7,000 µg/m³	WHO, 2010

To address long-term health risks, WHO (2010) recommended a 24-hour indoor air quality guideline of 7,000 µg/m³ based on epidemiological data that indicate increased emergency room visits for ischemic heart disease, congestive heart failure and cardiovascular disease. WHO (2010) indicates that the latest studies available in 2009, in particular epidemiological studies using very large databases, suggest that the chronic air quality guideline for carbon be positioned below the 8-hour guideline. The WHO (2010) 24-hour air quality guideline was used in the HHRA to assess risks associated with chronic exposures to CO.

References for Carbon Monoxide:

BC MoE, 2013. BC Ministry of Environment. Provincial Air Quality Objectives Information Sheet. British Columbia Ambient Air Quality Objectives, Updated August 2013. Available at http://www.bcairquality.ca/reports/pdfs/aqotable.pdf.

CCME, 1999. Canadian Council of Ministers of the Environment. Canadian national ambient air quality objectives: Process and status. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Metro Vancouver. 2011. 2011 Lower Fraser Valley Air Quality Monitoring Report Summary.

World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91, Copenhagen.

WHO, 2010. World Health Organization Guidelines for Indoor Air Quality, Selected Pollutants. The WHO European Centre for Environment and Health, Bonn Office, ISBN 978 92 890 0213 4. 2010. Available at http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf.

V.4.3. Nitrogen Dioxide

The acute inhalation exposure limits for NO_2 are summarized in the following table.

Table V-64. Acute Inhalation Exposure Limits for Nitrogen Dioxide

Source	Exposure Limit	Value	Reference
Metro Vancouver	1-hour AQO	200 µg/m³	Metro Vancouver, 2011
BC MoE	1-hour AQO	400 µg/m³	BC MoE, 2013
CCME	1-hour AQO	400 µg/m³ (Max Acceptable) 1,000 µg/m³ (Max Tolerable)	CCME, 1999
WHO	1-hour AQO	200 µg/m³	WHO, 2010

The available studies indicate that there is no clearly defined dose-response relationship for health effects caused by NO_2 exposure (WHO, 2000). To derive an AQO for NO_2, WHO applied a 0.5 uncertainty factor to the lowest observed effect level (375 µg/m^3 to 565 µg/m^3) for small changes in lung function and changes in airway responsiveness following NO_2 exposure, to derive a one hour average objective of 200 µg/m^3 (WHO, 2000). WHO (2006) indicates that at concentrations at approximately twice the AQO, asthmatics exhibit small pulmonary function decrements.

The WHO (2000, 2006) 1-hour AQO has been adopted by Metro Vancouver, and is more conservative than the CCME AQO and BC MoE AQO and, consequently, the Metro Vancouver/WHO 1 hour AQO was selected for use in the HHRA.

The chronic inhalation exposure limits for NO_2 are summarized in the following table.

Table V-65. Chronic Inhalation Exposure Limits for Nitrogen Dioxide

Source	Exposure Limit	Value	Reference
Metro Vancouver	Annual Average AQO	40 µg/m³	Metro Vancouver, 2011
BC MoE	Annual Average AQO	60 µg/m³	BC MoE, 2013
CCME	Annual Average AQO	60 µg/m³ (Max Desirable) 100 µg/m³ (Max Acceptable)	CCME, 1999
WHO	Annual Average AQO	40 µg/m³	WHO, 2010

Chronic exposure can result in long-term health effects and therefore, an annual average guideline of 40 µg/m^3 has been proposed (WHO, 2000). This value is based on the potential for direct toxic effects of chronic NO_2 exposure at low concentrations (WHO, 2000). In addition, during epidemiological studies NO_2 is often used as a marker for other combustion-generated pollutants and it is difficult to attribute health effects solely to NO_2 exposure when there are other correlated co-pollutants present; therefore, WHO (2006) indicated that retaining a conservative annual NO_2 guideline is considered prudent and health-protective.

Metro Vancouver (2011) has adopted the WHO annual average AQO of 40 µg/m^3, the most stringent of the available AQO and, consequently, the Metro Vancouver AQO was selected for use in the HHRA. It is noted that the BC MoE (2013) and CCME (1999) AQOs are less conservative and were not selected for use.

References for Nitrogen Dioxide:

BC MoE, 2013. BC Ministry of Environment. Provincial Air Quality Objectives Information Sheet. British Columbia Ambient Air Quality Objectives, Updated August 2013. Available at http://www.bcairquality.ca/reports/pdfs/aqotable.pdf.

CCME, 1999. Canadian Council of Ministers of the Environment. Canadian national ambient air quality objectives: Process and status. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Metro Vancouver. 2011. 2011 Lower Fraser Valley Air Quality Monitoring Report Summary.

WHO, 2000. World Health Organization. Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91, Copenhagen.

WHO, 2006. WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulphur Dioxide. Global Update 2005. Summary of Risk Assessment. WHO Press, World Health Organization, Geneva.

WHO, 2010. World Health Organization Guidelines for Indoor Air Quality, Selected Pollutants. The WHO European Centre for Environment and Health, Bonn Office, ISBN 978 92 890 0213 4. 2010. Available at http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf.

V.4.4. Sulphur Dioxide

The acute inhalation exposure limits for SO_2 are summarized in the following table.

Table V-66. Acute Inhalation Exposure Limits for Sulphur Dioxide

Source	Exposure Limit	Value	Reference
Metro Vancouver	1-hour AQO	450 µg/m³	Metro Vancouver, 2011
Metro Vancouver	24-hour AQO	125 µg/m³	Metro Vancouver, 2011
BC MoE	1-hour AQO	450 µg/m³ (Level A) 900 µg/m³ (Level B) 900 µg/m³ (Level C)	BC MoE, 2013
BC MoE	24-hour AQO	160 µg/m³ (Level A) 260 µg/m³ (Level B) 360 µg/m³ (Level C)	BC MoE, 2013
CCME	1-hour AQO	450 µg/m³ (Max Desirable) 900 µg/m³ (Max Acceptable)	CCME, 1999
CCME	24-hour AQO	150 µg/m³ (Max Desirable) 300 µg/m³ (Max Acceptable) 800 µg/m³ (Max Tolerable)	CCME, 1999
WHO	24-hour AQO	20 µg/m^3	WHO, 2006

WHO (2000) indicates that although individuals with asthma are more sensitive, there is a large range of sensitivity to SO_2 exposure throughout the general population (WHO, 2000). To be protective of the most sensitive sub-populations, guidelines for SO_2 were developed considering the minimum concentrations associated with adverse effects in asthmatics (WHO, 2000). WHO (2006) reports that there is uncertainty in the causality between SO_2 and adverse effects, which may be attributed to other factors such as ultrafine particles or another correlated pollutant. WHO (2006) recommends a more stringent 24-hour guideline (20 µg/m^3) compared to previous WHO values in order to provide greater protection as precautionary approach. The recommended 24-hour guideline was the most stringent found from the regulatory agencies searched. For 1-hour exposure, Metro Vancouver (2011), CCME (1999) and BC MoE (2013) provided an air quality objective of 450 µg/m^3, which represented the most stringent objective available from the above referenced agencies.

The WHO (2006) 24-hour AQO of 20 µg/m^3, and the 1-hour AQO of 450 µg/m^3 were used in the HHRA.

The chronic inhalation exposure limits for SO_2 are summarized in the following table.

Table V-67. Chronic Inhalation Exposure Limits for Sulphur Dioxide

Source	Exposure Limit	Value	Reference
Metro Vancouver	Annual Average AQO	30 µg/m³	Metro Vancouver, 2011
BC MoE	Annual Average AQO	25 µg/m³ (Level A) 50 µg/m³ (Level B) 80 µg/m³ (Level C)	BC MoE, 2013
CCME	Annual Average AQO	30 µg/m³ (Max Desirable) 60 µg/m³ (Max Acceptable)	CCME, 1999

For annual exposure, BC MoE (2013) provided the most stringent objective of 25 µg/m^3 which was be used in the HHRA.

References for Sulphur Dioxide:

BC MoE, 2013. BC Ministry of Environment. Provincial Air Quality Objectives Information Sheet. British Columbia Ambient Air Quality Objectives, Updated August 2013. Available at http://www.bcairquality.ca/reports/pdfs/aqotable.pdf.

CCME, 1999. Canadian Council of Ministers of the Environment. Canadian national ambient air quality objectives: Process and status. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

Metro Vancouver. 2011. 2011 Lower Fraser Valley Air Quality Monitoring Report Summary.

WHO, 2000. World Health Organization. Air Quality Guidelines for Europe, Second Edition. WHO Regional Publications, European Series, No. 91, Copenhagen.

WHO. 2006. WHO Air Quality Guidelines for Particulate Matter, Ozone, Nitrogen Dioxide and Sulphur Dioxide. Global Update 2005. Summary of Risk Assessment. WHO Press, World Health Organization, Geneva.

V.4.5. Diesel Particulate Matter

No acute inhalation exposure limits were available for DPM.

The chronic inhalation exposure limits for DPM are summarized in the following table.

Table V-68. Chronic Inhalation Exposure Limits for Diesel Particulate Matter

Source	Exposure Limit	Value	Reference
US EPA	***RfC***	***5 µg/m^3***	***US EPA, 2003***
OEHHA	***Unit Risk***	***3 x 10^{-4} (µg/m^3)$^{-1}$***	***OEHHA, 2009***

The US EPA (2003) recommended a RfC of 5 µg/m^3 for DPM. This RfC was based on the highest NOAEL of 0.46 mg/m^3 from a number of studies where rats were repeatedly exposed to DPM for chronic exposure durations. The critical health effect was pulmonary inflammation and histopathology. The NOAEL was converted to a human equivalent continuous exposure concentration of 144 µg/m^3 and an uncertainty factor of 30 was applied to account for interspecies extrapolation and inter-individual human variation in sensitivity. The US EPA (2014) recommended RfC of 5 µg/m^3 was used to assess potential non-carcinogenic health risks from inhalation exposures to DPM.

Although diesel emissions are classified as a known carcinogen (IARC) or a likely human carcinogen (USEPA), few agencies, including the USEPA, have derived carcinogenic TRVs (e.g. inhalation unit risk, slope factor) for diesel emissions. The USEPA (2003) indicates that a quantitative estimate of carcinogenic risk from inhalation exposure to diesel emissions has not been derived based on the absence of adequate data to develop a sufficiently confident dose-response relationship from the epidemiological studies. Nevertheless, the OEHHA (2009) recommended an inhalation unit risk of 3 x 10^{-4} (µg/m^3)$^{-1}$ which was based on a meta-analysis of human occupational exposure lung tumour incidence in studies of US railroad workers (OEHHA, 2009). It is noted that the unit risk value is considered to be conservative given that is based on occupational exposure data, and thus the incremental lifetime cancer risk estimate is inherently conservative and likely overestimates risks. In addition, personal communication with Health Canada suggests that the cancer risk value is not widely accepted within Canada and may overestimate the carcinogenic potency of diesel particulates. Notwithstanding the lack of support of the unit risk value by major health agencies such as Health Canada and the US EPA, the OEHHA inhalation UR was used in the HHRA to characterize carcinogenic risks associated with DPM.

References for DPM:

OEHHA, 2009. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 2003. United States Environmental Protection Agency IRIS Summary of Diesel Engine Exhaust (CASRN N.A). Washington, DC. Available at: http://www.epa.gov/iris/subst/0642.htm.

V.4.6. Total Particulate Matter

No acute or chronic inhalation exposure limits were identified for total particulate matter (TPM).

V.5. VOLATILE ORGANIC COMPOUNDS

As described in Section 3.3 of the HHRA, COPCs were classified as either gaseous or non-gaseous. Each of the VOC COPCs were determined to be gaseous and will be present in air, with human exposures to these COPCs limited to inhalation. The bioaccumulation potential of the gaseous COPCs was further evaluated; based on the BC MoE definition of a bioaccumulative substance, of the gaseous COPCs, only hexachlorobenzene was determined to be bioaccumulative; hexachlorobenzene was therefore retained for evaluation in the multi-media assessment.

Based on the above, the inhalation TRVs for the VOC COPCs are presented below, with the oral TRVs for hexachlorobenzene also presented.

V.5.1. Acetaldehyde

V.5.1.1. Inhalation TRVs

The acute inhalation TRVs for acetaldehyde are summarized in the following table.

Table V-69: Acute Inhalation TRVs for Acetaldehyde.

Source	Exposure Limit	Value	Reference
California OEHHA	***1 hour REL***	***470 µg/m^3***	***OEHHA, 2008***
California OEHHA	8 hour REL	300 µg/m^3	OEHHA, 2008

The California OEHHA (2008) recommends an acute (1 hour) inhalation REL for acetaldehyde of 470 µg/m^3. The acute REL was derived based on a study conducted in 2000 in which 61 human asthmatic volunteers were used to determine the concentration of acetaldehyde producing a 20% decrease in Forced Expiratory Volume in one second using ascending doses of aerosolized acetaldehyde solutions. The lower 95% confidence interval of 142 mg/m^3 was used as the LOAEL for the acute REL determination; an uncertainty factor of 300 (10 for use of a LOAEL vs. a NOAEL, and 30 for toxicodynamic intraspecies differences) was applied.

The OEHHA (2008) also recommended an 8-hour REL of 300 µg/m³ for acetaldehyde that is protective of repeated 8-hour exposures. The 8-hour REL is based on a 4-week study in Wistar rats exposed acetaldehyde for 6 hours/day, 5 days/week; the LOAEL for the study was 728 mg/m^3 for degeneration of the olfactory epithelium, and the NOAEL was 273 mg/m³. Through benchmark dose modeling and pharmacokinetic modeling, the human equivalent concentration for the NOAEL was determined to be 242 mg/m³; with adjustments for continuous exposure, a BMC05 of 86.5 mg/m^3 was determined. An uncertainty factor of 300 was applied to calculate the 8-hour REL of 300 µg/m³.

The OEHHA 1-hour REL was used in the HHRA in the estimation of acute human health risks. The 8-hour REL has not been used as it is based on repeat exposures.

The chronic inhalation TRVs for acetaldehyde are summarized in the following table.

Table V-70: Chronic Inhalation TRVs for Acetaldehyde.

Source/Agency	Exposure Limit	Value	Reference
Health Canada	**TC_{05} converted to a Unit Risk**	**5.8 x 10^{-7} (µg/m^3)$^{-1}$**	**CEPA, 1999**
Health Canada	**TC**	**390 µg/m^3**	**CEPA, 1999**
USEPA IRIS	Unit Risk	2.2 × 10^{-6} (µg/m^3)$^{-1}$	USEPA, 1991
USEPA IRIS	RfC	9 µg/m^3	USEPA, 1991
California OEHHA	Chronic REL	140 µg/m^3	OEHHA, 2008

Health Canada (2000) calculated a tumourigenic concentration with 5% response (TC05) of 86,000 µg/m³, which is associated with a 5% increase in nasal tumours in rats; the TC05 is based on a 1986 study where male and female Wistar rats were exposed to 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 the rats. The TC05 was converted to a Risk Specific Concentration (RSC) for 1 x 10^{-5} cancer risk of 17.2 µg/m^3. A unit risk was then calculated as the target cancer risk divided by RSC (i.e., 1 x 10^{-5}/17.2 µg/m^3 = 5.8 x 10^{-7} [µg/m^3]$^{-1}$).

The US EPA considered the same study in their estimation of cancer potency. EPA estimated an inhalation unit risk of 2.2 x 10^{-6} (µg/m^3)$^{-1}$ using low dose extrapolation.

Health Canada and the US EPA both recommend non-cancer inhalation TRVs for acetaldehyde. Health Canada derived a TC of 390 µg/m^3, and the US EPA derived a RfC of 9 µg/m^3. The two TRVs are based on the same study, however, the two agencies used different approaches to derive the TRVs. The US EPA used the NOAEL of 273 from the 1982 study, while Health Canada used the lower 95% confidence limit on a benchmark dose (BMD) for the concentration associated with a 5% increase in the incidence of nasal olfactory epithelial lesions in male

rats. Both agencies adjusted for continuous exposure, however, the US EPA also adjusted to a Human Equivalent Concentration (HEC). In addition, the US EPA used an uncertainty factor of 1000, with a factor of 10 for intraspecies variability, a factor of 10 for the use of a subchronic study, and a factor of ten for combined interspecies variability and database limitations. Health Canada did not use a factor for limitations in the database based on the rationale that the TC that is based on critical effects at the site of entry and is considered protective of systemic effects. In addition, Health Canada did not apply an uncertainty factor for the use of a subchronic study, based on the rationale that the available data did not indicate that the severity of the critical effect would increase with an increased exposure duration.

The OEHHA (2008) also has derived a chronic REL of 140 µg/m³ based on the incidence of olfactory epithelium degeneration following a 4-week exposure in rats.

As indicated, Health Canada and the US EPA based their inhalation TRVs (cancer and non-cancer) on the same studies. Therefore, the unit risk (converted from the Health Canada TC05) of 5.8 x 10^{-7} $(\mu g/m^3)^{-1}$ and TC (390 $\mu g/m^3$) were used to assess chronic inhalation exposures to acetaldehyde.

References for Acetaldehyde:

CEPA, 1999. Canadian Environmental Protection Agency. Priority Substances List Report, Acetaldehyde. May 2000.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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, 2009. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 1991. United States Environmental Protection Agency. IRIS Summary of Acetaldehyde (CASRN 75-07-0). Available at: http://www.epa.gov/iris/subst/0290.htm.

V.5.2. Acrolein

The acute inhalation TRVs for acrolein are summarized in the following table.

Table V-71: Acute Inhalation TRVs for Acrolein.

Source	Exposure Limit	Value	Reference
ATSDR	Acute MRL	6.9 µg/m^3	ATSDR, 2007
OEHHA	**1 hour REL**	**2.5 µg/m^3**	**OEHHA, 2008**
OEHHA	8 hour REL	0.7 µg/m^3	OEHHA, 2008
TCEQ	**Acute ReV (1 hour)**	**11 µg/m^3**	**TCEQ, 2014**

ATSDR derived an acute inhalation MRL of 6.9 µg/m^3 (0.003 ppm) based on a LOAEL of 0.3 ppm for nasal and throat irritation and decreased respiratory rate in volunteers exposed to acrolein for 60 minutes (Weber-Tschopp et al. 1977). An uncertainty factor of 100 (10 for use of a LOAEL versus a NOAEL, and 10 for intraspecies variability) was applied to the LOAEL.

The OEHHA (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 where human volunteers were exposed to arolein. In the first study (in 1960) 36 volunteers were exposed to varying concentrations of acrolein for 5 minutes using respirators and ensuring that only their eyes were exposed to acrolein. A LOAEL of 0.06 ppm (140 g/m³) was determined. An uncertainty factor of 60 (6 for use of a LOAEL versus a NOAEL and for a mild adverse effect, and 10 for intraspecies variability) was applied to the LOAEL to derive the acute REL of 2.3 µg/m^3. In the second study (in 1977), three groups of volunteers were exposed to varying concentrations of acrolein for varying durations. Subjective eye and nasal irritation were reported by the volunteers and eye-blink and respiratory rates were measured during the exposures. A LOAEL of 0.07 ppm was determined for subjective ocular irritation. An uncertainty factor of 60 (6 for use of a LOAEL versus a NOAEL and for a mild adverse effect, and 10 for intraspecies variability) was applied to the LOAEL to derive the acute MRL of 2.7 µg/m³.

The OEHHA (2008) also developed an 8-hour REL based on a 65-day study where Fischer 344 (F344) rats were exposed to varying concentrations of acrolein for 6 hours/day, 5 days/week over the 65-day exposure period.

Although not identified as a preferred source in the TRV selection hierarchy, the Texas Commission on Environmental Quality (TCEQ) inhalation TRVs for arcolein were reviewed; the TCEQ has recently (2014) conducted a comprehensive review for acrolein. Based on their review, the TCEQ has recommended an acute (1 hour) inhalation reference value (ReV) for acrolein of 11 µg/m^3. The acute ReV is based on the 1977 study discussed above where human subjects (three groups of males and females) were exposed for 1 hour to varying

concentrations of acrolein. The TCEQ identified a LOAEL for the study of 3 ppm based on eye, nose and throat irritation, and decreased respiratory rate. An uncertainty factor of 63, 10 for intraspecies variability and 6.3 for use of a LOAEL (based on mild irritation, with a < 10% decrease in respiratory rate), was applied to the LOAEL to derive the acute ReV of 4.8 ppm, or 11 µg/m^3. Based on the recent and very comprehensive nature of the review conducted by the TCEQ, and as the acute TRV is based on human exposure data, the TCEQ acute ReV was considered in the HHRA.

Both the OEHHA acute (1 hour) REL of 2.5 µg/m³ and the TCEQ acute ReV of 11 µg/m³ were used in the assessment of acute inhalation exposures to acrolein. The acute risk estimates based on the TRVs from the two sources will be discussed and compared in Section 6.0 of the HHRA.

The identified chronic inhalation TRVs for acrolein are summarized in the following table.

Table V-72: Chronic Inhalation TRVs for Acrolein.

