SCIENCE INTEGRITY KNOWLEDGE

HIGHLAND CREEK TREATMENT PLANT (HCTP) CLASS ENVIRONMENTAL ASSESSMENT

HUMAN HEALTH RISK ASSESSMENT (HHRA) REPORT

FINAL REPORT

October 19, 2015

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HIGHLAND CREEK TREATMENT PLANT (HCTP) CLASS EA
HUMAN HEALTH RISK ASSESSMENT (HHRA) REPORT

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List of Abbreviations

AAQC	Ambient Air Quality Criteria
ABTP	City of Toronto's Ashbridges Bay Treatment Plant
ADI	Acceptable Daily Intake
AQBAT	Health Canada's Air Quality Benefits Assessment Tool
ATSDR	Agency for Toxic Substances and Disease Registry
CACs	Criteria Air Contaminants
Cal EPA	California Environmental Protection Agency
CCME	Canadian Council of Ministers of the Environment
COC	Compound of Concern
CR	Concentration Ratio
CRF	Concentration Response Function
CSM	Conceptual Site Model
EA	Environmental Assessment
EPC	Exposure Point Concentration
TEF	Toxic Equivalency Factor
HCTP	Highland Creek Treatment Plant
HHRA	Human Health Risk Assessment
HQ	Hazard Quotient
ILCR	Incremental Lifetime Cancer Risk
LADD	Lifetime Average Daily Dose
LCR	Lifetime Cancer Risk
MEA	Municipal Engineers Association
MPOI	Maximum Point of Impingement
MOECC	Ontario Ministry of the Environment and Climate Change
NAAQO	National Ambient Air Quality Objective
NOAEL	No Observable Adverse Effect Level
PACs	Priority Air Contaminants
PAH	Polycyclic Aromatic Hydrocarbon
PEQ	Potency Equivalence Factors
PM	Particulate Matter
RfC	Reference Concentration
RfD	Reference Dose
SF	Slope Factor
TCEQ	Texas Commission on Environmental Quality
TDI	Tolerable Daily Intake
TEQ	Toxicity Equivalence Factors
TRV	Toxicological Reference Value
UR	Unit Risk
US EPA	United States Environmental Protection Agency
WHO	World Health Organization

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EXECUTIVE SUMMARY

Overview of the Study

The City of Toronto has four wastewater treatment plants, including the Highland Creek Treatment Plant (HCTP), with a rated capacity of 219,000 cubic metres per day (219 ML/d) that services approximately 500,000 people in the southeast portion of the City. The plant provides conventional activated sludge treatment and discharges treated effluent to Lake Ontario. Residue sludge from the wastewater treatment process is treated biologically by anaerobic digestion and mechanically processed to remove a significant portion of water. The resulting treated material, referred to as “biosolids”, is similar in appearance to a wet soil, and has high organic and nutrient content.

Approximately 40,000 cubic metres of dewatered biosolids are produced each year at the HCTP. Currently, the biosolids are processed in two multiple-hearth incinerators which have been operating for approximately 38 years, and are approaching the end of their service life. In order to provide continued safe operation consistent within applicable regulatory standards, the City initiated a major maintenance and refurbishment program for the incinerators. This work is underway; however, this will extend the life of the equipment for a maximum of 10 years. As such, the City has initiated a process to examine all viable biosolids management alternatives to select a preferred solution that would provide long-term reliability for the HCTP and its surrounding community.

The primary purpose of this project is to meet the requirements of the Municipal Engineers Association (MEA) Class Environmental Assessment (Class EA) process to identify a preferred approach for managing the biosolids generated at the HCTP. To address concerns with respect to potential human health impacts related to the management of biosolids, each of the potential management approaches were evaluated through the use of a Health Impact Assessment (HIA) framework. A key element of the proposed HIA was a quantitative evaluation of health risks related to potential exposures to chemicals released during the treatment or transportation of biosolids. The quantification of potential chemical health risks was conducted through the use of the human health risk assessment (HHRA) paradigm.

The primary objective of the HHRA was to determine the potential short- and long-term human health risks to individuals in the surrounding community who may be impacted by emissions from any of the proposed biosolids management alternatives. The HHRA has also undergone third party peer review by experts at both Toronto Public Health and Public Health Ontario.

What is a Human Health Risk Assessment (HHRA)?

In general, an HHRA is a scientific study that evaluates the potential for the occurrence of adverse health effects from exposures of people to chemicals of concern (COC) present in surrounding environmental media (*e.g.*, air, soil, food, *etc*.), under existing or predicted exposure conditions arising from the operation of the Project under review.

HHRA procedures are based on the fundamental dose-response principle of toxicology. The response of an individual to a chemical exposure increases in proportion to the chemical concentration in critical target tissues where adverse effects may occur. The concentrations of chemicals in the target tissues (the dose) depend on the chemical concentrations in the environment where the receptor resides, works or visits.