Source/Agency	Exposure Limit	Value	Reference
Health Canada	TC	0.4 µg/m^3	CEPA, 2000
USEPA IRIS	RfC	0.02 µg/m^3	USEPA, 2003
OEHHA	**Chronic REL**	**0.35 µg/m^3**	**OEHHA, 2008**
TCEQ	**Chronic ReV**	**2.7 µg/m^3**	**TCEQ, 2014**

Health Canada derived a tolerable concentration (TC) of 0.4 µg/m^3 based on a benchmark concentration (BMC05) of 0.14 mg/m^3 (adjusted to 0.035 mg/m^3 for continuous exposure) determined for necrosis, thickening, and desquamation in nasal respiratory epithelium of rats exposed to acrolein for 3 days (1996 study). Health Canada indicated that the 1996 study is the most sensitive of the inhalation studies, and that although the study was short-term, the effects observed in the nasal epithelium were similar to those observed in longer-term bioassays conducted at similar concentrations. An uncertainty factor of 100 (10 for interspecies variability and 10 for intraspecies variability) was applied to derive the TC. Health Canada did not apply an additional uncertainty factor for lack of a chronic study given that the available data did not indicate that the severity of the critical effects increased with duration of exposure.

The US EPA recommended a RfC for acrolein of 0.02 µg/m^3 based on a LOAEL of 0.9 mg/m^3 (equivalent to a human equivalent concentration of 0.02 mg/m^3) for nasal lesions in rats reported in a subchronic inhalation study (1978 study). The US EPA did consider the 1996 identified by Health Canada as the critical study in their review, but identified the 1978 study as the critical study noting the higher number of test animals, the longer exposure

duration and the testing of both genders for multiple species. The US EPA applied an uncertainty factor of 1000 to derive the RfC of 0.02 µg/m^3.

The OEHHA (2008) has derived a chronic REL of 0.35 µg/m³ based on the incidence of nasal lesions in a 2008 subchronic rat inhalation study. A NOAEL for nasal epithelial lesions was determined to be 0.2 ppm, which was adjusted for continuous exposure and converted to a HEC of 0.03 ppm (70 µg/m³). An uncertainty factor of 200 (for intraspecies variability, intraspecies variability, use of a subchronic study, toxicodynamic differences and toxicokinetic differences) was applied to the NOAELHEC to derive the chronic REL of 0.35 µg/m³.

As indicated, the TCEQ has conducted a recent (2014) and comprehensive toxicological review for acrolein. Based on their review, the TCEQ have recommended a chronic ReV of 2.7 µg/m³. The chronic ReV is based on the 2008 study identified by the OEHHA as the key study. The TCEQ identified a NOAEL of 0.2 ppm for nasal epithelial hyperplasia and squamous metaplasia, and a LOAEL of 0.6 ppm for hyperplasia of the nasal cavity, septum and larynx. The NOAEL of 0.2 ppm was selected as the POD, with an adjustment to account for continuous exposure and converted to a POD_{HEC} of 0.035 ppm. An uncertainty factor of 30 was applied to account for interspecies differences (3), and intraspecies variability (10), resulting in a chronic ReV of 1.2 ppb, or 2.7 µg/m³. The TCEQ determined a uncertainty factor of 3 for extrapolation from animals to humans as a regional gas dose ratio was applied in the estimation of the POD_{HEC} to account for toxicokinetic differences. The uncertainty factor of 3 therefore applies to toxicodynamic differences. The factor of 3 is further considered to be conservative as rats are obligatory nose breathers, unlike humans, making rats more vulnerable to nasal irriation (TCEQ, 2014). Based on the comprehensive nature of the toxicological assessment conducted by the TCEQ and the thorough evaluation of appropriate uncertainty factors, as well as the fact that the 2008 study would not have been available to Health Canada at the time of the derivation of their TC for acrolein, the TCEQ chronic ReV was considered in the HHRA.

The OEHHA chronic REL of 0.35 µg/m³ and the TCEQ chronic ReV of 2.7 µg/m³ were both selected for use in the assessment of chronic exposures to acrolein. The chronic risk estimates based on the TRVs from the two sources will be discussed and compared in Section 6.0 of the HHRA.

<u>References for Acrolein:</u>

CEPA, 1999. Canadian Environmental Protection Agency. Priority Substances List Report, Acrolein. May 2000.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

TCEQ, 2014. Texas Commission on Environmental Quality. Acrolein. CAS Registry Number: 107-02-8. Final Development Support Document. March 2014. Available at: http://www.tceq.com/assets/public/implementation/tox/dsd/final/mar2014/acrolein_107-02-8_revised.pdfUS EPA, 2003. United States Environmental Protection Agency. IRIS Summary of Acetaldehyde (CASRN 107-02-8). Available at: http://www.epa.gov/iris/subst/0364.htm.

V.5.3. Benzene

The acute inhalation TRVs for benzene are summarized in the following table.

Table V-74: Acute Inhalation TRVs for Benzene.

Source	Exposure Limit	Value	Reference
ATSDR	Acute (6 hour) MRL	29 µg/m^3	ATSDR, 2007b
OEHHA	**8-hour REL**	**27 µg/m^3**	**OEHHA, 2014**
OEHHA	24-hour REL	3 µg/m^3	OEHHA, 2014
US EPA	**Sub-chronic PPRTV**	**80 µg/m^3**	**US EPA, 2009**

ATSDR has derived an acute inhalation MRL of 29 µg/m^3 (0.009 ppm) for benzene based on a LOAEL for immunological effects of 10.2 ppm determined for mice exposed to benzene for 6 hours/day for 6 consecutive days (Rozen et al. 1984). The LOAEL of 10.2 ppm was adjusted to continuous exposure and converted to a human equivalent concentration (HEC) (LOAELHEC=2.55 ppm). An uncertainty factor of 300 (10 for use of a LOAEL versus a NOAEL, 3 for extrapolation from animals to humans using dosimetric conversion, and 10 for intraspecies variability) was applied. It is stressed that the ATSDR value was developed for continuous exposures that may last up to 14 days duration,

The OEHHA recently (2014) revised their inhalation RELs for benzene. The OEHHA derived an 8-hour REL for benzene of 27 µg/m^3; this value supercedes their previously recommended 6-hour REL of 1300 µg/m^3 . The 8-hour REL is based on developmental hematotoxicity observed in fetal and neonatal mice (1988 study). The study LOAEL was identified as5 ppm (16 mg/m^3), with an uncertainty factor of 600 (3 for the use of a LOAEL, 6 for interspecies variability, and 30 each for intraspecies variability).

The OEHHA also recommended a 24-hour inhalation REL of 3 µg/m^3. The 24-hour REL was set equivalent to the chronic REL (derivation discussed below). Because it is not based on acute exposures, the 24-hour REL was not further considered.

The US EPA (2009) recommends a subchronic RfC for benzene of 80 µg/m^3 based on a 1996 human study that was identified as the key study for the derivation of the chronic inhalation RfC. The US EPA would have had access to the 1984 and 1988 rat and mouse studies identified as key studies by the ATSDR and the OEHHA, respectively, but determined that the 1996 human exposure study was more appropriate for use. On this basis, and as the US EPA (2009) subchronic RfC is based on human versus animal data, it was selected for use in the HHRA. It is noted that use of a subchronic RfC to characterize acute exposures is considered conservative.

The chronic inhalation TRVs for benzene are summarized in the following table.

Table V-75: Chronic Inhalation TRVs for Benzene.

Source	Exposure Limit	Value	Reference
Health Canada	**Unit Risk**	**3.3 x 10^{-6} (µg/m^3)$^{-1}$**	**Health Canada, 2010**
US EPA	**RfC**	**30 µg/m^3**	**US EPA, 2003**
US EPA	Unit Risk	2.2 × 10^{-6} to 7.8 × 10^{-6} (µg/m³)$^{-1}$	US EPA, 2000
WHO	Unit Risk (calculated based on RSC)	5.9 × 10^{-6} (µg/m³)$^{-1}$	WHO, 2010
ATSDR	MRL	9.6 µg/m^3	ATSDR, 2007
OEHHA	Chronic REL	3 µg/m^3	OEHHA, 2014
OEHHA	Unit Risk	2.9 x10^{-5} (µg/m^3)$^{-1}$	OEHHA, 2009
RIVM	Unit Risk (calculated based on CR)	5 × 10^{-6} (µg/m³)$^{-1}$	RIVM, 2001

For evaluation of carcinogenic risks from inhalation exposures to benzene, Health Canada (2010) recommended an inhalation unit risk value of 3.3 x 10^{-6} (µg/m^3)$^{-1}$ for benzene based on the incidence of leukemia following occupational exposure in studies conducted in 1987, 1981.

The US EPA (2000) presents a range of potential carcinogenic risks from inhalation of benzene, with a recommended unit risk of 2.2 × 10^{-6} to 7.8 × 10^{-6} (µg/m³)$^{-1}$. The unit risk was derived based on the results of the same studies identified by Health Canada.

The WHO derived a risk specific concentration for a 1 in 100,000 risk level of 1.7 µg/m^3 based on the results of the same occupational exposure studies discussed above. The RSC has been converted to a unit risk by dividing the target risk level of 1x10^{-5} by the RSC of 1.7 µg/m^3, resulting in a unit risk estimate of 5.9 × 10^{-6} (µg/m³)$^{-1}$.

The OEHHA (2009) derived a unit risk estimate of 2.9 x10^{-5} (µg/m^3)$^{-1}$ based on epidemiological studies of Chinese workers, which were indicated by the US EPA to have methodological issues (poor exposure characterization, co-exposure to other agents, etc). The unit risk was therefore not further considered.

RIVM also calculated a cancer potency factor based on the results of the same studies used by Health Canada. RIVM estimated a CR for a 1 x 10^{-4} cancer risk of 20 µg/m^3. This value was to the concentration at the 1 in 100,000 (E-5) risk level by dividing by 10 (resulting in 2 µg/m^3), which was converted to a unit risk estimate of 5 x × 10^{-6} (µg/m³)$^{-1}$.

For evaluation of non-cancer risks from inhalation exposures to benzene, US EPA (2003) recommended a RfC of 30 µg/m^3. This RfC was based on a BMCL of 8.2 mg/m^3 (adjusted for 24-hour continuous exposures) for decreased lymphocyte count in workers occupationally exposed to benzene (Rothman et al., 1996) and then application of a 300-fold uncertainty factor.

ATSDR has derived a chronic inhalation MRL of 9.6 µg/m^3 (0.003 ppm) for benzene based on the results of benchmark dose (BMD) modeling of B cell counts in workers of shoe manufacturing industries in Tianjin, China (2004 study). The benchmark concentration (BMC) was adjusted for continuous exposure, and an uncertainty of 10 for intraspecies variability was applied. Although the ATSDR chronic inhalation MRL is more conservative than the US EPA RfC and based on a study not available when US EPA developed their value, it is recognized that the ATSDR does not develop unit risk values and instead their MRLs incorporate additional factors for cancer protection. These additional uncertainty factors are considered to be unnecessary since the HHRA is using the unit risk factor approach in separate calculations. Consequently, the ATSDR MRL was not selected for use but, nevertheless, is considered in the sensitivity analysis.

The OEHHA recently (2014) recommended a chronic inhalation REL for non-cancer effects of 3 µg/m^3; the chronic REL is based on a 2004 occupational exposure study in Chinese shoe workers. The workers were exposed to benzene via inhalation for 8 hours a day, 6 days a week. The OEHHA identified a LOAEL of 0.57 ± 0.24 ppm (1.86 ± 0.78 mg/m^3) for decreased peripheral blood cell counts. The OEHHA determined an HEC of 0.665 mg/m^3 and applied a cumulative uncertainty factor of 200 (3 for the use of what was considered sub-chronic data (8-12% of expected lifetime) and 60 for intraspecies variability). It is acknowledged that the OEHHA REL is based on a study that was published following the US EPA's last review of the IRIS chronic RfC for benzene. It is

however noted that the US EPA has indicated that the previous studies evaluating benzene exposures in Chinese shoe manufacturers have methodological issues (poor exposure characterization, coexposure to other agents, data quality). In addition, as discussed above, the US EPA (2009) derived a subchronic TRV for benzene, and although they would have had access to the 2004 study at that time (2009), they based the subchronic RfC on the same 1996 study that was the basis of the chronic TRV. It is also noted that given the date of the last revision to the US EPA RfC for benzene (2003) compared to the date of the 2004 Chinese shoe makers study, it is possible that the US EPA had access to the data from the study at the time of their review. Based on the above, and given the TRV hierarchy discussed in earlier sections of the HHRA, the OEHHA chronic REL was not selected for use in the HHRA.

The Health Canada unit risk of 3.3×10^{-6} $(\mu g/m^3)^{-1}$ and the US EPA RfC of 30 $\mu g/m^3$ were used in the assessment of cancer non-cancer risks associated with the inhalation of benzene, respectively. It is noted that use of the OEHHA REL of 3 $\mu g/m^3$ on the results of the HHRA will be included as part of the sensitivity analysis.

References for Benzene:

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

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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, 2009. California Office of Environmental Health Hazard Assessment. 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, 2014. California Office of Environmental Health Hazard Assessment. Technical Support Document for Non-Cancer RELs. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Branch. June 2014. Available at http://www.oehha.ca.gov/air/hot_spots/2008/AppendixD1_final.pdf#page=139.

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 2000 and 2003. United States Environmental Protection Agency. IRIS Summary of Benzene (CASRN 71-43-2). Available at: http://www.epa.gov/iris/subst/0276.htm.

U.S. EPA. 2009. Provisional Peer Reviewed Toxicity Values for Benzene (CASRN 71-43-2). 9-29-2005.

V.5.4. 1,3-Butadiene

The acute inhalation TRVs for 1,3-butadiene are summarized in the following table.

Table V-76: Acute Inhalation TRVs for 1,3-Butadiene.

Source	Exposure Limit	Value	Reference
OEHHA	**1 hour REL**	**660 µg/m^3**	**OEHHA, 2013**
OEHHA	8 hour REL	9 µg/m^3	OEHHA, 2013

The OEHHA derived 1 hour REL of 660 µg/m^3 (0.297 ppm) based on decreased male fetal weight at gestation day 18 in mice that were exposed to 1,3-butadiene for 6 hours on gestation days 1 through 15. A NOAELHEC of 29.7 ppm was determined for the study. An uncertainty factor of 100 (for toxicokinetic and toxicodynamic differences) was applied to derive the REL.

The OEHHA also derived an 8 hour REL for 1,3 butadiene of 9 µg/m^3 (0.0042 ppm). The 8 hour REL is based on ovarian atrophy observed in mice exposed to 1,3-butadiene for 6 hour/day, 5 days/week for 9 to 24 months (NTP, 1993). The OEHHA estimated a BMCL05, and applied an uncertainty factor of 300 for inter- and intraspecies variability.

The 1-hour REL was selected for use in the HHRA. The 8-hour REL will not be used as it is not based on acute exposures.

The chronic inhalation TRVs for 1,3-butadiene are summarized in the following table.

Table V-77: Chronic Inhalation TRVs for 1,3-Butadiene.

Source	Exposure Limit	Value	Reference
Health Canada	**Unit Risk (Converted from a TC01)**	**5.9×10^{-6} $(\mu g/m^3)^{-1}$**	**CEPA, 1999**
US EPA	RfC	2 $\mu g/m^3$	US EPA, 2002
US EPA	Unit Risk	3.0×10^{-5} $(\mu g/m^3)^{-1}$	US EPA, 2002
OEHHA	Chronic REL	2 $\mu g/m^3$	OEHHA, 2013

Health Canada calculated a tumourigenic concentration (TC01) of 1,700 $\mu g/m^3$, which represents the concentration of 1,3-butadiene associated with a 1% excess (i.e., 1 in 100) probability of dying from leukemia. This potency estimate is based on an epidemiological investigation of the association between exposure to 1,3-butadiene in the styrene-butadiene rubber industry and leukemia conducted in 1995. The TC01 was divided by 1,000 to calculate a RSC for a 1×10^{-5} cancer risk, resulting in an RSC of 1.7 $\mu g/m^3$. A unit risk was then calculated as the target cancer risk divided by RSC (i.e., $1 \times 10^{-5}/1.7$ $\mu g/m^3 = 5.9 \times 10^{-6}$ $(\mu g/m^3)^{-1}$).

The US EPA (2014) recommended an UR of 3.0×10^{-5} $(\mu g/m^3)^{-1}$ for the protection of carcinogenic effects (leukemia). Using the Health Canada regression analyses of the same study used by Health Canada, EPA derived an inhalation the unit risk value.

The US EPA (2014) recommended a RfC of 2 $\mu g/m^3$ based on ovarian atrophy observed in mice exposed to 1,3-butadiene for up to two years (1993 study). The EPA RfC was derived from a BMCL10(HEC) of 0.88 ppm and an uncertainty factor of 1000 (3 each for interspecies variability and an incomplete database, and 10 each for intraspecies variability and extrapolation to a level below the 10% effect level).

The OEHHA also recommends a chronic REL of 2 $\mu g/m^3$ based on ovarian atrophy observed in mice exposed to 1,3-butadiene for up to two years in the 1993 study.

The Health Canada unit risk (based on the TC01) of 5.9×10^{-6} $(\mu g/m^3)^{-1}$ and the US EPA RfC (which is equivalent to the OEHHA REL) of 2 $\mu g/m^3$ have been selected for use in the HHRA. It is noted that the US EPA unit risk is based on the same study (Delzell et al., 1995) as the Health Canada value.

References for 1,3-Butadiene:

CEPA, 1999. Canadian Environmental Protection Agency. Priority Substances List Report, 1,3-Butadiene. Revised August 2000.

OEHHA, 2013. 1,3-Butadiene Reference Exposure Levels, July, 2013. Available at http://www.oehha.ca.gov/air/chronic_rels/pdf/072613bentCREL.pdf.

US EPA, 2002. IRIS Summary of 1,3-Butadiene (CASRN 106-99-0). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0139.htm.

V.5.5. Ethylbenzene

The acute inhalation TRVs for ethylbenzene are summarized in the following table.

Table V-78: Acute Inhalation TRVs for Ethylbenzene.

Source	Exposure Limit	Value	Reference
ATSDR	**Acute MRL**	**21,700 µg/m^3**	**ATSDR, 2000**

The ATSDR derived an acute inhalation MRL of 21,700 µg/m^3 (5 ppm) for ethylbenzene based on the results of a study conducted in 2000 where auditory threshold shifts were observed in rats exposed to ethylbenzene 8 hours/day for 5 days. A HEC of the benchmark concentration (BMCL) was estimated; the BMCLHEC of 154.26 ppm was divided by an uncertainty factor of 30 (3 for animal to human extrapolation with dosimetric adjustment and 10 for human variability) to derive the acute inhalation MRL.

The ATSDR acute MRL of 21,700 µg/m^3 was selected for use in the HHRA.

The chronic inhalation TRVs for ethylbenzene are summarized in the following table.

Table V-79: Chronic Inhalation TRVs for Ethylbenzene.

Source	Exposure Limit	Value	Reference
Health Canada	TC	1,000 µg/m^3	Health Canada, 2010
US EPA	RfC	1,000 µg/m^3	US EPA, 1991
ATSDR	**MRL**	**260 µg/m^3**	**ATSDR, 2010**
RIVM	TCA	770 µg/m^3	RIVM, 2001

For evaluation of inhalation exposures to ethylbenzene, Health Canada (2010) recommended a TC of 1,000 µg/m^3, which was based on the US EPA (1991) recommended RfC of the same value. The RfC was based

on a NOAEL of 434 mg/m^3 for developmental effects in rats and rabbits and the application of a 300-fold uncertainty factor (based on studies conducted in 1981).

The ATSDR and RIVM both derived inhalation exposure limits based on a series of inhalation studies conducted in 1996 and 1999; these studies were not available at the time the US EPA conducted their assessment of ethylbenzene.

Using a 2-year 1999 study, ATSDR derived a chronic-duration inhalation MRL of 260 µg/m^3 (0.06 ppm) based on a LOAELHEC of 17.45 ppm (75.7 mg/m^3) for significant increases in the severity of nephropathy in female rats. An uncertainty factor of 300 (a factor of 10 for use of a LOAEL, a factor of 10 for intraspecies variability, and a factor of 3 for extrapolation from animals to humans with dosimetric adjustment).

Using a 13-week 1996 study, RIVM derived a TCA of 0.77 mg/m^3 based on a duration-adjusted NOAEC of 77 mg/m^3 for liver and kidney effects observed in rats and mice, and an uncertainty factor of 100 (a factor of 10 each for intra- and interspecies variability).

After reviewing the information summarized above, the ATSDR chronic inhalation MRL of 260 µg/m^3 was selected for the characterization of chronic inhalation exposures. The ATSDR MRL is based on data that was not available at the time of the US EPA assessment, and the ATSDR MRL is based on a chronic study, whereas the RIVM TCA is based on a subchronic study.

References for Ethylbenzene:

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

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1991. IRIS Summary of Ethylbenzene (CASRN 100-41-4). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0051.htm.

V.5.6. Ethylene

No acute or chronic inhalation TRVs were identified for ethylene.

V.5.7. Formaldehyde

The acute inhalation TRVs for formaldehyde are summarized in the following table.

Table V-80: Acute Inhalation TRVs for Formaldehyde.

Source	Exposure Limit	Value	Reference
Health Canada	Short-term exposure limit (1 hour)	123 µg/m^3	Health Canada, 2006
WHO	**30-min AQG**	**100 µg/m^3**	**WHO, 2010**
ATSDR	Acute MRL	50 µg/m^3	ATSDR, 1999
OEHHA	1 hour REL	55 µg/m^3	OEHHA, 2008
OEHHA	8 hour REL	9 µg/m^3	OEHHA, 2008

Health Canada has derived a short-term (1 hour) and a long-term (8 hour) residential indoor air quality guidelines for formaldehyde of 123 µg/m^3 and 50 µg/m^3, respectively. The 1 hour exposure limit is based on the NOAEL and LOAEL for eye irritation of 615 µg/m^3 and 1,230 µg/m^3, respectively following acute exposures in a study conducted in 1993. The 1 hour limit represents one fifth of the NOAEL and one tenth of the LOAEL determined for the 1993 study. The 8 hour exposure limit was determined as the lower end of the exposure category associated with no significant increase of asthma hospitalization for children following formaldehyde exposures in a study conducted in 2002.

The WHO (2010) recommends a 30-minute air quality guideline of 100 µg/m³ based a review of the available toxicological literature, and a NOAEL of 0.6 mg/m^3 for the eye blink response adjusted using an assessment factor of 5 (derived for sensory irritation) thresholds, leading to a value of 120 µg/m³, which was rounded down to 100 µg/m³. WHO recommends this air quality guideline to prevent sensory irritation in the general population. WHO (2010) also reported that the available literature indicates that associations between exposure to formaldehyde and nasopharyngeal malignancies and leukaemia in humans are limited to high exposure concentrations; with the 30-minute air quality guideline therefore being protective of chronic effects, including cancer.

The ATSDR (2011) derived 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 in occupationally exposed patients with skin hypersensitivity to formaldehyde. A total uncertainty factor of 9 was applied to the LOAEL, with a factor of three for intraspecies variability, and a factor of 3 for use of a minimal LOAEL. ATSDR noted that a factor of 3 for human variability was determined as the effects were observed in subjects with demonstrated sensitivity to formaldehyde.

The OEHHA (2008) derived 1-hour and 8-hour RELs for formaldehyde. The acute 1-hour REL of 55 µg/m³ is based on a study involving 19 healthy non-smokers exposed to formaldehyde for a single 3-hour period. A NOAEL of 0.5 ppm and a LOAEL of 1 ppm were determined for ocular irritation. Benchmark dose modelling was conducted, and the BMCL05 was determined to be about 0.44 ppm (530 µg/m³). An uncertainty factor of 10 for intraspecies variability was applied to the BMCL05.

The 8-hour REL derived by the OEHHA was not based on acute exposures; the REL was derived based on long-term occupational studies and therefore has not been further considered.

The WHO (2010) 30 minute AQG for formaldehyde was selected for use in the HHRA. The derivation of the WHO guideline is thoroughly documented, and is considered to be the most defensible exposure limit.

The chronic inhalation TRVs for formaldehyde are summarized in the following table.

Table V-81: Chronic Inhalation TRVs for Formaldehyde.

Source	Exposure Limit	Value	Reference
Health Canada	**Long-term exposure limit (as an 8 hour average concentration)**	**50 µg/m^3**	**Health Canada, 2006**
US EPA	Unit Risk	1.3 x 10^{-5} (µg/m^3)$^{-1}$	US EPA, 1991
WHO	30-min AQG (indicated to be protective of chronic effects, including cancer)	100 µg/m^3	WHO, 2010
ATSDR	Chronic MRL	9.8 µg/m^3	ATSDR, 1999
OEHHA	Chronic REL	9 µg/m^3	OEHHA, 2008

In a review of the toxicological literature, Health Canada (2006) concluded that an 8 hour air concentration of 50 µg/m^3 would be protective of chronic health effects and would also be associated with negligible cancer risks since it is below the level associated with respiratory tract irritation.

US EPA (1991) provided an inhalation unit risk of 1.3 x 10^{-5} (µg/m^3)$^{-1}$. The unit risk was based on the additional risk of squamous cell carcinomas in rats following inhalation exposure (Kerns et al., 1983).

As indicated above, the WHO (2010) recommends a 30-minute air quality guideline of 100 µg/m³ based a review of the available toxicological literature and has indicated that the 30-minute air quality guideline is protective of chronic effects, including cancer.

The ATSDR recommends a chronic inhalation MRL of 9.8 µg/m^3 (0.008 ppm) based on a minimal LOAEL of 0.24 ppm for histological evidence of mild damage to the nasal epithelial tissue in workers exposed to formaldehyde (1989 study). To derive the MRL, the minimal LOAEL was divided by an uncertainty factor of 30 (a factor of 3 for the use of a minimal LOAEL and a factor of 10 for intraspecies variability).

The OEHHA (2008) derived a chronic REL of 9 µg/m³ is based on eye, nasal and respiratory irritation in exposed workers in a study conducted in 1992. An uncertainty factor of 10 (for intraspecies variability) was applied to the NOAEL (0.09 mg/m³), resulting in the REL of 9 µg/m³.

Based on the previously described toxicological hierarchy, the Health Canada 8 hour guideline of 50 µg/m³ was considered to be protective of long term health effects and was subsequently used in the HHRA to assess risks from chronic exposures.

References for Formaldehyde:

ATSDR, 1999. Toxicological Profile for Formaldehyde. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. 1999.

CEPA, 1999. Canadian Environmental Protection Agency. Priority Substances List Report, Formaldehyde. Report dated February 2001.

Health Canada. 2006. Residential Indoor Air Quality Guideline: Formaldehyde. Health Canada, Ottawa, ON. Available at: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/air/formaldehyde-eng.pdf.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 1991. IRIS Summary of Formaldehyde (CASRN 50-0-0). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0419.htm.

V.5.8. Hexachlorobenzene

V.5.81. Inhalation TRVs

No acute inhalation TRVs were identified for hexachlorobenzene.

The chronic inhalation TRVs for hexachlorobenzene are summarized in the following table.

Table V-82: Chronic Inhalation TRVs for Hexachlorobenzene.

Source	Exposure Limit	Value	Reference
US EPA	**Inhalation UR**	**4.6 x 10^{-4} (µg/m^3)$^{-1}$**	**US EPA, 1996**
RIVM	pCRinhal (converted to Inhalation UR)	0.75 µg/m^3 (1.3 x 10^{-4} [µg/m^3]$^{-1}$)	RIVM, 2001
California OEHHA	Inhalation UR	5.1 x 10^{-4} (µg/m^3)$^{-1}$	OEHHA, 2009

US EPA provided an inhalation UR of 4.6 x 10^{-4} (µg/m^3)$^{-1}$ for hexachlorobenzene, which was last reviewed in 1996. The UR was based on route-to-route extrapolation from the US EPA's recommended oral slope factor; the oral slope was derived based on the results of a 1986 study where rats were fed hexachlorobenzene in their diets for 90 days prior to mating until 21 days after parturition. A NOAEL of 0.08 mg/kg bw/d was established based on liver effects, and an uncertainty factor of 100 was applied to account for inter- and intra species variability.

RIVM (2001) provided a provisional CRinhal of 0.75 µg/m^3, based on a 1:104 lifetime excess cancer risk, for hexachlorobenzene. The CR can be converted to a unit risk by dividing the lifetime excess cancer risk by the CRinhal, resulting in a unit risk of 1.3 x 10^{-4} (µg/m^3)$^{-1}$. The CRinhal was based on route-to-route extrapolation from the CRoral, was based on a 1985 chronic rat study.

OEHHA (2009) recommended an inhalation unit risk of 5.1 x 10^{-4} (µg/m^3)$^{-1}$ for hexachlorobenzene. The unit risk was based on the oral slope factor derived by OEHHA; the oral slope factor was based on a linearized multistage model applied to the dose-response data for the induction of hepatomas in male hamsters (1977 study), heptocellular carcinomas (1983 study) and pheochromocytomas (1983 study and the same 1985 study considered by RIVM) in female rats.

The US EPA unit of 4.6 x 10^{-4} (µg/m^3)$^{-1}$ was selected for use in the HHRA. The US EPA would have had access to each of the studies considered by the OEHHA in the derivation of their unit risk estimate, and as indicated previously, US EPA sources were given priority. The US EPA unit risk will be used to assess carcinogenic risks following chronic inhalation exposure to hexachlorobenzene.

V.5.8.2. Oral TRVs

The oral TRVs for hexachlorobenzene are summarized in the following table.

Table V-83: Oral TRVs for Hexachlorobenzene.

Source	Exposure Limit	Value	Reference
Health Canada	**TDI**	**0.5 µg/kg bw/d**	**Health Canada, 1993**
Health Canada	**pTDI**	**0.27 µg/kg bw/d**	**Health Canada, 2013**
US EPA	RfD	0.8 µg/kg bw/d	US EPA, 1991
ATSDR	Chronic oral MRL	0.07 µg/kg bw/d	ATSDR, 2013
Health Canada	TD05 (converted to an oral slope factor)	60 µg/kg bw/d (8.3×10^{-4} [µg/kg bw/d]$^{-1}$)	Health Canada, 1993
US EPA	**Oral SF**	**1.6×10^{-3} (µg/kg bw/d)$^{-1}$**	**US EPA, 1996**
RIVM	CRoral (converted to SF)	0.16 µg/kg bw/d (6.3×10^{-4} [µg/kg bw/d]$^{-1}$)	RIVM, 2001
OEHHA	Oral SF	1.8×10^{-3} (µg/kg bw/d)$^{-1}$	OEHHA, 2009

Health Canada (1993) recommended a TDI of 0.5 µg/kg bw/d for hexachlorobenzene. The TDI was based on several types of effects reported in several studies and the lowest NOEL of 0.05 mg/kg bw/d was selected based primarily on hepatic effects in two species as reported in diet exposure studies including a 1978 subchronic study in pigs, a 1985 chronic study in rats, and a 1975/1976 chronic study in rats. An uncertainty factor of 1000 was applied to the NOEL to account for inter and intraspecies variability and evidence of carcinogenicity.

Health Canada (2013) CHHAD recommended a provisional TDI of 0.27 µg/kg bw/d for hexachlorobenzene. The details of the derivation of the provisional TDI were not available for review.

US EPA recommended a RfD of 0.8 µg/kg bw/d for hexachlorobenzene, which was last reviewed by the agency in 1991. The RfD was based on the above mentioned 1985 chronic rat study; in the study rats were fed hexachlorobenzene in their diets for 90 days prior to mating until 21 days after parturition. A NOAEL of 0.08 mg/kg bw/d was established based on liver effects. An uncertainty factor of 100 was applied to account for inter- and intra species variability.

ATSDR (2013) recommended a chronic oral MRL of 0.07 µg/kg bw/d for hexachlorobenzene. The MRL was also based on the 1985 chronic rat study, but the LOAEL of 0.022 mg/kg bw/d, rather than the NOAEL, for hepatic effects was selected to derive the MRL. An uncertainty factor of 300 was applied to account for inter- and intraspecies variability and use of a LOAEL.

Health Canada (1993) recommended a TD05 of 60 µg/kg bw/d. By dividing the TD05 by 5,000, the TD05 was converted to an RsD for 1×10^{-5} cancer risk of 0.012 µg/kg bw/d. A unit risk was then calculated as the target cancer risk divided by RsD (i.e., $1 \times 10^{-5}/0.012$ µg/kg bw/d = 8.3×10^{-4} [µg/kg bw/d]$^{-1}$). The TD05 was based on

the 1985 rat study described above, using a multistage model. The TD05 values calculated from the study ranged from 0.06 mg/kg bw/d for hepatic neoplastic nodules in female rats to 0.17 mg/kg bw/d for parthyroid adenomas in males.

US EPA also recommended an oral slope factor of 1.6 x 10^{-3} (µg/kg bw/d)$^{-1}$ for hexachlorobenzene, which was last reviewed by the agency in 1996. The slope factor was based on a 1986 study where rats were fed hexachlorobenzene in their diets for up to two years. A linearized multistage model was used to establish the slope factor, which was based on incidence of hepatocellular carcinomas.

RIVM (2001) provided a CRoral of 0.16 µg/kg bw/d, based on a 1:104 lifetime excess cancer risk, for hexachlorobenzene. The CRoral can be converted to an oral slope factor by dividing the lifetime excess cancer risk by CRoral, resulting in a slope factor of 6.3 x 10^{-4} (µg/kg bw/d)$^{-1}$. The CRoral was based on the application of a linear extrapolation model to the results of the 1985 rat study described above.

OEHHA (2009) recommended an oral slope factor of 1.8 x 10^{-3} (µg/kg bw/d)$^{-1}$ for hexachlorobenzene. The slope factor was based on a linearized multistage model applied to the dose-response data for the induction of hepatomas in male hamsters (1977 study), heptocellular carcinomas (1983 study) and pheochromocytomas (same 1983 study, as well as the 1985 study discussed above) in female rats. Surface area scaling was used to extrapolate from animal to human cancer potency factors.

For estimating non-cancer effects following oral and dermal exposure to hexachlorobenzene, the Health Canada pTDI of 0.27 µg/kg bw/d was used; it is noted that although the derivation of this value is not available, it is recommended by Health Canada's Health Products and Food Branch is more conservative than the 1993 TDI. For carcinogenic effects, the US EPA slope factor of 1.6 x 10^{-3} (µg/kg bw/d)$^{-1}$ was used. The US EPA would have had access to all of the studies considered by the other agencies, and as previously discussed; US EPA was identified as a preferred source.

References for Hexachlorobenzene:

ATSDR, 2013. Toxicological Profile for Hexachlorobenzene. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Health Canada. 1993. Hexachlorobenzene. Priority Substance List 1. Environment Canada. Health Canada. Archive June 24, 2013. Available at: http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/hexachlorobenzene/index-eng.php.

Health Canada. 2010. Toxicological Reference Doses for Organic Contaminants. Chemical Health Hazard Assessment Division (CHHAD), Bureau of Chemical Safety, Health Products and Food Branch, Health Canada. Last Updated September 2010.

OEHHA. 2009. Air toxics Hot Spots Risk Assessment Guidelines Part II: Technical Support Document for Cancer Potency Factors. Appendix B. Updated 2011. Available at: http://www.oehha.ca.gov/air/hot_spots/tsd052909.html

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1991 and 1996. IRIS Summary of Hexachlorobenzene (CASRN 118-74-1). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0374.htm.

V.5.9. n-Hexane

The acute inhalation TRVs for n-hexane are summarized in the following table.

Table V-84: Acute Inhalation TRVs for n-Hexane.

Source	Exposure Limit	Value	Reference
US EPA	PPRTV, Subchronic RfC	2,000 µg/m^3	US EPA, 2009

No acute duration inhalation exposure limits were identified for n-hexane. The US EPA have derived a subchronic duration RfC (PPRTV) for n-hexane of 2000 µg/m^3. The subchronic RfC is based on the neurological effects (Peripheral neuropathology) observed in rats exposed to n-hexane for 12 hours/day, 7 days/week for 16 weeks (Huang et al. 1989). The BMCL of 430 mg/m^3 was duration-adjusted and converted to a human equivalent concentration, and an uncertainty factor of 100 was applied.

Based on a lack of acute TRVs, and because the critical effects for n-hexane is reported to be systemic (vs. irritant effects), acute exposures and associated risks will not be assessed in the HHRA. The assessment of chronic exposures/risks will be protective of acute exposures/risks.

The chronic inhalation TRVs for n-hexane are summarized in the following table.

Table V-85: Chronic Inhalation TRVs for n-Hexane.

Source	Exposure Limit	Value	Reference
Health Canada	**TC**	**700 µg/m^3**	**Health Canada, 2010**
ATSDR	Chronic MRL	2,000 µg/m^3	ATSDR, 1999
OEHHA	Chronic REL	7,000 µg/m^3	OEHHA, 2008
US EPA	Chronic RfC	700 µg/m^3	US EPA, 2005

Health Canada (2010) and the US EPA (2005) recommend a TC/RfC of 700 µg/m^3 for inhalation exposures to n-hexane. The TC/RfC is based on a 1989 study where male Wistar rats were exposed to 0, 500, 1200, or 3000 ppm of n-hexane for 12 hours/day, 7 days/week for 16 weeks. A benchmark concentration of 215 mg/m^3, adjusted for continuous exposure and human exposure factors, was established based on neurological effects (peripheral neuropathy), specifically, decreased motor nerve conduction velocity. An uncertainty factor of 300 was applied (a factor of 10 for intraspecies variability, 3 for interspecies variability and 3 for use of a subchronic study).

The ATSDR has derived a chronic inhalation MRL of 2000 µg/m^3 (0.6 ppm) based on a LOAEL of 58 ppm for reduced motor nerve conduction velocity in occupationally exposed workers in a 1980 study. An uncertainty factor of 100 was applied to the LOAEL to derive the MRL (10 each for use of a LOAEL and for intraspecies variability).

The OEHHA recommend a chronic inhalation REL of 7000 µg/m^3. The REL is based on peripheral neuropathy (electromyographic alterations; dose-related abnormal posture and muscle atrophy) in male mice exposed to n-hexane vapour for 24 hours/day, 6 days/week for 1 year (1967 study). An HEC was determined for the NOAEL of the study, and an uncertainty factor of 30 (3 for interspecies variability and 10 for intraspecies variability) was applied to the HEC.

The Health Canada TC of 700 µg/m^3 was selected for use in the HHRA.

References for n-Hexane:

ATSDR, 1999. Toxicological Profile for n-Hexane. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 2005. IRIS Summary of n-Hexane (CASRN 110-54-3). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/toxreviews/0486tr.pdf

US EPA, 2009. Provisional Peer Reviewed Subchronic Toxicity Values for n-Hexane (CASRN 110-54-3). 9-30-2009.

V.5.10. Propionaldehyde

No acute inhalation exposure limits were identified for propionaldehyde.

The chronic inhalation TRVs for propionaldehyde are summarized in the following table.

Table V-86: Chronic Inhalation TRVs for Propionaldehyde.

Source	Exposure Limit	Value	Reference
US EPA	**RfC**	**8 µg/m^3**	**US EPA, 2008**

The US EPA derived a RfC for propionaldehyde of 8 µg/m^3. The RfC was based on a BMC for a 10% extra risk of olfactory atrophy from a subchronic inhalation study in male rats (Union Carbide, 1993). An uncertainty factor of 1000 was applied (10 each for intraspecies variability and use of a subchronic study, and 3 each for extrapolation from animals to humans and database deficiencies).

No other chronic inhalation exposure limit for propionaldehyde was identified and, therefore, the US EPA RfC was used in the HHRA.

References for Propionaldehyde:

US EPA, 2008. IRIS Summary of Propionaldehyde (CASRN 123-38-6). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/1011.htm.

V.5.11. Propylene (1-Propene)

No acute inhalation exposure limits were identified for propylene.

The chronic inhalation TRVs for propylene are summarized in the following table.

Table V-87: Chronic Inhalation TRVs for Propylene.

Source	Exposure Limit	Value	Reference
OEHHA	**Chronic REL**	**3,000 µg/m^3**	**OEHHA, 2008**

The OEHHA recommend a chronic inhalation REL of 3,000 µg/m^3 for propylene. The REL was based on a study where rats and mice were exposed to 0, 5,000 or 10,000 ppm propylene in controlled inhalation chambers for 6 hours/day, 5 days/week for a duration of 103 weeks. A LOAEL of 190 ppm, adjusted for continuous exposure and differences in body weight and surface are between rats and humans, was derived based on respiratory system effects including squamous metaplasia, epithelial hyperplasia and inflammation of the nasal cavity observed in rats. An uncertainty factor of 100 was applied to account for interspecies differences, intraspecies variability and the use of a minimal LOAEL, instead of a NOAEL. The OEHHA chronic REL of 3,000 µg/m^3 (2 ppm) was used to estimate potential risks associated with inhalation of propylene in the HHRA.

References for Propylene:

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

V.5.12 Styrene

The acute inhalation TRVs for styrene are summarized in the following table.

Table V-88: Acute Inhalation TRVs for Styrene

Source	Exposure Limit	Value	Reference
ATSDR	Acute MRL	21,300 µg/m^3	ATSDR, 2010
OEHHA	**Acute REL (1 hour)**	**21,000 µg/m³**	**OEHHA, 2008**

The ATSDR has derived an acute-duration inhalation MRL of 21,300 µg/m^3 (5 ppm) for styrene. This MRL is based on a NOAEL of 49 ppm for neurological effects in subjects exposed to styrene for 6 hours (2003 study). An uncertainty factor of 10 was applied to the NOAEL to account for intraspecies variability.

The OEHHA recommends an acute MRL of 21,000 µg/m^3 based on a NOAEL of 51 ppm, with an uncertainty factor of 10 for intraspecies variability, for nose and throat irritation for 3 volunteers exposed to styrene vapours (1968 study).

The values recommended by the two agencies are approximately equal; the more conservative exposure limit of 21,000 µg/m^3 was used in the HHRA.

The chronic inhalation TRVs for styrene are summarized in the following table.

Table V-89: Chronic Inhalation TRVs for Styrene

Source	Exposure Limit	Value	Reference
Health Canada	**TC**	**92 µg/m^3**	**Health Canada, 2010**
US EPA	RfC	1000 µg/m^3	US EPA, 1993
ATSDR	Chronic MRL	850 µg/m^3	ATSDR, 2010
RIVM	TCA	900 µg/m^3	RIVM, 2001
OEHHA	Chronic REL	900 µg/m^3	OEHHA, 2008

Health Canada (2010) recommended a TC of 92 µg/m^3. The TC was based on chronic inhalation study where rats were exposed to 0, 50, and 300 ppm styrene for six hours per day for days 7 to 21 of gestation, followed by postnatal exposure of pups to 217 mg/m^3 for seven hours per day for 48 days following birth (1992 studies). A LOAEL of 260 mg/m^3 was established for decreased pup weight, decreased neuroamines, neurological and behavioural changes. An uncertainty factor of 500 was applied to account for inter and intraspecies variability and for use of a LOAEL.