All chemicals (both natural and man-made) have the potential to cause effects in people and the ecosystem. It is the chemical concentration, the route and amount of exposure, and the inherent toxicity of the chemical that determines the level of risk for adverse health effects to occur. Where technically and economically feasible, methods can be used to mitigate adverse effects. It is acknowledged that the various uncertainties associated with the HHRA process have the potential to influence estimates of exposure and risk. The methods and assumptions used in this HHRA were designed to be highly cautious (*i.e.,* health protective), and have a built-in tendency to overestimate, rather than underestimate, potential health risks.

The HHRA carried out for the Project followed the standard HHRA framework that is composed of the following general steps:

I. Problem formulation;
II. Exposure assessment;
III. Hazard assessment; and,
IV. Risk characterization.

The HHRA was conducted according to widely accepted risk assessment methodologies and guidance documents published and endorsed by regulatory agencies including the Ontario Ministry of the Environment and Climate Change, Health Canada, and the United States Environmental Protection Agency. Intrinsik consulted with Toronto Public Health (TPH) during the development of this HHRA and has made every effort to address the concerns and recommendations provided by TPH to ensure consistency with previous Local Air Quality (LAQ) assessments completed by the City and related health-based policies.

What are the Proposed Preferred Biosolids Management Alternatives?

Based on the initial review conducted as part of the EA process, the following short-listed biosolids management alternatives have been selected for evaluation through the EA, including the HHRA:

1. On-site fluidized bed incineration and off-site ash management;
2. Transporting biosolids off-site for further management; and,
3. On-site processing of biosolids into pellets (a fertilizer product) and transporting pellets off-site for further management.

Each of these three biosolids treatment alternatives were compared to predicted health risks related to the existing conditions arising from the operation of the current HCTP multiple hearth incinerators. Each alternative was also "added" to the existing background air quality conditions in the Wards to get a sense of the "cumulative risks" for each option within the Study Area.

Based on these short-listed biosolids management options, the following table provides a list of the project alternatives that were evaluated in the HHRA.

Table ES-1 List of Evaluated Project Scenarios based on Short-Listed Biosolids Treatment Alternatives

Project Scenario	Description
Base Case	Existing multiple hearth incineration (*i.e.*, baseline, current conditions)
Alternative 1	New fluidized bed incineration
Alternative 2a	Off-site haulage of biosolids along Haul Route 1
Alternative 2b	Off-site haulage of biosolids along Haul Route 4
Alternative 3a	On-site pelletization plus off-site haulage along Haul Route 1
Alternative 3b	On-site pelletization plus off-site haulage along Haul Route 4

Both the Base Case and Alternative 1 incinerator scenarios also include the contribution of truck traffic for two weeks annually when accumulated incineration bottom ash is removed off-site for landfill disposal.

Who are the Sensitive Receptors in the Surrounding Area?

The area surrounding the HCTP is composed of mixed industrial, parkland and residential uses. To assess potential risks related to the projected emissions from the either on-site emission sources or transportation route emission sources for off-site management, the project team selected key sensitive locations representative of the surrounding community.

So as to avoid identifying specific residential properties within the HHRA, the entire Study Area was broken down into a grid of exposure areas where similar exposure conditions would be expected. This allowed the evaluation of any trends of potential exposures and related health risks associated with emissions arising from the various short-listed biosolids management alternatives. Each grid area is up to 1 km^2 in size, depending on where it is located (as the wards are not completely square).

The following two figures provide an overview of the individual receptor grid locations within the Study Area evaluating the emission impacts from proposed facility-based and haul route sources.