The US EPA, RIVM and the OEHHA derived their chronic inhalation exposure limits based on the results of a study conducted by in 1984; the study examined the neuro- psychological function in 50 workers whose mean duration of styrene exposure was 8.6 years. The US EPA calculated the 95% lower confidence limit of the NOAEL, resulting in a NOAEL of 22 ppm (or 94 mg/m^3), and applied an uncertainty factor of 30 (3 for database limitations and 10 for intraspecies variability), to derive a RfC of 1000 µg/m^3. RIVM derived a TCA of 900 µg/m^3 using the LOAEC of 107 mg/m^3 from the study, and applying an uncertainty factor of 30 (10 for human variability and 3 for extrapolation from a marginal effect). The OEHHA used a benchmark concentration approach, and derived an REL equivalent to the RIVM TCA of 900 µg/m^3.

The ATSDR has derived a chronic inhalation MRL of 850 µg/m^3 (0.2 ppm) for styrene based on a minimal LOAEL of 20 ppm (2005 study) from occupational exposure studies where alterations in choice reaction time and color discrimination were reported in workers exposed to styrene. The minimal LOAEL was adjusted for continuous exposure and divided by an uncertainty factor of 30 (3 for use of a minimal LOAEL and 10 for intraspecies variability).

Based on the previous discussed hierarchical approach, the Health Canada TC of 92 µg/m^3 was selected for use in the HHRA.

References for Styrene:

ATSDR, 2010. Toxicological Profile for Styrene. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 1993. IRIS Summary of Styrene (CASRN 100-42-5). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0104.htm.

V.5.13. Toluene

The acute inhalation TRVs for toluene are summarized in the following table.

Table V-90. Acute Inhalation TRVs for Toluene.

Source	Exposure Limit	Value	Reference
Health Canada	**Short-term exposure limit (8 hour)**	**15,000 µg/m^3**	**Health Canada, 2011**
ATSDR	Acute MRL	3,800 µg/m^3	ATSDR, 2000
OEHHA	1 hour REL	37,000 µg/m^3	OEHHA, 2008

Health Canada, the ATSDR and the OEHHA used the results of the same 1983 study to derive acute duration inhalation exposure limits. The study involved exposing 16 healthy subjects to toluene for 6 hours/day over 4 consecutive days. Significant increased ocular and nasal irritation was observed at 100 ppm, and neurological effects, including headaches, dizziness, and feeling of intoxication were reported. A NOAEL of 40 ppm (150 mg/m³) was determined for the study.

Health Canada derived a short-term inhalation exposure limit for toluene of 15 000 µg/m^3 based on the NOAEL of the 1983 study, and the application of an uncertainty factor of 10 (3.16 for pharmacokinetics and 3.16 for pharmacodynamics) to account for the potential intraspecies variability.

The ATSDR adjusted the NOAEL of the 1983 study for continuous exposure, and applied an uncertainty factor of 10 to the adjusted NOAEL to account for intraspecies variability. The resulting MRL 0f 0.6 ppm, which was rounded to 1 ppm (3,800 µg/m³), was recommended as an acute inhalation MRL.

Using the same study, the OEHHA (2008) derived a 1-hour REL of 37,000 µg/m³ by converting the 6-hour exposure duration to a 1-hour REL of 98 ppm (370 mg/m³) and applying an uncertainty factor of 10 for intraspecies variability.

Considering that the same study was used by all agencies to derive the acute TRV, the Health Canada short-term exposure limit of 15,000 µg/m³ was used to characterize acute inhalation exposure to toluene.

The chronic inhalation TRVs for toluene are summarized in the following table.

Table V-91: Chronic Inhalation TRVs for Toluene

Source	Exposure Limit	Value	Reference
Health Canada	**Long-term exposure limit**	**2,300 µg/m^3**	**Health Canada, 2011**
Health Canada	TC	3,800 µg/m^3	Health Canada, 2010
US EPA	RfC	5,000 µg/m^3	US EPA, 2005
ATSDR	Chronic MRL	300 µg/m^3	ATSDR, 2000
OEHHA	Chronic REL	300 µg/m^3	OEHHA, 2000
RIVM	TCA	400 µg/m^3	RIVM, 2000

For evaluation of inhalation exposures to toluene, Health Canada (2010) recommends a TC of 3,800 µg/m^3 based on a NOAEL (37.5 mg/m^3) for the absence of neurological effects or respiratory irritation in acutely exposed workers and the application of a ten-fold uncertainty factor.

Health Canada (2011) also recommends a Residential Indoor Air Quality Guideline for 24-hour exposure of 2,300 µg/m^3 for protection of long term effects. The exposure limit was derived based on a NOAEL of 98 mg/m³ for neurobehavioral effects from 2004 and 2005 studies of printing shop workers exposed for more than 20 years to toluene. The NOAEL was adjusted for continuous exposure, and an uncertainty factor of 10 (a factor of 3.16 for pharmacokinetics and 3.16 for pharmacodynamics) to account for intraspecies 110ariability/sensitive individuals.

The US EPA derived a RfC of 5000 µg/m^3 based on an adjusted average NOAEL of 46 000 µg/m^3 from multiple occupational studies. An uncertainty factor of 10 was applied for intraspecies variation.

The ATSDR recommends a chronic MRL of 300 µg/m^3 (0.08 ppm) based on a LOAEL of 35 ppm for neurological effects in toluene-exposed shoe-makers (Zavalic et al., 1998). An uncertainty factor of 100 was applied to the LOAEL (a factor of 10 for use of a LOAEL, and a factor of 10 for intraspecies variability).

The OEHHA derived a chronic REL of 300 µg/m^3 based on neurological effects in rats exposed to toluene for 6 hours/day, 5 days/week for 4 weeks (Hillefors-Berglund et al., 1995). An uncertainty factor of 100 (10 for use of a subchronic study, 10 for intraspecies variability) was applied to the NOAEL for the study to derive the REL.

RIVM derived a TCA of 400 µg/m^3 based on a LOAEL of 88 ppm for deficits in a battery of neurological tests in electronics workers (Foo et al., 1990), with an uncertainty factor of 300 applied to the LOAEL (10 for intraspecies variability, 10 for the use of a LOAEL and 3 to account for database deficiencies).

The Health Canada long-term exposure limit (24-hour Residential Indoor Air Quality Guideline) of 2,300 µg/m^3 was used to assess chronic inhalation exposures to toluene. The exposure limit has been derived using the results for chronically exposed humans, and the assessment conducted by Health Canada considered the recent available toxicity data for toluene.

References for Toluene:

ATSDR, 2000. Toxicological Profile for Toluene. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. 2000.

Health Canada, 2011. Residential Indoor Air Quality Guideline, Toluene. Available on-line at http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/air/toluene/toluene-eng.pdf.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 2005. IRIS Summary of Toluene (CASRN 108-88-3). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0118.htm.

V.5.14. 2,2,4-Trimethylpentane

No acute or chronic inhalation TRVs were identified for 2,2,4-trimethylpentane.

V.5.15. Xylenes

The acute inhalation TRVs for xylenes are summarized in the following table.

Table V-92: Acute Inhalation TRVs for Xylenes

Source	Exposure Limit	Value	Reference
ATSDR	**Acute MRL (2 hour)**	**8,700 µg/m^3**	**ATSDR, 2007**
OEHHA	Acute REL (1 hour)	22,000 µg/m^3	OEHHA, 2008

The ATSDR (2007) derived an acute inhalation MRL of 8,700 µg/m^3 (2 ppm) based on a minimal LOAEL of 50 ppm (217 mg/m^3) for mild respiratory effects and neurological effects in individuals exposed to m-xylene vapour for 2 hours (2002 study); an uncertainty factor of 30 (10 for intraspecies variability, 3 for use of a minimal LOAEL) was applied to the minimal LOAEL.

The OEHHA recommends an acute (1 hour) REL for xylenes of 22,000 µg/m^3. The REL is based on a NOAEL for subjective throat, eye and nasal irritation reported by 50 volunteers exposed to xylenes vapour for 30 minutes (Hastings et al., 1984). The NOAEL was adjusted for a 1 hour exposure, and an uncertainty factor of 10 (for intraspecies variability) was applied.

The ATSDR acute inhalation MRL of 8,700 µg/m^3 was selected for use in the HHRA. The ATSDR MRL is more conservative that the OEHHA REL, and both studies are based on human data.

The chronic inhalation TRVs for xylenes are summarized in the following table.

Table V-93: Chronic Inhalation TRVs for Xylenes

Source	Exposure Limit	Value	Reference
Health Canada	**TC**	**180 µg/m^3**	**Health Canada, 2010**
US EPA	RfC	100 µg/m^3	US EPA, 2003
ATSDR	Chronic MRL	220 µg/m^3	ATSDR, 2007
OEHHA	Chronic REL	700 µg/m^3	OEHHA, 2008
RIVM	TCA	870 µg/m^3	RIVM, 2001

Health Canada derived a TC of 180 µg/m^3 based on a LOAELHEC of 180 mg/m^3 for maternal and fetal effects observed in rats (1985 study) and an uncertainty factor of 1000 (10 each for intra-, inter-species variability, and use of a LOAEL).

The US EPA derived a RfC of 100 µg/m^3 based on a NOAELHEC of 39 mg/m^3 for neurological effects in rats (1994 study). An uncertainty factor of 300 (10 for intraspecies variability and 3 each for interspecies differences,

extrapolation from subchronic to chronic duration study, and uncertainties in the data base) was applied to the NOAELHEC.

The ATSDR derived chronic inhalation MRL of 220 µg/m^3 (0.05 ppm) based on a LOAEL of 14 ppm (geometric mean) for mild subjective respiratory and neurological symptoms in workers exposed to xylenes for 8 hours/day, 5 days/week for up to 7 years (1993 study). An uncertainty factor of 100 and a modifying factor of 3 was applied to the LOAEL.

RIVM derived a TCA of 870 µg/m^3 based on a LOAEL of 870 mg/m^3 for developmental toxicity in rats and an uncertainty factor of 1000 (10 each for intra-, inter-species variability, and use of a LOAEL).

The OEHHA (2008) has developed a chronic REL of 700 µg/m³ based on the incidence of eye irritation, sore throat and mild neurological effects reported in the above discussed 1993 study. The OEHHA adjusted the LOAEL of 14 ppm for continuous exposure and applied an uncertainty factor of 30 to the adjusted LOAEL; 3 for the use of a LOAEL (due to the minor nature of the adverse effects) and 10 for intraspecies variability.

Based on the previous discussed hierarchical approach, the Health Canada (2010) TC of 180 µg/m³ was used in the HHRA.

References for Xylenes:

ATSDR, 2007. Toxicological Profile for Xylenes. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. 2007.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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

RIVM, 2001. Re-evaluation of human toxicological maximum permissible risk levels. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 025. March 2001.

US EPA, 2003. IRIS Summary of Xylenes (CASRN 1330-20-7). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0270.htm.

V.6. DUST PALLIATIVES CHEMICAL CONTITUENTS

Of the dust palliative chemical constituents, propylene oxide and epichlorohydrin were identified as being gaseous. Although neither propylene oxide or epichlorohydrin were identified as being bioaccumulative, epichlorohydrin was conservatively retained in the multi-media assessment as of the dust palliative constituents, it has the highest relative toxicity (see below evaluation).

V.6.1. Adipic Acid

V.6.1.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for adipic acid.

V.6.1.2. Oral TRVs

The oral TRVs for adipic acid are summarized in the following table.

Table V-94. Oral TRVs for Adipic Acid.

Source	Exposure Limit	Value	Reference
WHO	ADI	Maximum of 5,000 µg/kd bw/d	JECFA, 1967
NSF International	**Oral RfD**	**4,000 µg/kg bw/d**	**NSF International, 2006**

JECFA (1967) provided an ADI range of 0 to 5000 µg/kd bw/d for adipic acid. The ADI was based on a 1957 study where rats were administered adipic acid for 2 years. A dose equivalent to 500 mg/kg bw/d was established as the NOAEL based on body weight changes. It was assumed that an uncertainty factor of 100 was applied to the NOAEL to achieve a maximum ADI of 5,000 µg/kd bw/d.

Although not listed as a preferential source for TRVs by Health Canada, NSF International (2006) recommended an oral RfD of 4,000 µg/kd bw/d. The RfD was based on a 33-week chronic repeated dose study in rats completed in 1953 with support from a 1957 analysis. A weight of evidence NOAEL of 400 mg/kg bw/d was established based on effects, including reduced survival, diarrhea, decreased body weight during growth, and intestinal and liver pathology. An uncertainty factor of 100 was applied to account for inter- and intraspecies extrapolation.

The more recently established NSF International (2006) RfD of 4,000 µg/kd bw/d was used to assess oral and dermal risks from exposure to adipic acid.

References for Adipic Acid:

JECFA. 1967. Toxicological Evaluation of Some Antimicrobials, Antioxidants, Emulsifiers, Stabilizers, Flour-treatment Agents, Acids and Bases. World Health Organization, 1967.

National Sanitation Foundation (NSF) International. 2006. NSF Adipic Acid Standard. Published November 1, 2006. Available on-line at: http://www.techstreet.com/nsf/products/1287485#jumps.

V.6.2. Diethylaminoethanol

No acute or chronic inhalation TRVs or oral TRVs were identified for diethylaminoethanol.

In the absence of TRVs and to evaluate the relative toxicity of the chemical constituents of the dust palliatives, reference was made to the Worksafe BC Occupational Exposure Limit (8 hour Time Weighted Average [TWA] limit) for diethylaminoethanol; the 8 hour TWA is 2 ppm or 9586 µg/m^3.

V.6.3. Diethylenetriamine

No acute or chronic inhalation TRVs or oral TRVs were identified for diethylenetriamine.

In the absence of TRVs and to evaluate the relative toxicity of the chemical constituents of the dust palliatives, reference was made to the Worksafe BC Occupational Exposure Limit (8 hour Time Weighted Average [TWA] limit) for diethylaminoethanol; the 8 hour TWA is 1 ppm or 4220 µg/m^3.

V.6.4. Epichlorohydrin

V.6.4.1. Inhalation TRVs

The chronic inhalation TRVs for epichlorohydrin are summarized in the following table.

Table V-95: Acute Inhalation TRVs Epichlorohydrin.

Source	Exposure Limit	Value	Reference
OEHHA	**1 hour REL**	**1,300 µg/m^3**	**OEHHA, 2008**

The OEHHA derived an acute (1 hour) REL of 1300 µg/m^3 (0.33 ppm) based on the results of Wexler (1971) as cited in NIOSH, 1976. A LOAEL of 20 ppm was reported for irritation of eyes and nasal passages in workers exposed to epichlorohydrin. An uncertainty factor of 60 (6 for use of a LOAEL (based on mild effects) and 10 for intraspecies variability) was applied to the LOAEL to derive the REL.

The OEHHA 1 hour REL of 1300 µg/m^3 was used to characterize risks associated with acute inhalation exposures to epichlorohydrin.

The chronic inhalation TRVs for epichlorohydrin are summarized in the following table.

Table V-96: Chronic Inhalation TRVs Epichlorohydrin.

Source	Exposure Limit	Value	Reference
US EPA	**RfC**	**1 µg/m^3**	**US EPA, 1992**
US EPA	**Unit Risk**	**1.2 x 10^{-6} (µg/m^3)$^{-1}$**	**US EPA, 1994**
OEHHA	Chronic REL	3 µg/m^3	OEHHA, 2008
OEHHA	Unit Risk	2.3 x 10^{-5} (µg/m^3)$^{-1}$	OEHHA, 2009

Health Canada did not provide exposure limits for epichlorohydrin.

The US EPA and the OEHHA have both derived non-cancer inhalation exposure limits (a RfC and Chronic REL, respectively) based on changes in the nasal turbinates in rats following inhalation exposures to epichlorohydrin in a study conducted in 1979. For non-cancer inhalation effects, US EPA (1992) provided a RfC of 1 µg/m^3. The RfC was based on the NOAEL for the 1979 study of 5 ppm, which was adjusted for continuous exposures and converted to an HEC, and an uncertainty factor of 300 (10 for intraspecies variability, 3 for interspecies variability and 10 for use of a subchronic study) was applied to calculate the RfC. The OEHHA identified the same NOAEL for the study, but applied an uncertainty factor of 100 (3 for use of a subchronic study, 3 for interspecies variability and 10 for intraspecies variability). Because the US EPA and the OEHHA values are based on the same study, and the US EPA RfC is more conservative, the US EPA RfC will be used in the HHRA.

Both the US EPA (1994) and the OEHHA (2009) also provided an inhalation unit risks for epichlorohydrin of 1.2 x 10^{-6} (µg/m^3)$^{-1}$ and 2.3 x 10^{-5} (µg/m^3)$^{-1}$, respectively. The US EPA unit risk was derived based on nasal cavity tumours in male rats following inhalation in a study conducted in 1980. The OEHHA derived their unit risk based on male Wistar rat forestomach papilloma and squamous cell carcinoma observed in male Wistar rats following oral (drinking water) exposures (second 1980 study). The OEHHA identified the second 1980 study as the key study over the study identified by the US EPA, due to the poor survival of the animals in the Laskin study. However, the US EPA unit risk is considered to be most appropriate for use based on: 1) it is based on inhalation (versus oral) exposures; and, 2) the relevance of forestomach tumours is questionable given that humans do not have forestomachs.

Although inhalation (and oral) TRVs are available for epichlorohydrin, to allow for the evaluation of the relative toxicity of epichlorohydrin compared to the chemical constituents of the dust palliatives that lack TRVs, reference

was made to the Worksafe BC Occupational Exposure Limit (8 hour Time Weighted Average [TWA] limit) for epichlorohydrin; the 8 hour TWA is 0.1 ppm or 391 µg/m^3. As is discussed in Section 5 of the HHRA, based on the available TRVs for the chemical constituents of the dust palliatives, as well a comparison of the 8 hour TWAs for the constituents lacking TRVs, epichlorohydrin has the highest relative toxicity of the constituents of the dust palliatives.

V.6.4.2. Oral TRVs

The oral TRVs for epichlorohydrin are summarized in the following table.

Table V-97. Oral TRVs for Epichlorohydrin.

Source	Exposure Limit	Value	Reference
US EPA	**Oral SF**	**9.9 x 10^{-6} (µg/kg bw/d)$^{-1}$**	**US EPA, 1994**
US EPA	**RfD**	**6 µg/kg bw/d**	**US EPA PPRTV, 2006**

US EPA (1994) provided an oral slope factor of 9.9 x 10^{-6} (µg/kg bw/d)$^{-1}$ for assessing the carcinogenic effects of epichlorohydrin. The slope factor was based on papillomas and carcinomas of the forestomach in male Wistar rats following drinking water exposure for 81 weeks in a study by Konishi et al. (1980).

US EPA (2006) provided a PPRTV oral RfD of 6 µg/kg bw/d for exposure to epichlorohydrin. The RfD was based on a 1991 study where Long-Evans rats were orally administered epichlorohydrin for 23 days and mating trials were conducted at study days 19 and 22 to evaluate fertility. A LOAEL of 6.25 mg/kg bw/d was established based on reduced male fertility. A total uncertainty factor of 1000 was applied to the LOAEL to account for estimating a NOAEL from a minimal LOAEL, interspecies extrapolation, variation in human sensitivity, and an incomplete database (i.e., multi-generation reproduction study and a longer-term toxicity study in a second species are missing). A subchronic to chronic factor was not applied because there is no evidence that epichlorohydrin becomes more toxic with increased exposure duration and epichlorohydrin is rapidly eliminated and does not accumulate in tissues.

The US EPA (1994) oral slope factor of 9.9 x 10^{-6} (µg/kg bw/d)$^{-1}$ was used to assess cancer risks and the US EPA (2006) PPRTV oral RfD of 6 µg/kg bw/d was used to assess non-cancer risks following oral and dermal exposure to epichlorohydrin.

References for Epichlorohydrin:

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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, 2009. California Office of Environmental Health Hazard Assessment. 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.

US EPA, 1992 and 1994. IRIS Summary of Epichlorohydrin (CASRN 106-89-8). United States Environmental Protection Agency, Washington, DC Available at: http://www.epa.gov/iris/subst/0050.htm.

US EPA. 2006. Provisional Peer Reviewed Toxicity Values for Epichlorohydrin. National Center for Environmental Assessment. Office of Research and Development, United States Environmental Protection Agency, Cincinnati, OH.

V.6.5. Linear Alkyl Sulfonate

No acute or chronic inhalation TRVs or oral TRVs were identified for linear alkyl sulfonate.

No Worksafe BC Occupational Exposure Limits were available for linear alkyl sulfonate.

V.6.6. Propylene Glycol

V.6.6.1. Inhalation TRVs

No acute or chronic inhalation exposure limits were identified for propylene glycol.

V.6.6.2. Oral TRVs

The oral TRVs for propylene glycol are summarized in the following table.

Table V-98. Oral TRVs for Propylene Glycol.

Source	Exposure Limit	Value	Reference
US EPA	**RfD**	**20,000 µg/kg bw/d**	**US EPA, 2008**

US EPA (2008) derived a PPRTV oral RfD of 20,000 µg/kg bw/d for propylene glycol. The RfD was based on a 1971 study where rats were exposed to propylene glycol in drinking water for five weeks. A LOAEL of 5,200 mg/kg bw/d was established based on reduced red blood cell counts and hyperglycemia. A total uncertainty factor of 300 was applied to the LOAEL to account for estimating a NOAEL from a minimal LOAEL, interspecies extrapolation, and variation in human sensitivity.

The US EPA (2008) PPRTV oral RfD of 20,000 µg/kg bw/d was considered to be appropriate to assess chronic oral, dermal and inhalation exposures to propylene glycol. In addition, it is noted that propylene glycol is on the US Food and Drug Administration GRAS (Generally Recognized as Safe) status list (available at http://www.fda.gov/Food/IngredientsPackagingLabeling/GRAS/SCOGS/default.htm).

References for Propylene Glycol:

US EPA. 2008. Provisional Peer Reviewed Toxicity Values for Propylene Glycol. National Center for Environmental Assessment. Office of Research and Development, United States Environmental Protection Agency, Cincinnati, OH.

V.6.7. Propylene Oxide

No acute inhalation TRVs were identified for propylene oxide.

The chronic inhalation TRVs for propylene oxide are summarized in the following table.

Table V-99: Chronic Inhalation TRVs for Propylene Oxide

Source	Exposure Limit	Value	Reference
US EPA	**Unit Risk**	**3.7 x 10^{-6} $(\mu g/m^3)^{-1}$**	**US EPA 1994**
US EPA	**RfC**	**30 $\mu g/m^3$**	**US EPA 1990**

US EPA (1994) provided an inhalation unit risk of 3.7 x 10^{-6} $(\mu g/m^3)^{-1}$, which was based on nasal cavity hemangioma or hemangiosarcoma in male mice following inhalation.

For non-cancer inhalation effects, US EPA (1990) provided a RfC of 30 $\mu g/m^3$. The RfC was based on a 2 year chronic rat inhalation study where a LOAEL of 2.9 mg/m^3 was established for the development of nest-like infold of the nasal respiratory epithelium. An uncertainty factor of 100 was applied to the LOAEL.

No other inhalation exposure limits were identified for propylene oxide, and therefore, the US EPA RfC and inhalation unit risk was used in the HHRA.

References for Propylene Oxide:

US EPA, 1990 and 1994. IRIS Summary of Propionaldehyde (CASRN 75-56-9). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0403.htm.

V.6.8. Succinic Acid

No acute or chronic inhalation TRVs or oral TRVs were identified for succinic acid. It is noted that succinic acid is on the US Food and Drug Administration GRAS (Generally Recognized as Safe) list (available at http://www.fda.gov/Food/IngredientsPackagingLabeling/GRAS/SCOGS/default.htm).

V.7 OTHERS

Two additional COPCs, sulfate and PCBs, are discussed below. Both of the COPCs were determined to be non-gaseous and were included in the multi-media assessment.

V.7.1. Polychlorinated Biphenyls (PCB)

Although reported by Levelton (2014) that PCBs are present in combustion emissions from the Project, the available data were limited and Levelton was not able to determine the congeners present in the emissions. On this basis, the PCBs were assumed to be entirely present as the most potent PCB congener. As described in Health Canada (2012) and WHO (2005), PCB 126 was estimated to have a toxic equivalency factor (TEF) of 0.1 relative to 2,3,7,8-tetrachlorodibenzodioxin (TCDD). It is stressed that consideration of all PCBs occurring as PCB 126 is an extremely conservative assumption and as shown in Health Canada (2012) and WHO (2005) most PCBs are much less potent than this congener. Consequently, it is advised that the potential for unacceptable risks is predicted using this approach, that the assumption be revisited.

V.7.1.1. Inhalation TRVs

No acute or chronic inhalation TRVs were identified for PCBs.

V.7.1.2. Oral TRVs

The oral TRVs for 2,3,7,8-TCDD are summarized in the following table.

Table V-100. Oral TRVs for 2,3,7,8-TCDD.

Source	Exposure Limit	Value	Reference
Health Canada	**Oral TDI**	**2.3 x 10^{-6} µg/kg bw/d**	**Health Canada, 2010**
WHO	Oral TDI (based on monthly intake)	2.3 x 10^{-6} µg/kg bw/d	WHO, 2001
US EPA	RfD	7 x 10^{-7} µg/kg bw/d	US EPA, 2012
RIVM	pTDI	2 x 10^{-6} µg/kg bw/d	RIVM, 2009
ATSDR	Chronic oral MRL	1 x 10^{-6} µg/kg bw/d	ATSDR, 1998
California OEHHA	Chronic Oral REL	1 x 10^{-5} µg/kg bw/d	OEHHA, 2008

Health Canada (2010) recommended an oral TDI of 2.3 x 10^{-6} µg/kg bw/d for 2,3,7,8-TCDD. The oral TDI was based on a suite of subchronic and developmental studies in rats administered 2,3,7,8-TCDD through diet (study reported in 2001) or subcutaneous injection (study reported in 1998). The TDI was based on developmental effects, including immune and reproductive effects in offspring of exposed dams. A PTMI of 70 pg/kg bw/month was established as the mid-point of the range of tolerable intakes (40 to 100 pg/kg bw/month) with an uncertainty factor applied. This rationale for this PTMI was similar to that provided by WHO (2001). Health Canada (2010) then divided this PTMI by 30 to estimate the TDI of 2.3 pg/kg bw/d (i.e., 2.3 x 10^{-6} µg/kg bw/d). Consequently, using the assumption that all PCBs are as potent as PCB 126 (i.e., TEF of 0.1), a TDI of 2.3 x 10^{-5} µg/kg bw/d was estimated.

US EPA recommended a RfD of 7 x 10^{-7} µg/kg bw/d for 2,3,7,8-TCDD, which was last reviewed in 2012. The RfD was based on 2008 epidemiological cohort studies where humans were exposed to TCDD through an industrial accident. An adjusted LOAEL of 2 x 10^{-8} mg/kg bw/d was established based on decreased sperm count and motility in men exposed to TCDD as boys and increased thyroid stimulating hormone in neonates. An uncertainty factor of 30 was applied to account for LOAEL to NOAEL extrapolation and inter-individual variability.

RIVM (2009) presented a provisional TDI of 2 x 10^{-6} µg/kg bw/d for 2,3,7,8-TCDD, which was based on the same studies presented by Health Canada (2010).

ATSDR (1998) recommended a chronic oral MRL of 1 x 10^{-6} µg/kg bw/d for 2,3,7,8-TCDD. The MRL was based on a study by Shantz et al. (1992) where rhesus monkeys were exposed to TCDD. A LOAEL of 0.00012 µg/kg bw/d was derived based on developmental toxicity. An uncertainty factor of 90 was applied to the LOAEL.

OEHHA (2008) recommended a chronic oral REL of 1 x 10^{-5} µg/kg bw/d for 2,3,7,8-TCDD. The REL was based on a study by Kociba et al. (1978) were rats were continuously exposed to 2,3,7,8-TCDD in their diet, starting at

seven weeks of age for a duration of 2 years. A NOAEL of 0.001 µg/kg bw/d was established based on a variety of critical effects including increased mortality, decreased weight gain and depression of hematologic measures.

Based on the TRV selection hierarchy, the Health Canada (2010) TDI of 2.3 x 10^{-6} µg/kg bw/d for 2,3,7,8-TCDD and thus, 2.3 x 10^{-5} µg/kg bw/d for PCB 126 was assumed. Personal communications with Health Canada's food branch has indicated that this value is still being used. Nevertheless, it is recognized that the US EPA (2012) RfD was based on toxicological studies that were not available at the time of the Health Canada derivation and therefore, the US EPA (2012) RfD of 7 x 10^{-6} µg/kg bw/d that would be estimated for PCB 126 using the US EPA RfD for 2,3,7,8-TCDD was considered in the sensitivity assessment.

References for PCBs:

ATSDR, 1998. Toxicological Profile for Polychlorinated Biphenyls. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

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. September 2010. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

Health Canada, 2012. Federal Contaminated Site Risk Assessment in Canada – Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA). Version 2.0. September 2010, revised 2012. Contaminated Sites Division, Safe Environments Programme, Health Canada, Ottawa, Ontario.

OEHHA. 2008. Updated August 2013. Air Toxics Hot Spots Program Technical Support Document for the Derivation of Noncancer Reference Exposure Levels. California Environmental Protection Agency.

RIVM. 2009. Re-evaluation of some human-toxicological maximum permissible risk levels earlier evaluated in the period 1991-2001. Bilthoven, Netherlands.

US EPA. 2012. IRIS Summary of 2,3,7,8-Tetrachlorodibenzodioxin (CASRN: 1746-01-6). United States Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/1024.htm.

WHO. 2001. WHO Food Additives Series: 48 Safety Evaluation of Certain Food Additives and Contaminants. Prepared by the Fifty-seventh Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), World Health Organization, Geneva, Switzerland. Available at: http://www.inchem.org/documents/jecfa/jecmono/v48je20.htm accessed June 2014.

WHO. 2005. 2005 Re-evaluation of human and mammalian toxic equivalency factors (TEFs) (last updated November 2011). World Health Organization, Geneva, Switzerland. Available at: http://www.who.int/foodsafety/chem/tef_update/en/ accessed June 2014.

V.7.2. Sulfate

V.7.2.1. Inhalation TRVs

The acute inhalation TRVs for sulfate are summarized in the following table.

Table V-101: Acute Inhalation TRVs for Sulfate.

Source	Exposure Limit	Value	Reference
OEHHA	Acute inhalation REL	120 µg/m^3	OEHHA, 2008

OEHHA (2008) recommended an acute inhalation REL of 120 µg/m^3 for a 1-hour exposure. The REL was based on a 1983 study where asthmatics were exposed to sulfate through inhalation for 16 minutes. A NOAEL of 450 µg/m^3 was established based on small changes in airway function tests. The 1-hour REL was extrapolated from the NOAEL by multiplying the NOAEL by 0.27 (equal to 16 minutes/60 minutes).

The OEHHA (2008) acute inhalation REL was used to assess acute inhalation exposures to sulfate.

No chronic inhalation TRVs were identified for sulfate.

V.7.2.2. Oral TRVs

No oral TRVs were identified for sulfate.

References for Sulfate:

OEHHA, 2008. California Office of Environmental Health Hazard Assessment. 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.

APPENDIX VI

SNC·LAVALIN

Statistical Analysis of Coal and Background Soil Results

APPENDIX VI: STATISTICAL ANALYSES OF COAL AND BACKGROUND SOIL RESULTS

This appendix provides details on the statistical analyses used to calculate exposure concentrations to chemicals of potential concern (COPC) from background soil and source coal in the area of the Fraser Surrey Docks (FSD) Direct to Barge (DTB) facility in Surrey, BC (the Project).

The Human Health Risk Assessment (HHRA) evaluated the potential for people living and working in the Project area to be exposed to emissions, including coal dust, from the Project. The HHRA was conducted to address public concerns on the potential for adverse health effects if there is exposure to coal dust by inhalation and/or through secondary routes such as consumption of crops grown on soils impacted by coal dust. Metals and polycyclic aromatic hydrocarbons (PAHs) are components of coal and coal dust, and the HHRA estimated exposures to these parameters from coal dust. Metals and PAHs are also present in soil from both natural and anthropogenic sources; as per standard HHRA methods, to assess overall exposures to these substances, the background soil concentrations of these substances were determined. The laboratory results for metals and PAHs for surface soil samples were used to estimate the background concentrations of each of the individual constituents in the surface soil in the various communities within the Project area, which were subsequently used in the Exposure Assessment of the HHRA to estimate exposure point concentrations for these parameters in background surface soils.

VI.1. SAMPLING PLAN DETAILS

To determine the concentrations of metals/metalloids and PAHs in the coal that is proposed to be transported as part of the Project, coal samples were obtained from the producers that will supply coal to FSD (i.e., the source coal) and submitted to ALS Environmental in Burnaby, BC (ALS) for laboratory analysis of total metals (including metalloids), chromium speciation (i.e., concentrations of both chromium III and chromium VI were determined), PAHs, and crystalline silica. The coal samples were collected on February 19, 2014. Source coal was sampled from two suppliers (CUST-A and CUST-B). The laboratory results for metals/metalloids and PAHs provided the percent composition of each of the individual constituents in the source coal. Table V1-1 summarizes the number of samples collected and analysed from each coal sample location.

Table VI-1. The Number of Surface Soil Samples Analysed from Each Coal Source

Parameter	Number of Samples (n)		
	CUST-B	CUST-A	All Coal Samples
Total Metals and Chromium Speciation	6	6	12
PAHs	10	10	20
Crystalline Silica	5	5	10

Statistical analyses were completed on the samples collected from the individual coal sources and on the dataset from both of the coal sources combined. Statistical analyses were not completed on the crystalline silica analytical results because all of the data were below laboratory detection limits (DL).

To determine background concentrations of COPC in soil, surface soil samples were collected from the municipalities of Delta, Richmond, Surrey, and White Rock, BC. Surface soil samples were submitted to ALS for laboratory analysis of total metals, chromium speciation, and PAHs. The soil samples were collected on February 25 and 26, 2014. Table V1-2 summarizes the number of samples collected and analysed from each municipality.

Table V1-2. The Number of Surface Soil Samples Analysed from Each Municipality

Parameter	Number of Samples (n)						
	Delta	Richmond	Surrey	White Rock	Richmond + Delta	Agricultural Areas	All Soil Sample Locations
Total Metals and Chromium Speciation	11	11	13	13	22	14	48
PAHs	11	11	13	13	22	14	48

Summary statistics were calculated for the samples collected from individual municipalities, as well as the combined results from Richmond and Delta. Select samples from the Richmond and Delta datasets were complied to represent agricultural areas in the vicinity of the Project. Finally, the datasets from all four municipalities were combined and analysed as one dataset.

VI-1.1. Statistical Analyses

For each sample analysed, summary statistics, including maximum concentrations, arithmetic means, standard deviation and 90th percentiles were determined using Microsoft 2007© Excel software. The 95% upper confidence limit of the mean (UCLM's) were derived using United States Environmental Protection Agency (US EPA) ProUCL Version 5.0.00 software.

In the event that a COPC concentration in surface soil or coal was below laboratory DL, a proxy value equal to the DL was used to represent the concentration for that parameter when calculating maximum concentrations, arithmetic means, standard deviation of the means, and 90th percentiles.

Descriptions of each of the summary statistics used to describe the distribution of the background soil and source coal samples are provided below.

VI-1.2. Maximum

The maximum represents the highest concentration of each COPC in the dataset. Using the maximum concentration of the COPC in the exposure assessment would represent the reasonable worst-case scenario. According to Health Canada (2012, 2010) risk assessment guidance, the maximum concentration is typically used in preliminary quantitative risk assessment where there is limited data (Health Canada, 2010).

VI-1.3. Arithmetic Mean

The arithmetic mean is the average concentration of the sample data and it is computed as the sum of all the observed concentrations from the sample, divided by the total number of samples. The arithmetic mean can be used to represent COPC concentrations in detailed quantitative risk assessment when it is supported by the available data (Health Canada, 2010).

VI-1.4. Standard Deviation

Standard deviation of the distribution of the sample mean represents the degree to which individual COPC concentrations differ from the sample arithmetic mean. The standard deviation represents the amount of variability in COPC concentrations. A low standard deviation demonstrates that the sampled concentrations tend to be close to the average concentration; conversely, a high standard deviation shows that the COPC concentrations are spread out from the average COPC concentration. The standard deviation of a sample mean

can help elucidate potential outliers or extraneous data points in a dataset that may require further examination or exclusion from statistical analyses.

VI-1.5. 90th Percentile

The 90th percentile represents the COPC concentration below which 90% of the sample concentrations fall.

VI-1.6. 95% UCLM

Since infinite sampling would be required to determine the true mean COPC concentrations in soil or coal, representative samples are collected and statistical theory is applied to estimate the true mean concentrations (i.e., the population mean concentration). The 95% UCLM represents the upper boundary or limit of a confidence interval of the population mean, such that repeated sampling of the population (e.g., COPC concentrations in soil) would result in the confidence interval capturing the true population mean 95% of the time. According to Health Canada guidance, the 95% UCLM can be used to represent COPC concentrations in detailed quantitative risk assessment when supported by the data (Health Canada, 2010).

The 95% UCLM was calculated for those COPC with sufficient samples size (i.e., at least 10 records). 95% UCLM were determined using US EPA's ProUCL Version 5.0.00. ProUCL is able to incorporate non-detect data, including data with multiple detection levels into the calculation of 95% UCLM based on statistical theorems established in Singh, Singh, and Laci (2002) and Singh and Singh (2003).

Recent guidance from Helsel and Gilroy (2012) presented at the Contaminated Sites Approved Professionals (CSAP) 2012 Statistics Workshop was used to select the appropriate 95% UCLM generated by ProUCL software. Helsel and Gilroy indicated that the "potential [upper confidence limit] UCL to use" provided by ProUCL is based on an outdated method and recommended that the selection of the UCL should be based on the distribution of the data, as follows:

- Normal distribution – 95% Student's-t UCL;
- Gamma distribution – depending on the sample size, either the 95% Approximate Gamma UCL ($n \geq 40$) or the 95% Adjusted Gamma UCL ($n < 40$);
- Lognormal distribution – 95% Percentile Bootstrap; and
- No discernible distribution – 95% Kaplan-Meier (KM) Percentile Bootstrap.

The 95% UCLM values for this assessment were selected based on the methods recommended by Helsel and Gilroy (2012).

VI.2. SUMMARY STATISTIC RESULTS

The summary statistics for concentrations of COPC in source coal and background soil, including the selected ProUCL values, are presented in the attached tables.

VI-3. REFERENCES:

Health Canada. 2010. Federal Contaminated Site Risk Assessment in Canada, Part V: Guidance on Human Health Detailed Quantitative Risk Assessment for Chemicals ($DQRA_{chem}$). Ottawa, ON.

Helsel, D.R. and E.J. Gilroy. 2012. Environmental Statistics using ProUCL. Contaminated Sites Approved Professionals Workshop. Vancouver, BC. January 19, 2012. Presented by Practical Stats.

Singh, A., Singh, A.K., and Iaci, R.J. 2002. *Estimation of the Exposure Point Concentration Term Using a Gamma Distribution,* EPA/600/R-02/084, October 2002.

Singh, A. and Singh, A.K. 2003. *Estimation of the Exposure Point Concentration Term (95% UCL) Using Bias-Corrected Accelerated (BCA) Bootstrap Method and Several other methods for Normal, Lognormal, and Gamma Distributions*. Draft EPA Internal Report.

APPENDIX VII

SNC·LAVALIN

Analytical Chemistry Results

TABLE VII-1: Summary of Analytical Results for PAHs and Total Metals in CUST-A COAL

Sample Location		CUST-A-1															
Sample ID		CUST-A-1-01	CUST-A-1-02	CUST-A-1-03	CUST-A-1-04	CUST-A-1-05	CUST-A-1-06	CUST-A-1-11	CUST-A-1-12	CUST-A-1-13	CUST-A-1-14	CUST-A-1-15	CUST-A-1-16	CUST-A-1-17	CUST-A-1-18	CUST-A-1-19	CUST-A-1-20
Sample Date (yyyy mm dd)		2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19
Parameter	**Units**	**Analytical Results**															
Physical Parameters																	
pH	pH	6.46	6.41	6.42	6.4	6.38	6.36	-	-	-	-	-	-	-	-	-	-
Polycyclic Aromatic Hydrocarbons																	
Naphthalene	µg/g	-	-	-	-	-	-	0.081	0.088	0.072	0.075	0.082	0.104	0.097	0.103	0.099	0.114
2-Methylnaphthalene	µg/g	-	-	-	-	-	-	0.085	0.084	0.072	0.077	0.078	0.082	0.085	0.086	0.09	0.098
Acenaphthylene	µg/g	-	-	-	-	-	-	0.099	0.098	0.073	0.088	0.093	< 0.06	< 0.06	< 0.05	< 0.05	< 0.06
Acenaphthene	µg/g	-	-	-	-	-	-	0.689	0.706	0.569	0.614	0.648	0.57	0.576	0.568	0.517	0.526
Fluorene	µg/g	-	-	-	-	-	-	< 0.2	< 0.2	< 0.08	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2
Phenanthrene	µg/g	-	-	-	-	-	-	0.367	0.362	0.278	0.335	0.335	0.335	0.332	0.32	0.345	0.386
Anthracene	µg/g	-	-	-	-	-	-	0.17	0.17	0.13	0.146	0.156	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2
Fluoranthene	µg/g	-	-	-	-	-	-	2.06	2.06	1.7	1.98	1.89	1.79	1.8	1.72	1.82	2.07
Pyrene	µg/g	-	-	-	-	-	-	2.38	2.43	1.5	2.22	2.25	1.48	1.5	1.43	1.54	1.73
Benzo(a)anthracene	µg/g	-	-	-	-	-	-	0.436	0.457	0.378	0.409	0.409	0.39	0.405	0.384	0.411	0.449
Chrysene	µg/g	-	-	-	-	-	-	0.317	0.349	0.273	0.247	0.307	< 0.3	< 0.3	< 0.3	< 0.3	< 0.4
Benzo(b)fluoranthene	µg/g	-	-	-	-	-	-	0.665	0.765	0.607	0.67	0.718	0.447	0.466	0.435	0.471	0.513
Benzo(b+j+k)fluoranthene	µg/g	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Benzo(k)fluoranthene	µg/g	-	-	-	-	-	-	0.147	0.159	0.132	0.161	0.122	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2
Benzo(a)pyrene	µg/g	-	-	-	-	-	-	0.138	0.147	0.114	0.13	0.125	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2
Indeno(1,2,3-cd)pyrene	µg/g	-	-	-	-	-	-	< 0.08	< 0.2	< 0.1	< 0.09	< 0.2	< 0.1	< 0.2	< 0.2	< 0.2	< 0.2
Dibenz(a,h)anthracene	µg/g	-	-	-	-	-	-	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05
Benzo(g,h,i)perylene	µg/g	-	-	-	-	-	-	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.06	< 0.06	< 0.08	< 0.06	< 0.08
Total Metals																	
Antimony	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Arsenic	µg/g	1.37	1.22	1.39	1.28	1.4	1.45	-	-	-	-	-	-	-	-	-	-
Barium	µg/g	61.4	34.1	69.5	215	68.9	309	-	-	-	-	-	-	-	-	-	-
Beryllium	µg/g	0.22	0.23	0.19	0.23	0.24	0.25	-	-	-	-	-	-	-	-	-	-
Cadmium	µg/g	0.146	0.108	0.131	0.123	0.12	0.166	-	-	-	-	-	-	-	-	-	-
Chromium	µg/g	3.02	3.19	3.24	3.25	3.25	3.17	-	-	-	-	-	-	-	-	-	-
Chromium (+6)	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Chromium (+3)	µg/g	3.02	3.19	3.24	3.25	3.25	3.17	-	-	-	-	-	-	-	-	-	-
Cobalt	µg/g	1.46	1.41	1.49	1.48	1.49	1.56	-	-	-	-	-	-	-	-	-	-
Copper	µg/g	11.1	10.6	11.4	11.2	11.7	12	-	-	-	-	-	-	-	-	-	-
Lead	µg/g	2.65	2.47	1.89	2.59	2.88	2.8	-	-	-	-	-	-	-	-	-	-
Lithium	µg/g	< 5	< 5	< 5	< 5	< 5	< 5	-	-	-	-	-	-	-	-	-	-
Manganese	µg/g	18.8	18	19.3	18.6	19.5	20.2	-	-	-	-	-	-	-	-	-	-
Mercury	µg/g	0.0924	0.0913	0.098	0.0961	0.0888	0.0865	-	-	-	-	-	-	-	-	-	-
Molybdenum	µg/g	0.58	0.62	0.46	0.59	0.63	0.56	-	-	-	-	-	-	-	-	-	-
Nickel	µg/g	5.21	5.02	5.39	5.45	5.47	5.7	-	-	-	-	-	-	-	-	-	-
Selenium	µg/g	0.62	0.74	0.77	0.8	0.9	0.73	-	-	-	-	-	-	-	-	-	-
Silver	µg/g	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	-	-	-	-	-	-	-	-	-	-
Strontium	µg/g	153	150	122	159	161	172	-	-	-	-	-	-	-	-	-	-
Tin	µg/g	< 0.2	< 0.2	< 0.2	0.23	< 0.2	< 0.2	-	-	-	-	-	-	-	-	-	-
Uranium	µg/g	0.414	0.43	0.32	0.438	0.445	0.448	-	-	-	-	-	-	-	-	-	-
Vanadium	µg/g	11.8	11.4	11.6	12	11.7	12.2	-	-	-	-	-	-	-	-	-	-
Zinc	µg/g	16.5	15.4	16.4	15.3	17.6	17.8	-	-	-	-	-	-	-	-	-	-
Aluminum	µg/g	2,570	2,450	2,570	2,520	2,470	2,560	-	-	-	-	-	-	-	-	-	-
Bismuth	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Boron	µg/g	18	17	14	20	19	18	-	-	-	-	-	-	-	-	-	-
Calcium	µg/g	7,670	7,790	6,300	7,870	8,290	8,400	-	-	-	-	-	-	-	-	-	-
Iron	µg/g	2,800	2,980	3,220	3,220	3,350	3,350	-	-	-	-	-	-	-	-	-	-
Magnesium	µg/g	1,380	1,310	1,370	1,370	1,340	1,420	-	-	-	-	-	-	-	-	-	-
Phosphorus	µg/g	315	276	315	280	312	320	-	-	-	-	-	-	-	-	-	-
Potassium	µg/g	100	< 100	< 100	< 100	100	100	-	-	-	-	-	-	-	-	-	-
Sodium	µg/g	640	610	630	640	630	640	-	-	-	-	-	-	-	-	-	-
Thallium	µg/g	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	-	-	-	-	-	-	-	-	-	-
Titanium	µg/g	155	148	164	172	161	162	-	-	-	-	-	-	-	-	-	-

Associated ALS files: L1424156.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

TABLE VII-2: Summary of Analytical Results for PAHs and Total Metals in CUST-B COAL

Sample Location		CUST-B-1															
Sample ID		CUST-B-1-01	CUST-B-1-02	CUST-B-1-03	CUST-B-1-04	CUST-B-1-05	CUST-B-1-06	CUST-B-1-11	CUST-B-1-12	CUST-B-1-13	CUST-B-1-14	CUST-B-1-15	CUST-B-1-16	CUST-B-1-17	CUST-B-1-18	CUST-B-1-19	CUST-B-1-20
Sample Date (yyyy mm dd)		2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19	2014 02 19
Parameter	**Units**	**Analytical Results**															
Physical Parameters																	
pH	pH	7.25	7.29	7.2	7.23	7.25	7.23	-	-	-	-	-	-	-	-	-	-
Polycyclic Aromatic Hydrocarbons																	
Naphthalene	µg/g	-	-	-	-	-	-	< 0.05	< 0.05	< 0.05	0.052	< 0.05	< 0.05	< 0.05	0.052	0.051	0.053
2-Methylnaphthalene	µg/g	-	-	-	-	-	-	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05
Acenaphthylene	µg/g	-	-	-	-	-	-	0.12	0.12	0.12	0.128	0.116	0.106	0.114	0.125	0.123	0.13
Acenaphthene	µg/g	-	-	-	-	-	-	0.115	0.115	0.132	0.144	0.098	0.115	0.122	0.091	0.128	0.129
Fluorene	µg/g	-	-	-	-	-	-	< 0.1	< 0.2	< 0.1	< 0.2	< 0.09	< 0.1	< 0.2	< 0.2	< 0.2	< 0.2
Phenanthrene	µg/g	-	-	-	-	-	-	0.413	0.445	0.471	0.512	0.417	0.397	0.502	0.473	0.499	0.473
Anthracene	µg/g	-	-	-	-	-	-	0.194	0.19	0.208	0.267	0.204	0.188	0.239	0.238	0.221	0.229
Fluoranthene	µg/g	-	-	-	-	-	-	1.41	1.23	1.3	1.53	1.29	1.26	1.39	1.44	1.45	1.52
Pyrene	µg/g	-	-	-	-	-	-	2.22	2.17	2.26	2.55	2.2	2.04	2.32	2.49	2.36	2.49
Benzo(a)anthracene	µg/g	-	-	-	-	-	-	0.661	0.681	0.772	0.87	0.704	0.679	0.778	0.755	0.778	0.845
Chrysene	µg/g	-	-	-	-	-	-	0.425	0.337	0.482	0.497	0.45	0.3	0.358	0.291	0.375	0.444
Benzo(b)fluoranthene	µg/g	-	-	-	-	-	-	0.93	0.971	1.13	1.2	1.08	1.02	1.16	1.19	1.22	1.31
Benzo(b+j+k)fluoranthene	µg/g	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Benzo(k)fluoranthene	µg/g	-	-	-	-	-	-	0.226	0.148	0.173	0.18	0.18	0.16	0.15	0.224	0.193	0.201
Benzo(a)pyrene	µg/g	-	-	-	-	-	-	0.254	0.29	0.314	0.315	0.296	0.296	0.325	0.329	0.332	0.342
Indeno(1,2,3-cd)pyrene	µg/g	-	-	-	-	-	-	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.1	< 0.2	< 0.2	< 0.2	< 0.2
Dibenz(a,h)anthracene	µg/g	-	-	-	-	-	-	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05
Benzo(g,h,i)perylene	µg/g	-	-	-	-	-	-	0.071	0.068	0.074	0.073	0.061	0.059	0.073	0.073	0.065	0.07
Total Metals																	
Antimony	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Arsenic	µg/g	1.24	1.2	1.4	1.35	1.38	1.19	-	-	-	-	-	-	-	-	-	-
Barium	µg/g	200	289	824	912	784	446	-	-	-	-	-	-	-	-	-	-
Beryllium	µg/g	0.19	0.19	0.23	0.19	0.2	0.19	-	-	-	-	-	-	-	-	-	-
Cadmium	µg/g	0.078	0.094	0.09	0.102	0.082	0.082	-	-	-	-	-	-	-	-	-	-
Chromium	µg/g	3.11	2.92	2.83	2.96	2.5	2.67	-	-	-	-	-	-	-	-	-	-
Chromium (+6)	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Chromium (+3)	µg/g	3.11	2.92	2.83	2.96	2.5	2.67	-	-	-	-	-	-	-	-	-	-
Cobalt	µg/g	1.07	1.07	1.12	1.02	1.03	0.96	-	-	-	-	-	-	-	-	-	-
Copper	µg/g	12.5	23.6	20.9	10.4	9.87	9.55	-	-	-	-	-	-	-	-	-	-
Lead	µg/g	1.83	1.9	2.05	1.91	1.89	1.75	-	-	-	-	-	-	-	-	-	-
Lithium	µg/g	< 5	< 5	< 5	< 5	< 5	< 5	-	-	-	-	-	-	-	-	-	-
Manganese	µg/g	19.7	21.9	21.8	18.3	18.2	17.2	-	-	-	-	-	-	-	-	-	-
Mercury	µg/g	0.0597	0.0592	0.0704	0.0688	0.0656	0.0629	-	-	-	-	-	-	-	-	-	-
Molybdenum	µg/g	0.45	0.44	0.46	0.43	0.43	0.4	-	-	-	-	-	-	-	-	-	-
Nickel	µg/g	2.72	2.68	2.59	2.82	2.6	2.59	-	-	-	-	-	-	-	-	-	-
Selenium	µg/g	0.53	0.51	0.6	0.54	0.55	0.48	-	-	-	-	-	-	-	-	-	-
Silver	µg/g	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	-	-	-	-	-	-	-	-	-	-
Strontium	µg/g	282	283	305	288	302	279	-	-	-	-	-	-	-	-	-	-
Tin	µg/g	0.49	0.2	< 0.2	< 0.2	< 0.2	< 0.2	-	-	-	-	-	-	-	-	-	-
Uranium	µg/g	0.287	0.285	0.306	0.286	0.293	0.285	-	-	-	-	-	-	-	-	-	-
Vanadium	µg/g	9.14	9.24	9.85	9.27	9.26	8.82	-	-	-	-	-	-	-	-	-	-
Zinc	µg/g	10	11.6	11.4	10.6	9.8	10.4	-	-	-	-	-	-	-	-	-	-
Aluminum	µg/g	2,040	1,950	2,090	2,000	2,030	1,960	-	-	-	-	-	-	-	-	-	-
Bismuth	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	-	-	-	-	-	-	-	-	-	-
Boron	µg/g	16	15	20	19	18	16	-	-	-	-	-	-	-	-	-	-
Calcium	µg/g	5,780	5,940	6,140	5,620	5,910	5,610	-	-	-	-	-	-	-	-	-	-
Iron	µg/g	1,850	1,850	2,080	1,960	1,910	1,770	-	-	-	-	-	-	-	-	-	-
Magnesium	µg/g	1,170	1,160	1,200	1,150	1,180	1,140	-	-	-	-	-	-	-	-	-	-
Phosphorus	µg/g	57	75	72	58	74	68	-	-	-	-	-	-	-	-	-	-
Potassium	µg/g	130	120	150	120	130	120	-	-	-	-	-	-	-	-	-	-
Sodium	µg/g	2,830	2,620	2,830	2,790	2,830	2,700	-	-	-	-	-	-	-	-	-	-
Thallium	µg/g	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	-	-	-	-	-	-	-	-	-	-
Titanium	µg/g	152	152	167	156	158	141	-	-	-	-	-	-	-	-	-	-

Associated ALS files: L1424155.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

TABLE VII-3: Summary of Analytical Results for Crystalline Silica in CUST-A and CUST-B

Sample ID	Silica		
	Quartz (%)	Cristobalite (%)	Tridymite (%)
LOD (%)[1]	0.75	1	1
RL (%)[1]	1.5	1.5	1.5
CUST-A-1-21	< 0.75	< 1.0	< 1.0
CUST-A-1-22	< 0.75	< 1.0	< 1.0
CUST-A-1-23	< 0.75	< 1.0	< 1.0
CUST-A-1-24	< 0.75	< 1.0	< 1.0
CUST-A-1-25	< 0.75	< 1.0	< 1.0
CUST-B-1-21	< 0.75	< 1.0	< 1.0
CUST-B-1-22	< 0.75	< 1.0	< 1.0
CUST-B-1-23	< 0.75	< 1.0	< 1.0
CUST-B-1-24	< 0.75	< 1.0	< 1.0
CUST-B-1-25	< 0.75	< 1.0	< 1.0

[1] LOD = Limit of Detection, RL = Reporting Limit

TABLE VII-4: Summary of Results for Physiologically Based Extraction Test (PBET) for Arsenic and Lead - CUST-A and CUST-B Coal

	PBET Results	
Sample ID	Arsenic Bioaccessibility (%)	Lead Bioaccessibility (%)
CUST-A-1-26	10.1	11.0
CUST-A-1-27	8.9	11.6
CUST-A-1-28	11.0	11.9
CUST-B-1-26	17.5	27.8
CUST-B-1-27	15.1	26.3
CUST-B-1-28	15.2	26.7

TABLE VII-5: Summary of Analytical Results for PAHs and Total Metals in Background Surface Soil Samples - Delta

Sample Location		SS-DEL											BC Standards			
Sample ID		SS-DEL-01	SS-DEL-02	SS-DEL-03	SS-DEL-04	SS-DEL-05	SS-DEL-06	SS-DEL-07	SS-DEL-08	SS-DEL-09	SS-DEL-10	SS-DEL-11	CSR Agricultural Land Use[a] (AL)	CSR Residential Land Use (RL)	CCME CEQG Agricultural Land Use (AL)	CCME CEQG Residential Land Use (RL)
Sample Date (yyyy mm dd)		2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25				
Parameter	**Units**	**Analytical Results**														
Physical Parameters																
pH	pH	6.17	8.45	8.5	6.1	6.47	7.63	6.56	5.78	5.65	7.05	6	n/a	n/a	6 - 8	6 - 8
Polycyclic Aromatic Hydrocarbons																
Naphthalene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.1	5	0.6	0.6
2-Methylnaphthalene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	n/a	n/a	n/a	n/a
Acenaphthylene	µg/g	< 0.005	< 0.005	< 0.005	0.0069	< 0.005	< 0.005	0.0136	< 0.005	< 0.005	< 0.005	0.0072	n/a	n/a	n/a	n/a
Acenaphthene	µg/g	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	0.0067	< 0.005	< 0.005	< 0.005	< 0.005	n/a	n/a	n/a	n/a
Fluorene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	n/a	n/a	n/a	n/a
Phenanthrene	µg/g	0.014	< 0.01	0.028	0.027	< 0.01	< 0.01	0.103	< 0.01	< 0.01	0.019	0.021	0.1	5	0.1	5
Anthracene	µg/g	< 0.004	< 0.004	0.0057	0.0051	< 0.004	< 0.004	0.019	< 0.004	< 0.004	< 0.004	0.0059	n/a	n/a	2.5	2.5
Fluoranthene	µg/g	0.022	0.011	0.026	0.028	< 0.01	< 0.01	0.114	0.012	< 0.01	0.023	0.051	n/a	n/a	50	50
Pyrene	µg/g	0.021	< 0.01	0.053	0.029	< 0.01	< 0.01	0.144	0.011	< 0.01	0.016	0.045	0.1	10	0.1	10
Benzo(a)anthracene	µg/g	< 0.01	< 0.01	0.014	0.015	< 0.01	< 0.01	0.065	< 0.01	< 0.01	< 0.01	0.029	0.1	1	0.1	1
Chrysene	µg/g	0.013	< 0.01	0.023	0.02	< 0.01	< 0.01	0.1	< 0.01	< 0.01	0.012	0.038	n/a	n/a	n/a	n/a
Benzo(b)fluoranthene	µg/g	0.021	0.012	0.025	0.026	< 0.01	< 0.01	0.096	< 0.01	0.014	0.017	0.07	0.1	1	0.1	1
Benzo(b+j+k)fluoranthene	µg/g	0.021	< 0.015	0.025	0.026	< 0.015	< 0.015	0.127	< 0.015	< 0.015	0.017	0.092	n/a	n/a	n/a	n/a
Benzo(k)fluoranthene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.031	< 0.01	< 0.01	< 0.01	0.022	0.1	1	0.1	1
Benzo(a)pyrene	µg/g	0.014	< 0.01	0.012	0.023	< 0.01	< 0.02	0.075	< 0.01	< 0.02	0.01	0.035	5	5	20	20
Indeno(1,2,3-cd)pyrene	µg/g	0.01	< 0.01	< 0.01	0.012	< 0.01	< 0.01	0.044	< 0.01	< 0.01	< 0.01	0.035	0.1	1	0.1	1
Dibenz(a,h)anthracene	µg/g	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	0.0113	< 0.005	< 0.005	< 0.005	0.0072	0.1	1	0.1	1
Benzo(g,h,i)perylene	µg/g	0.016	< 0.01	0.014	0.012	< 0.01	< 0.01	0.045	< 0.01	< 0.01	< 0.01	0.03	n/a	n/a	n/a	n/a
Index of Additive Cancer Risk	None	-0.24	-0.15	-0.29	-0.33	< -0.15	< -0.15	-1.31	< -0.15	-0.18	-0.2	-0.82	n/a	n/a	n/a	n/a
B(a)P Equivalency	µg/g	0.021	< 0.02	0.02	0.031	< 0.02	< 0.02	0.111	< 0.02	< 0.02	< 0.02	0.059	n/a	n/a	n/a	n/a
Total Metals																
Antimony	µg/g	0.49	0.69	0.16	0.51	0.17	0.33	0.99	0.24	0.31	0.43	0.51	20	20	20	20
Arsenic	µg/g	5.67	2.93	1.92	10.3	3.34	6.31	9	3.69	4.32	7.56	5.97	100	100	12	12
Barium	µg/g	53	47.2	44.1	166	57	28.5	68.2	43.4	42.3	63.5	71.5	6,500	6,500	750	500
Beryllium	µg/g	0.24	< 0.2	< 0.2	0.32	< 0.2	< 0.2	0.31	< 0.2	< 0.2	0.29	0.29	4	4	4	4
Cadmium	µg/g	0.202	0.095	0.121	0.206	0.112	0.804	0.428	0.123	0.145	0.231	0.156	35	35	1.4	10
Chromium	µg/g	28.8	15.7	16.4	40.5	17.7	7.91	36.3	21.1	27.9	33.5	31.9	100	100	64	64
Chromium (+6)	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	100	100	0.4	0.4
Chromium (+3)	µg/g	28.8	15.7	16.3	40.5	17.7	7.88	36.2	21	27.9	33.4	31.9	n/a	n/a	n/a	n/a
Cobalt	µg/g	10.4	7.34	6.98	12.6	8.06	4.1	11.1	7.2	8.88	10	9.02	40	50	40	50
Copper	µg/g	24.6	25.5	21.3	34.9	20.3	18.1	37.2	17.5	16.6	32.3	25.9	15,000	15,000	63	63
Lead	µg/g	31.9	4.2	2.68	21.6	2.32	3.87	64.9	22.7	10.1	11	27.5	400	400	70	140
Lithium	µg/g	11.8	8.5	9	17.7	10.8	< 5.0	18	9.5	9.1	11.9	13.3	1,600	1,600	n/a	n/a
Manganese	µg/g	499	315	306	371	373	177	415	340	405	400	301	1,800	1,800	n/a	n/a
Mercury	µg/g	0.0924	0.0061	0.0053	0.0644	0.0106	0.047	0.0554	0.0235	0.0334	0.0592	0.0445	15	15	6.6	6.6
Molybdenum	µg/g	1.27	< 0.5	< 0.5	1.37	< 0.5	< 0.5	2.27	0.64	0.63	1.09	1.34	5	10	5	10
Nickel	µg/g	24.5	11.2	11.4	35.9	17.4	10.2	30.9	18	32	32	30.7	150	100	50	50
Selenium	µg/g	< 0.2	< 0.2	< 0.2	0.25	< 0.2	0.3	0.35	< 0.2	< 0.2	< 0.2	0.22	2	3	1	1
Silver	µg/g	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	0.13	< 0.1	< 0.1	< 0.1	< 0.1	20	20	20	20
Strontium	µg/g	36	42.5	43.3	40.5	23.9	445	39.6	23.3	27.3	43.8	30.5	47,000	47,000	n/a	n/a
Tin	µg/g	< 2	< 2	< 2	< 2	< 2	< 2	2.5	< 2	< 2	4.2	< 2	5	50	5	50
Uranium	µg/g	0.899	0.325	0.358	0.93	0.318	1.19	1.64	0.597	0.374	0.754	1.23	16	16	23	23
Vanadium	µg/g	48.2	48.9	51.4	58.6	47.5	14.5	54.5	42.1	41.7	50.2	53.8	200	200	130	130
Zinc	µg/g	80.1	43.9	41.6	87.6	42.9	142	163	50.2	64.6	83.9	66.5	10,000	10,000	200	200
Aluminum	µg/g	13,200	12,200	11,800	16,600	12,000	3,620	16,600	10,900	10,000	14,000	15,300	n/a	n/a	n/a	n/a
Bismuth	µg/g	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	n/a	n/a	n/a	n/a
Boron	µg/g	-	-	-	-	-	-	-	-	-	-	-	2	n/a	n/a	n/a
Calcium	µg/g	6,140	12,200	11,700	5,860	5,500	247,000	6,490	4,120	5,230	8,670	4,760	n/a	n/a	n/a	n/a
Iron	µg/g	21,800	18,800	18,300	29,400	19,300	7,660	27,300	18,600	18,300	24,500	21,400	n/a	n/a	n/a	n/a
Magnesium	µg/g	6,370	6,230	5,990	8,940	6,270	3,200	7,690	5,630	6,490	7,360	6,680	n/a	n/a	n/a	n/a
Phosphorus	µg/g	698	517	480	1,240	636	558	1,140	648	873	1,050	615	n/a	n/a	n/a	n/a
Potassium	µg/g	1,340	860	940	2,410	1,200	520	1,470	950	1,410	1,450	1,270	n/a	n/a	n/a	n/a
Sodium	µg/g	350	480	500	420	370	< 100	470	240	230	320	290	n/a	n/a	n/a	n/a
Thallium	µg/g	0.08	< 0.05	< 0.05	0.097	0.051	< 0.05	0.086	< 0.05	0.053	0.079	0.105	2	n/a	1	1
Titanium	µg/g	772	1,010	1,060	957	920	171	774	695	736	795	803	n/a	n/a	n/a	n/a

Associated ALS files: L1426040.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

SHADED	Concentration greater than CSR Agricultural Land Use (AL) standard.	OUTLINE	Concentration greater than or equal to CCME CEQG Agricultural Land Use (AL) guideline.
BOLD	Concentration greater than CSR Residential Land Use (RL) standard.	UNDERLINE	Concentration greater than or equal to CCME CEQG Residential Land Use (RL) guideline.

[a] The site-specific factors used for determining the matrix standards for this site include:intake of contaminated soil (whichever is most stringent).

TABLE VII-6: Summary of Analytical Results for PAHs and Total Metals in Background Surface Soil Samples - Richmond

Sample Location		SS-RICH											BC Standards			
Sample ID		SS-RICH-01	SS-RICH-02	SS-RICH-03	SS-RICH-04	SS-RICH-05	SS-RICH-06	SS-RICH-07	SS-RICH-08	SS-RICH-09	SS-RICH-10	SS-RICH-11	CSR Agricultural Land Use[a] (AL)	CSR Residential Land Use (RL)	CCME CEQG Agricultural Land Use (AL)	CCME CEQG Residential Land Use (RL)
Sample Date (yyyy mm dd)		2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26				
Parameter	**Units**	**Analytical Results**														
Physical Parameters																
pH	pH	6.66	6.91	6.81	6.35	6.52	7.11	7.01	7.92	6.75	7.78	7.32	n/a	n/a	6 - 8	6 - 8
Polycyclic Aromatic Hydrocarbons																
Naphthalene	µg/g	< 0.01	0.014	< 0.01	0.466	< 0.01	0.021	0.013	< 0.01	< 0.01	< 0.01	< 0.04	0.1	5	0.6	0.6
2-Methylnaphthalene	µg/g	< 0.01	0.013	< 0.01	0.431	< 0.01	0.011	< 0.01	< 0.01	< 0.01	< 0.01	< 0.04	n/a	n/a	n/a	n/a
Acenaphthylene	µg/g	< 0.005	0.105	0.0144	< 0.03	0.0062	0.174	0.159	< 0.005	0.0222	< 0.005	< 0.03	n/a	n/a	n/a	n/a
Acenaphthene	µg/g	< 0.005	0.005	< 0.005	0.253	< 0.005	< 0.009	< 0.02	< 0.005	< 0.005	< 0.005	< 0.04	n/a	n/a	n/a	n/a
Fluorene	µg/g	< 0.01	< 0.02	< 0.01	0.375	< 0.01	< 0.02	< 0.04	< 0.01	< 0.01	< 0.01	< 0.04	n/a	n/a	n/a	n/a
Phenanthrene	µg/g	< 0.01	0.101	0.074	2	0.048	0.225	0.379	0.024	0.041	< 0.01	0.051	0.1	5	0.1	5
Anthracene	µg/g	< 0.004	0.0555	0.0124	0.454	0.0072	0.087	0.0883	0.0053	0.0143	< 0.004	< 0.04	n/a	n/a	2.5	2.5
Fluoranthene	µg/g	< 0.01	0.363	0.125	0.93	0.058	0.617	0.913	0.052	0.119	< 0.01	0.143	n/a	n/a	50	50
Pyrene	µg/g	< 0.01	0.516	0.115	1.21	0.063	0.694	0.97	0.046	0.124	0.01	0.144	0.1	10	0.1	10
Benzo(a)anthracene	µg/g	< 0.01	0.279	0.052	0.593	0.022	0.461	0.528	0.023	0.08	< 0.01	0.065	0.1	1	0.1	1
Chrysene	µg/g	< 0.01	0.315	0.073	0.687	0.034	0.559	0.601	0.033	0.092	< 0.01	0.098	n/a	n/a	n/a	n/a
Benzo(b)fluoranthene	µg/g	< 0.01	0.37	0.104	0.52	0.043	0.632	0.782	0.054	0.142	0.013	0.212	0.1	1	0.1	1
Benzo(b+j+k)fluoranthene	µg/g	< 0.015	0.523	0.139	0.736	0.06	0.891	1.08	0.071	0.193	< 0.015	0.287	n/a	n/a	n/a	n/a
Benzo(k)fluoranthene	µg/g	< 0.01	0.153	0.036	0.215	0.017	0.258	0.296	0.017	0.051	< 0.01	0.076	0.1	1	0.1	1
Benzo(a)pyrene	µg/g	< 0.01	0.252	0.055	0.432	0.026	0.529	0.604	0.023	0.096	< 0.01	0.121	5	5	20	20
Indeno(1,2,3-cd)pyrene	µg/g	< 0.01	0.163	0.041	0.193	0.02	0.394	0.438	0.019	0.068	< 0.01	0.116	0.1	1	0.1	1
Dibenz(a,h)anthracene	µg/g	< 0.005	0.0505	0.0087	0.0624	< 0.005	0.0872	0.0877	< 0.005	0.0147	< 0.005	0.027	0.1	1	0.1	1
Benzo(g,h,i)perylene	µg/g	< 0.01	0.156	0.037	0.172	0.02	0.35	0.363	0.028	0.061	< 0.01	0.151	n/a	n/a	n/a	n/a
Index of Additive Cancer Risk	None	< -0.15	-5.25	-1.27	-8.26	-0.55	-9.24	-10.8	-0.61	-1.85	-0.16	-2.55	n/a	n/a	n/a	n/a
B(a)P Equivalency	µg/g	< 0.02	0.404	0.089	0.655	0.039	0.8	0.905	0.037	0.147	< 0.02	0.197	n/a	n/a	n/a	n/a
Total Metals																
Antimony	µg/g	0.78	0.47	0.68	1.28	0.39	0.46	0.45	0.8	0.67	0.28	0.71	20	20	20	20
Arsenic	µg/g	6.71	6.43	5.75	7.93	4.13	5.44	4.88	9.14	6	2.34	4.85	100	100	12	12
Barium	µg/g	65.5	55.6	74.1	71.1	56.9	52.8	64.8	29.7	58.6	77.9	116	6,500	6,500	750	500
Beryllium	µg/g	0.23	0.24	0.28	0.27	0.23	0.22	0.25	< 0.2	< 0.2	0.23	0.23	4	4	4	4
Cadmium	µg/g	0.45	0.253	0.211	0.337	0.169	0.201	0.237	0.152	0.25	0.077	0.786	35	35	1.4	10
Chromium	µg/g	27.2	32.8	31.6	37.8	27.8	32.4	28.3	12.5	30.2	9.08	19	100	100	64	64
Chromium (+6)	µg/g	< 0.1	< 0.1	< 0.2	< 0.1	< 0.2	< 0.1	< 0.1	< 0.1	< 0.2	< 0.1	< 0.1	100	100	0.4	0.4
Chromium (+3)	µg/g	27.1	32.8	31.6	37.8	27.7	32.4	28.3	12.5	30.2	9.05	19	n/a	n/a	n/a	n/a
Cobalt	µg/g	12	8.12	7.75	8.48	7.75	8.99	8.69	2.34	7.64	6.77	6.71	40	50	40	50
Copper	µg/g	48.2	24.4	31.5	65.1	22.2	26.2	25.7	11.7	26.7	11.1	30.4	15,000	15,000	63	63
Lead	µg/g	9.67	34	19.5	101	11.7	12.5	13.3	17	14.1	4.28	92.7	400	400	70	140
Lithium	µg/g	15.3	11.7	13.3	15.6	8	11.8	10.9	< 5	8.3	< 5	7.3	1,600	1,600	n/a	n/a
Manganese	µg/g	706	307	277	303	294	280	263	144	305	473	371	1,800	1,800	n/a	n/a
Mercury	µg/g	0.0292	0.0443	0.0457	0.0609	0.0522	0.038	0.0393	0.0404	0.0364	0.0157	0.0518	15	15	6.6	6.6
Molybdenum	µg/g	0.71	1.04	1.21	2.03	0.84	1.01	1.07	0.55	0.85	< 0.5	1.08	5	10	5	10
Nickel	µg/g	22.2	26.7	27.1	28.1	29.8	30.5	29.1	6.43	26.4	6.71	13.9	150	100	50	50
Selenium	µg/g	0.4	0.2	0.3	0.28	0.2	0.24	0.22	0.25	< 0.2	< 0.2	0.29	2	3	1	1
Silver	µg/g	0.14	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	20	20	20	20
Strontium	µg/g	31.8	29.3	37.2	41.4	30.4	32.4	32.3	556	31.2	49	129	47,000	47,000	n/a	n/a
Tin	µg/g	< 2	< 2	< 2	2.7	< 2	2	< 2	< 2	< 2	< 2	< 2	5	50	5	50
Uranium	µg/g	0.365	0.987	1.25	1.9	0.702	0.899	0.794	1.7	0.68	0.28	0.822	16	16	23	23
Vanadium	µg/g	69.5	47.1	46.9	50.9	40.5	47.5	43.3	17.8	41.3	46.7	37.3	200	200	130	130
Zinc	µg/g	138	66	78.9	127	74.5	73.6	77.8	105	71.4	45	401	10,000	10,000	200	200
Aluminum	µg/g	16,400	12,100	14,400	15,300	9,820	12,000	11,100	2,590	10,400	15,600	12,800	n/a	n/a	n/a	n/a
Bismuth	µg/g	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	n/a	n/a	n/a	n/a
Boron	µg/g	-	-	-	-	-	-	-	-	-	-	-	2	n/a	n/a	n/a
Calcium	µg/g	6,780	6,700	8,540	7,400	5,770	7,320	6,760	278,000	7,360	13,900	50,300	n/a	n/a	n/a	n/a
Iron	µg/g	28,900	22,000	20,200	24,200	17,900	21,400	19,500	4,980	18,900	22,100	17,100	n/a	n/a	n/a	n/a
Magnesium	µg/g	10,100	7,540	6,300	6,760	6,350	7,340	6,470	2,660	6,390	7,120	5,830	n/a	n/a	n/a	n/a
Phosphorus	µg/g	1,030	922	689	869	626	744	827	314	852	691	1,500	n/a	n/a	n/a	n/a
Potassium	µg/g	850	1,350	880	1,500	860	1,240	1,220	160	850	660	910	n/a	n/a	n/a	n/a
Sodium	µg/g	280	290	310	380	210	240	200	< 100	210	280	270	n/a	n/a	n/a	n/a
Thallium	µg/g	< 0.05	0.07	0.077	0.079	< 0.05	0.058	0.063	0.052	0.05	< 0.05	0.056	2	n/a	1	1
Titanium	µg/g	1,090	792	774	807	746	787	621	128	716	677	498	n/a	n/a	n/a	n/a

Associated ALS files: L1426592.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

SHADED Concentration greater than CSR Agricultural Land Use (AL) standard.

BOLD Concentration greater than CSR Residential Land Use (RL) standard.

OUTLINE Concentration greater than or equal to CCME CEQG Agricultural Land Use (AL) guideline.

UNDERLINE Concentration greater than or equal to CCME CEQG Residential Land Use (RL) guideline.

[a] The site-specific factors used for determining the matrix standards for this site include:intake of contaminated soil (whichever is most stringent).

TABLE VII-7: Summary of Analytical Results for PAHs and Total Metals in Background Surface Soil Samples - Surrey

Sample Location		SS-SUR													BC Standards			
Sample ID		SS-SUR-01	SS-SUR-02	SS-SUR-03	SS-SUR-04	SS-SUR-05	SS-SUR-06	SS-SUR-07	SS-SUR-08	SS-SUR-09	SS-SUR-10	SS-SUR-11	SS-SUR-12	SS-SUR-13	CSR Agricultural Land Use[a] (AL)	CSR Residential Land Use (RL)	CCME CEQG Agricultural Land Use (AL)	CCME CEQG Residential Land Use (RL)
Sample Date (yyyy mm dd)		2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25	2014 02 25				
Parameter	**Units**	**Analytical Results**																
Physical Parameters																		
pH	pH	7.74	7.68	6.1	8.03	6.06	6	7.65	7.32	6.14	6.74	6.95	6.01	5.33	n/a	n/a	6 - 8	6 - 8
Polycyclic Aromatic Hydrocarbons																		
Naphthalene	µg/g	0.029	< 0.01	0.02	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.1	5	0.6	0.6
2-Methylnaphthalene	µg/g	0.106	< 0.01	0.021	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	n/a	n/a	n/a	n/a
Acenaphthylene	µg/g	0.0717	0.0343	0.0772	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	0.0071	< 0.005	0.024	< 0.005	n/a	n/a	n/a	n/a
Acenaphthene	µg/g	0.529	< 0.005	0.0303	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	n/a	n/a	n/a	n/a
Fluorene	µg/g	0.735	0.01	0.047	< 0.01	< 0.01	< 0.02	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.01	< 0.01	n/a	n/a	n/a	n/a
Phenanthrene	µg/g	**19.3**	0.071	0.684	< 0.01	0.01	0.014	< 0.01	0.015	0.012	0.033	< 0.01	0.101	< 0.01	0.1	5	0.1	5
Anthracene	µg/g	2	0.0301	0.139	< 0.004	< 0.004	< 0.004	< 0.004	< 0.004	< 0.004	0.0066	< 0.004	0.219	< 0.004	n/a	n/a	2.5	2.5
Fluoranthene	µg/g	34	0.135	0.918	< 0.01	0.02	0.029	< 0.01	0.026	0.023	0.097	0.013	0.122	< 0.01	n/a	n/a	50	50
Pyrene	µg/g	**25.7**	0.147	1.01	< 0.01	0.016	0.024	< 0.01	0.025	0.021	0.068	0.011	0.115	< 0.01	0.1	10	0.1	10
Benzo(a)anthracene	µg/g	**11.9**	0.081	0.432	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.01	0.021	< 0.01	0.069	< 0.01	0.1	1	0.1	1
Chrysene	µg/g	13.8	0.093	0.466	< 0.01	0.012	0.017	< 0.01	0.018	0.017	0.061	< 0.01	0.15	< 0.01	n/a	n/a	n/a	n/a
Benzo(b)fluoranthene	µg/g	**22.4**	0.203	0.695	< 0.01	0.022	0.035	< 0.01	0.027	0.023	0.115	0.013	0.221	< 0.01	0.1	1	0.1	1
Benzo(b+j+k)fluoranthene	µg/g	31.6	0.264	0.943	< 0.015	0.022	0.035	< 0.015	0.027	0.023	0.148	< 0.015	0.287	< 0.015	n/a	n/a	n/a	n/a
Benzo(k)fluoranthene	µg/g	**9.19**	0.061	0.248	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.033	< 0.01	0.066	< 0.01	0.1	1	0.1	1
Benzo(a)pyrene	µg/g	**14.6**	0.111	0.472	< 0.01	< 0.01	0.013	< 0.01	0.017	< 0.02	0.049	< 0.01	0.071	< 0.01	5	5	20	20
Indeno(1,2,3-cd)pyrene	µg/g	**9.64**	0.091	0.294	< 0.01	0.011	0.014	< 0.01	0.012	0.012	0.054	< 0.01	0.089	< 0.01	0.1	1	0.1	1
Dibenz(a,h)anthracene	µg/g	**2.01**	0.0206	0.0715	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	0.0099	< 0.005	0.0174	< 0.005	0.1	1	0.1	1
Benzo(g,h,i)perylene	µg/g	9.21	0.094	0.267	< 0.01	< 0.01	0.014	< 0.02	0.013	0.014	0.056	< 0.01	0.074	< 0.01	n/a	n/a	n/a	n/a
Index of Additive Cancer Risk	None	-293	-2.38	-9.16	< -0.15	-0.22	-0.33	< -0.15	-0.28	-0.26	-1.22	-0.16	-2.39	< -0.15	n/a	n/a	n/a	n/a
B(a)P Equivalency	µg/g	22.1	0.177	0.718	< 0.02	< 0.02	0.021	< 0.02	0.024	< 0.02	0.083	< 0.02	0.135	< 0.02	n/a	n/a	n/a	n/a
Total Metals																		
Antimony	µg/g	1.95	0.48	0.38	0.31	0.52	0.54	0.24	0.38	0.41	0.93	0.4	0.59	0.51	20	20	20	20
Arsenic	µg/g	5.77	8.82	4.28	5.8	3.58	4.59	3.33	5.44	3.39	4.13	5.07	7.47	7.49	100	100	12	12
Barium	µg/g	76.9	58.9	48.4	45.1	82.2	84.4	58.6	55.1	65	127	127	74.3	57.2	6,500	6,500	750	500
Beryllium	µg/g	0.26	< 0.2	< 0.2	< 0.2	0.41	0.43	0.21	< 0.2	0.21	0.22	0.36	0.27	0.31	4	4	4	4
Cadmium	µg/g	0.314	0.147	0.16	0.146	0.213	0.208	0.09	0.22	0.146	0.394	0.09	0.288	0.128	35	35	1.4	10
Chromium	µg/g	22.1	26.4	27.1	17.1	23.9	25.9	22.3	18.4	21.8	24.9	34.8	25.6	33.1	100	100	64	64
Chromium (+6)	µg/g	< 0.1	< 0.2	< 0.1	< 0.1	< 1[b]	< 1[b]	0.19	< 0.1	< 0.1	< 0.2	< 0.1	< 0.1	< 0.1	100	100	0.4	0.4
Chromium (+3)	µg/g	22.1	26.4	27.1	17.1	23.9	25.9	22.2	18.4	21.8	24.9	34.8	25.6	33.1	n/a	n/a	n/a	n/a
Cobalt	µg/g	7.98	6.23	6.45	5.45	3.93	4.33	7.44	5.83	6.44	6.65	12.1	8.43	5.82	40	50	40	50
Copper	µg/g	33.2	25.2	24.2	17.3	24.6	25.2	17.5	17.8	18.6	49.8	26.1	30	27.8	15,000	15,000	63	63
Lead	µg/g	34.1	14.5	58.9	10.2	15.8	20.2	21	51.7	12	29.6	7.95	43.1	11.4	400	400	70	140
Lithium	µg/g	7.5	6.4	7	6.2	6.9	7.5	6.7	8	7.6	6.2	11.1	7.5	13.5	1,600	1,600	n/a	n/a
Manganese	µg/g	461	329	307	271	206	218	418	391	337	426	531	756	310	1,800	1,800	n/a	n/a
Mercury	µg/g	0.0746	0.0841	0.0361	0.0369	0.12	0.109	0.0248	0.0642	0.0533	0.0664	0.0444	0.087	0.084	15	15	6.6	6.6
Molybdenum	µg/g	1.12	0.52	0.66	0.5	0.64	0.68	< 0.5	< 0.5	0.51	0.99	0.52	1.4	8.23	5	10	5	10
Nickel	µg/g	15.8	18.1	15.7	14.6	14.2	16.3	20.9	14.2	15.8	22.8	28.8	17.9	16.6	150	100	50	50
Selenium	µg/g	0.28	< 0.2	< 0.2	< 0.2	1	0.9	< 0.2	0.21	0.27	< 0.2	< 0.2	0.4	0.75	2	3	1	1
Silver	µg/g	0.11	< 0.1	< 0.1	< 0.1	0.11	0.1	< 0.1	< 0.1	< 0.1	0.11	< 0.1	0.12	0.11	20	20	20	20
Strontium	µg/g	132	64.6	22.9	212	33.1	30.8	29.6	26.9	28.3	71.6	39	31	26.1	47,000	47,000	n/a	n/a
Tin	µg/g	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	5	50	5	50
Uranium	µg/g	0.641	0.381	0.295	0.735	0.741	0.759	0.32	0.361	0.323	0.493	0.462	0.461	5.21	16	16	23	23
Vanadium	µg/g	39.8	42.4	43.7	33.3	31.4	36.2	47.1	46.2	48.2	32.9	64.8	45.6	59.8	200	200	130	130
Zinc	µg/g	105	64.1	68.5	46.6	112	92.2	44.4	85.8	51.3	151	56.6	235	52.6	10,000	10,000	200	200
Aluminum	µg/g	15,900	12,400	14,000	9,650	23,000	24,200	13,200	15,300	16,700	10,500	23,700	17,900	19,800	n/a	n/a	n/a	n/a
Bismuth	µg/g	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	n/a	n/a	n/a	n/a
Boron	µg/g	-	-	-	-	-	-	-	-	-	-	-	-	-	2	n/a	n/a	n/a
Calcium	µg/g	44,600	21,000	4,090	83,600	7,050	6,620	8,900	10,400	4,000	13,700	5,260	5,650	3,290	n/a	n/a	n/a	n/a
Iron	µg/g	17,700	15,800	16,600	13,700	10,800	12,700	19,700	17,100	17,200	14,900	25,700	18,500	24,700	n/a	n/a	n/a	n/a
Magnesium	µg/g	4,290	4,410	4,740	4,480	2,600	2,900	6,460	4,390	4,010	4,820	7,070	4,120	4,680	n/a	n/a	n/a	n/a
Phosphorus	µg/g	1,150	763	565	540	1,170	1,070	474	719	473	1,140	428	1,220	867	n/a	n/a	n/a	n/a
Potassium	µg/g	740	720	690	580	620	650	680	580	790	670	1,320	670	1,240	n/a	n/a	n/a	n/a
Sodium	µg/g	150	230	240	160	120	130	330	210	180	280	190	150	220	n/a	n/a	n/a	n/a
Thallium	µg/g	0.059	0.069	< 0.05	< 0.05	0.067	0.067	< 0.05	0.053	0.052	0.051	0.069	0.075	0.078	2	n/a	1	1
Titanium	µg/g	661	800	757	551	503	573	883	834	874	571	1,260	691	846	n/a	n/a	n/a	n/a

Associated ALS files: L1426039.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

SHADED Concentration greater than CSR Agricultural Land Use (AL) standard.

BOLD Concentration greater than CSR Residential Land Use (RL) standard.

OUTLINE Concentration greater than or equal to CCME CEQG Agricultural Land Use (AL) guideline.

UNDERLINE Concentration greater than or equal to CCME CEQG Residential Land Use (RL) guideline.

[a] The site-specific factors used for determining the matrix standards for this site include:intake of contaminated soil (whichever is most stringent).

[b] Laboratory detection limit exceeds regulatory standard.

TABLE VII-8: Summary of Analytical Results for PAHs and Total Metals in Background Surface Soil Samples - White Rock

Sample Location		SS-WR													BC Standards			
Sample ID		SS-WR-01	SS-WR-02	SS-WR-03	SS-WR-04	SS-WR-05	SS-WR-06	SS-WR-07	SS-WR-08	SS-WR-09	SS-WR-10	SS-WR-11	SS-WR-12	SS-WR-13	CSR Agricultural Land Use[a] (AL)	CSR Residential Land Use (RL)	CCME CEQG Agricultural Land Use (AL)	CCME CEQG Residential Land Use (RL)
Sample Date (yyyy mm dd)		2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26	2014 02 26				
Parameter	**Units**	**Analytical Results**																
Physical Parameters																		
pH	pH	6.6	7.65	7.45	7.92	6.39	5.34	5.25	5.88	7.33	5.55	4.73	5.63	7.18	n/a	n/a	6 - 8	6 - 8
Polycyclic Aromatic Hydrocarbons																		
Naphthalene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.033	0.02	0.1	5	0.6	0.6
2-Methylnaphthalene	µg/g	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	0.038	0.021	n/a	n/a	n/a	n/a
Acenaphthylene	µg/g	0.0158	< 0.005	0.158	< 0.005	0.0052	0.0054	0.0073	0.0152	0.0097	< 0.005	< 0.005	0.04	0.122	n/a	n/a	n/a	n/a
Acenaphthene	µg/g	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	< 0.005	0.0458	0.0111	n/a	n/a	n/a	n/a
Fluorene	µg/g	< 0.01	< 0.01	< 0.02	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01	< 0.02	0.088	0.053	n/a	n/a	n/a	n/a
Phenanthrene	µg/g	0.063	0.013	0.123	< 0.01	0.04	0.032	0.038	0.031	0.02	0.022	0.018	0.563	0.486	0.1	5	0.1	5
Anthracene	µg/g	0.0152	< 0.004	0.0751	< 0.004	0.0071	0.0062	0.0067	0.0081	0.0049	0.0053	< 0.004	0.144	0.107	n/a	n/a	2.5	2.5
Fluoranthene	µg/g	0.094	0.028	0.784	< 0.01	0.078	0.032	0.038	0.077	0.035	0.03	0.015	0.429	0.554	n/a	n/a	50	50
Pyrene	µg/g	0.09	0.026	0.774	< 0.01	0.072	0.034	0.041	0.079	0.041	0.032	0.016	0.509	0.657	0.1	10	0.1	10
Benzo(a)anthracene	µg/g	0.036	0.012	0.493	< 0.01	0.035	0.016	0.018	0.053	0.024	0.014	< 0.01	0.271	0.379	0.1	1	0.1	1
Chrysene	µg/g	0.04	< 0.02	0.526	< 0.01	< 0.06	0.021	0.026	0.067	< 0.04	< 0.02	< 0.02	0.302	0.424	n/a	n/a	n/a	n/a
Benzo(b)fluoranthene	µg/g	0.053	0.03	0.681	< 0.01	0.065	0.025	0.026	0.097	0.047	0.034	0.021	0.315	0.377	0.1	1	0.1	1
Benzo(b+j+k)fluoranthene	µg/g	0.075	0.03	0.921	< 0.015	0.084	0.025	0.037	0.132	0.061	0.034	0.021	0.429	0.532	n/a	n/a	n/a	n/a
Benzo(k)fluoranthene	µg/g	0.022	< 0.01	0.239	< 0.01	0.019	< 0.01	0.011	0.035	0.014	< 0.01	< 0.01	0.114	0.155	0.1	1	0.1	1
Benzo(a)pyrene	µg/g	0.041	< 0.02	0.447	< 0.01	0.041	< 0.02	0.022	0.074	0.028	0.018	< 0.02	0.227	0.354	5	5	20	20
Indeno(1,2,3-cd)pyrene	µg/g	0.025	0.015	0.279	< 0.01	0.049	0.012	0.013	0.041	0.021	0.012	< 0.01	0.115	0.218	0.1	1	0.1	1
Dibenz(a,h)anthracene	µg/g	0.0056	< 0.005	0.0676	< 0.005	< 0.02	< 0.005	< 0.005	0.0094	0.0055	< 0.005	< 0.005	0.0333	0.0525	0.1	1	0.1	1
Benzo(g,h,i)perylene	µg/g	0.027	0.019	0.207	< 0.01	0.048	< 0.01	0.011	0.032	0.022	0.012	< 0.01	0.094	0.212	n/a	n/a	n/a	n/a
Index of Additive Cancer Risk	None	-0.75	-0.31	-9.13	< -0.15	-0.83	-0.29	-0.38	-1.28	-0.57	-0.36	-0.22	-4.46	-5.97	n/a	n/a	n/a	n/a
B(a)P Equivalency	µg/g	0.061	< 0.02	0.691	< 0.02	0.069	< 0.02	0.032	0.107	0.044	0.027	< 0.02	0.345	0.526	n/a	n/a	n/a	n/a
Total Metals																		
Antimony	µg/g	0.42	1.01	0.3	0.35	0.29	0.23	0.24	0.32	0.39	0.35	0.29	0.49	0.36	20	20	20	20
Arsenic	µg/g	5.11	6.49	5.69	18	4.93	5.56	4.65	4.54	11.7	4.85	17.6	5.2	3.26	100	100	12	12
Barium	µg/g	77.3	79.3	79.9	28.8	57.8	51.2	47.8	74.9	67.7	53.5	78.9	69.6	58.6	6,500	6,500	750	500
Beryllium	µg/g	0.25	0.26	< 0.2	< 0.2	0.26	< 0.2	< 0.2	0.35	< 0.2	< 0.2	< 0.2	0.25	0.21	4	4	4	4
Cadmium	µg/g	0.159	0.306	0.153	0.091	0.205	0.077	0.082	0.218	0.253	0.151	0.166	0.104	0.158	35	35	1.4	10
Chromium	µg/g	40.7	35.4	24.7	19.1	24.2	25.3	23.2	26.9	27.7	24.8	26.4	27.7	24.6	100	100	64	64
Chromium (+6)	µg/g	< 0.1	< 0.1	< 0.1	0.15	< 0.1	< 0.1	< 0.1	< 0.1	< 0.2	< 0.1	< 0.2	< 0.1	< 0.1	100	100	0.4	0.4
Chromium (+3)	µg/g	40.6	35.4	24.6	19	24.2	25.3	23.2	26.9	27.7	24.8	26.4	27.7	24.6	n/a	n/a	n/a	n/a
Cobalt	µg/g	8.95	7.53	8.78	2.6	7.2	6.02	5.93	8.14	5.15	6.61	6.12	7.43	7.25	40	50	40	50
Copper	µg/g	17.8	29.3	23.9	14.2	15.2	17.7	16.7	15.7	18	21.5	13.4	21.5	22.6	15,000	15,000	63	63
Lead	µg/g	27.1	45	11	10.4	9.87	5.05	5.86	14.1	7.8	18.1	9.06	18.4	8.21	400	400	70	140
Lithium	µg/g	9.2	8.2	10.2	< 5	8.9	6.3	7.2	9.2	6.9	6.9	7.8	8.4	8.3	1,600	1,600	n/a	n/a
Manganese	µg/g	308	369	404	150	358	252	249	502	304	361	395	354	432	1,800	1,800	n/a	n/a
Mercury	µg/g	0.0434	0.0543	0.0197	0.0655	0.0502	0.0262	0.0196	0.08	0.089	0.046	0.0697	0.0624	0.0365	15	15	6.6	6.6
Molybdenum	µg/g	0.88	0.85	< 0.5	< 0.5	0.81	< 0.5	< 0.5	0.95	1.3	1.85	2.09	< 0.5	2.14	5	10	5	10
Nickel	µg/g	25.6	23.7	22.7	7.89	21.8	23.4	23	19.4	18.7	23.6	20	21.9	19.9	150	100	50	50
Selenium	µg/g	< 0.2	0.32	< 0.2	0.43	0.31	< 0.2	< 0.2	0.52	0.57	< 0.2	0.57	< 0.2	< 0.2	2	3	1	1
Silver	µg/g	< 0.1	0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	20	20	20	20
Strontium	µg/g	40.3	45.2	50.7	292	18.3	16.5	15.4	29.4	94.9	18.3	29.4	26.2	32.4	47,000	47,000	n/a	n/a
Tin	µg/g	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	5	50	5	50
Uranium	µg/g	0.494	0.411	0.307	0.999	0.445	0.271	0.264	0.454	0.77	0.245	0.434	0.304	0.366	16	16	23	23
Vanadium	µg/g	56	48.3	46.3	26.6	47.7	45	41.5	46.9	35.4	43	42.2	48.8	46.2	200	200	130	130
Zinc	µg/g	65.8	97.7	62.1	20.4	57.5	44.6	40.4	38.9	70.2	54.6	54.8	55.2	49.6	10,000	10,000	200	200
Aluminum	µg/g	14,400	16,800	12,700	9,170	16,400	12,300	10,900	14,700	8,940	11,300	11,900	13,800	12,100	n/a	n/a	n/a	n/a
Bismuth	µg/g	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	n/a	n/a	n/a	n/a
Boron	µg/g	-	-	-	-	-	-	-	-	-	-	-	-	-	2	n/a	n/a	n/a
Calcium	µg/g	7,030	9,380	16,300	152,000	3,410	3,170	3,420	4,620	28,400	3,710	4,570	4,370	6,240	n/a	n/a	n/a	n/a
Iron	µg/g	23,200	20,100	20,400	10,800	19,600	17,700	16,400	19,800	13,200	16,800	17,000	18,500	17,200	n/a	n/a	n/a	n/a
Magnesium	µg/g	6,290	5,500	7,160	1,820	4,620	5,820	6,270	3,300	4,610	5,110	4,140	4,730	5,040	n/a	n/a	n/a	n/a
Phosphorus	µg/g	557	899	783	479	913	632	612	563	2,270	782	846	658	637	n/a	n/a	n/a	n/a
Potassium	µg/g	1,180	930	1,230	210	850	560	540	980	790	670	500	880	900	n/a	n/a	n/a	n/a
Sodium	µg/g	340	260	350	< 100	190	220	180	110	280	170	150	190	220	n/a	n/a	n/a	n/a
Thallium	µg/g	0.071	0.065	0.068	< 0.05	< 0.05	< 0.05	< 0.05	0.066	0.05	0.051	< 0.05	0.053	< 0.05	2	n/a	1	1
Titanium	µg/g	1,030	779	794	395	823	781	790	778	516	639	667	829	779	n/a	n/a	n/a	n/a

Associated ALS files: L1426591.

All terms defined within the body of SNC-Lavalin's report.

< Denotes concentration less than indicated detection limit or RPD less than indicated value.

- Denotes analysis not conducted.

n/a Denotes no applicable standard.

SHADED Concentration greater than CSR Agricultural Land Use (AL) standard.

OUTLINE Concentration greater than or equal to CCME CEQG Agricultural Land Use (AL) guideline.

BOLD Concentration greater than CSR Residential Land Use (RL) standard.

UNDERLINE Concentration greater than or equal to CCME CEQG Residential Land Use (RL) guideline.

[a] The site-specific factors used for determining the matrix standards for this site include:intake of contaminated soil (whichever is most stringent).

APPENDIX VIII

SNC·LAVALIN

Sensitivity Analysis: Supporting Information

TABLE VIII-A : Sensitivity Analysis, Project Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Toddler)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project using Mean Coal Concentraitons

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	3.7E-04	2.8E-10	1.3E-06	1.0E-06	3.8E-04	5.9E-08	5.1E-08	4.7E-12	1.7E-07	2.1E-07	6.4E-06	4.9E-07	6.9E-06
Benzo[b]fluoranthene	1.4E-04	1.1E-10	7.1E-07	3.7E-07	2.6E-04	2.2E-08	1.9E-08	1.8E-12	9.6E-08	7.8E-08	4.4E-06	2.2E-07	4.6E-06
Carcinogenic PAH Mixture											3.1E-05	3.9E-06	2.6E-05
Metals and Metalloids													
Barium	5.4E+00	4.1E-06	1.2E-01	1.2E-01	1.1E-01	1.3E-04	1.1E-04	4.1E-06	2.5E-03	3.8E-03	1.1E-01	6.6E-03	1.2E-01
Mercury	1.2E-03	9.1E-10	1.6E-04	1.6E-04	5.8E-06	1.9E-05	1.7E-05	3.0E-09	2.2E-03	3.4E-03	1.9E-05	5.7E-03	5.7E-03
Uranium	5.4E-03	4.1E-09	7.0E-06	6.9E-06	1.8E-05	4.4E-05	3.8E-05	5.1E-09	4.8E-05	7.3E-05	2.2E-05	2.0E-04	2.2E-04

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE VIII-B: Sensitivity Analysis, Project Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Toddler)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project using 95% UCLM Coal Concentrations

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	HQ Soil Ingestion	HQ Soil Dermal	HQ Soil Dust Inhalation	HQ Plant (Aboveground)	HQ Plant (Belowground)	HQ Air Inhalation	HQ Soil / Vegetation	HQ All Routes	Relative Percent Difference of Overall HQ between 95% UCLM and Mean Coal Concentrations
Polycyclic Aromatic Hydrocarbons														
Carcinogenic PAHs														
Benzo[a]anthracene	4.1E-04	3.1E-10	1.4E-06	1.1E-06	3.8E-04	6.6E-08	5.6E-08	5.18E-12	1.9E-07	2.3E-07	6.4E-06	5.5E-07	7.0E-06	1%
Benzo[b]fluoranthene	1.6E-04	1.2E-10	8.1E-07	4.2E-07	2.6E-04	2.6E-08	2.2E-08	2.02E-12	1.1E-07	8.9E-08	4.4E-06	2.5E-07	4.7E-06	1%
Carcinogenic PAH Mixture													2.6E-05	0%
Metals and Metalloids														
Barium	7.9E+00	6.0E-06	1.8E-01	1.8E-01	1.1E-01	1.9E-04	1.6E-04	6.01E-06	3.6E-03	5.7E-03	1.1E-01	9.6E-03	1.2E-01	3%
Mercury	1.3E-03	1.0E-09	1.8E-04	1.8E-04	5.8E-06	2.1E-05	1.8E-05	3.34E-09	2.4E-03	3.8E-03	1.9E-05	6.2E-03	6.3E-03	10%
Uranium	6.0E-03	4.5E-09	7.8E-06	7.6E-06	1.8E-05	4.8E-05	4.2E-05	5.69E-09	5.3E-05	8.1E-05	2.2E-05	2.2E-04	2.5E-04	10%

HQ = Hazard Quotient

NA = not applicable, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2

TABLE VIII-C : Sensitivity Analysis, Project Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Adult)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project Using Arithmetic Mean Coal Concentrations

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	ILCR Soil Ingestion	ILCR Soil Dermal	ILCR Soil Dust Inhalation	ILCR Plant (Aboveground)	ILCR Plant (Belowground)	ILCR Air Inhalation	ILCR All Routes
Polycyclic Aromatic Hydrocarbons												
Carcinogenic PAHs												
Benzo[a]anthracene	3.7E-04	2.8E-10	1.3E-06	1.0E-06	3.8E-04	2.4E-11	3.0E-11	8.6E-16	5.6E-10	6.1E-10	1.5E-10	1.4E-09
Benzo[b]fluoranthene	1.4E-04	1.1E-10	7.1E-07	3.7E-07	2.6E-04	9.0E-12	1.1E-11	3.3E-16	3.2E-10	2.3E-10	1.0E-10	6.7E-10
Carcinogenic PAH Mixture												1.9E-08
Metals and Metalloids												
Barium	5.4E+00	4.1E-06	1.2E-01	1.2E-01	1.1E-01	NA	NA	NA	NA	NA	NA	NA
Mercury	1.2E-03	9.1E-10	1.6E-04	1.6E-04	5.8E-06	NA	NA	NA	NA	NA	NA	NA
Uranium	5.4E-03	4.1E-09	7.0E-06	6.9E-06	1.8E-05	NA	NA	NA	NA	NA	NA	NA

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

[1] maximum outdoor air concentration assumed equal to the analytical detection limit

TABLE VIII-D : Sensitivity Analysis, Project Scenario Risk Estimates for the Maximum North Delta Residential Receptor (Adult)
(Based on Maximum Multi-Media Concentrations)

Scenario: Project Using 95% UCLM Coal Concentrations

	Soil Concentration µg/g	Soil Dust Concentration µg/m^3	Plant Concentration (Aboveground) µg/g	Plant Concentration (Belowground) µg/g	Air Concentration µg/m^3	ILCR Soil Ingestion	ILCR Soil Dermal	ILCR Soil Dust Inhalation	ILCR Plant (Aboveground)	ILCR Plant (Belowground)	ILCR Air Inhalation	ILCR All Routes	Relative Percent Difference of Overall ILCR between 95% UCLM and Mean Coal Concentrations
Polycyclic Aromatic Hydrocarbons													
Carcinogenic PAHs													
Benzo[a]anthracene	4.1E-04	3.1E-10	1.4E-06	1.1E-06	3.8E-04	2.6E-11	3.3E-11	9.6E-16	6.2E-10	6.8E-10	1.5E-10	1.5E-09	10%
Benzo[b]fluoranthene	1.6E-04	1.2E-10	8.1E-07	4.2E-07	2.6E-04	1.0E-11	1.3E-11	3.7E-16	3.6E-10	2.6E-10	1.0E-10	7.4E-10	12%
Carcinogenic PAH Mixture												1.9E-08	1%
Metals and Metalloids													
Barium	7.9E+00	6.0E-06	1.8E-01	1.8E-01	1.1E-01	NA	NA	NA	NA	NA	NA	NA	NA
Mercury	1.3E-03	1.0E-09	1.8E-04	1.8E-04	5.8E-06	NA	NA	NA	NA	NA	NA	NA	NA
Uranium	6.0E-03	4.5E-09	7.8E-06	7.6E-06	1.8E-05	NA	NA	NA	NA	NA	NA	NA	NA

HQ = Hazard Quotient

ILCR = Incremental Lifetime Cancer Risk

NA = not applicable, not a carcinogen, no COPC identified for the media/pathway, or suitable TRV not identified, see report text for details

Bold HQ > 0.2 ILCR >1E-05

[1] maximum outdoor air concentration assumed equal to the analytical detection limit