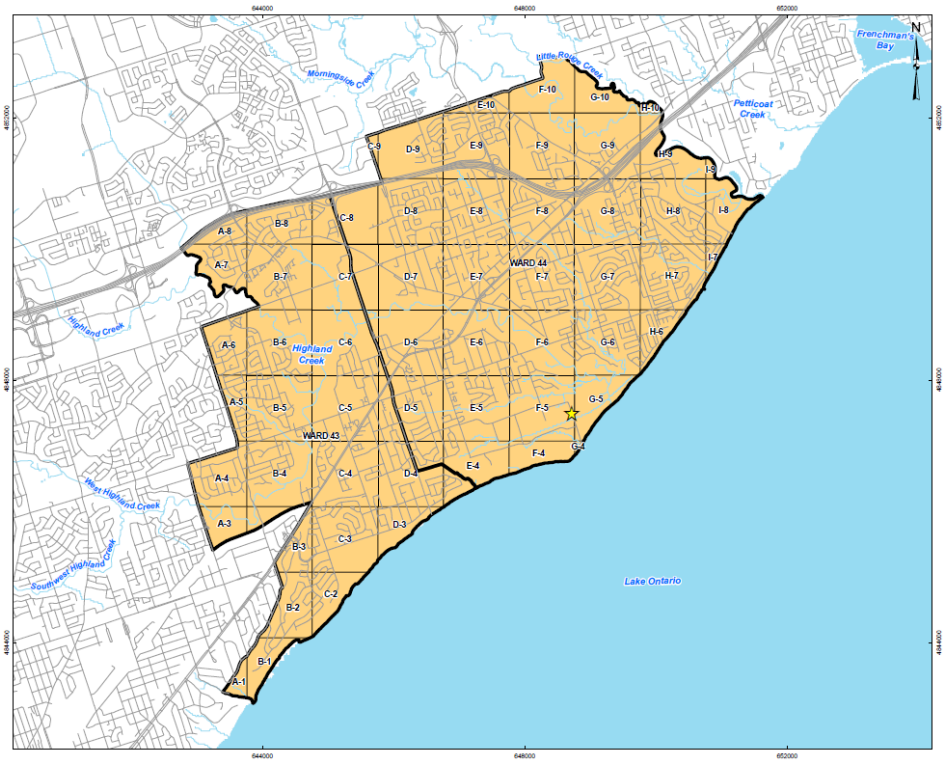


Figure ES-1 Facility Emission Receptor Grid Locations within Study Area

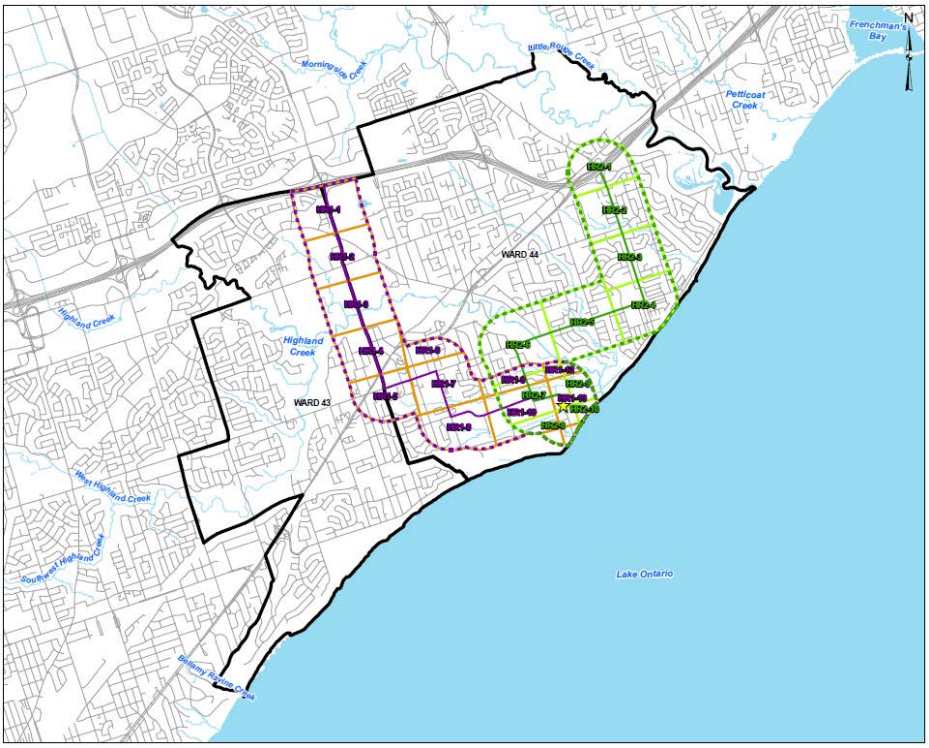


Figure ES-2 Haul Route Receptor Grid Locations within Study Area

For the transportation alternatives, a total of six potential routes from the HCTP to the nearest Highway 401 intersections were evaluated based on fifteen criteria related to safety, operations and community impact. As a result of the evaluation, the two highest ranked options were from the plant *via* Coronation, Manse, Lawrence and Morningside (*i.e.*, Haul Route 1, or HR1) and from the plant *via* Beechgrove Drive, Lawrence Avenue and Port Union Road (*i.e.*, Haul Route 4, or HR4). The receptor grid locations for HR1 and HR4 are labelled as purple and green, respectively, in the figure above.

For the purpose of the current assessment, and to ensure a conservative approach to evaluating risk, a residential scenario was considered in each of the receptor grid locations outlined in above figures based on the maximum ground-level air concentrations predicted for those locations. These worst-case exposures were used in the HHRA to estimate potential health risks related to individuals living within that grid area.

How were Potential Exposures Evaluated?

The HHRA included an inhalation assessment that evaluated short- and long-term health risk (*i.e*., exposure over a long duration *via* direct air inhalation) at each of the receptor grid locations noted in the figures above for all COC. An individual's exposure (*via* inhalation) was assumed to equal the predicted ground-level air concentration (expressed as $\mu g/m^3$) for a particular chemical, duration and location. Health risk estimates were subsequently calculated by directly comparing predicted ground-level air concentrations with the appropriate inhalation toxicity reference values.

Two specific exposure conditions were evaluated:

- *Project Alone* exposures; and,
- *Cumulative* exposures.

The *Project Alone* assessment evaluates the potential health impact related to the predicted ground-level air concentrations of each of the COCs contributed by each of the proposed biosolids management alternatives to off-site residential locations in the surrounding community.

The *Cumulative* assessment evaluates the potential health impact related to the predicted ground-level air concentrations of each of the COCs contributed by the proposed biosolids management alternative **plus** the existing background ambient concentrations of the COC based on the Project Team's modelling of local air quality within the Study Area.

In some cases, a number of the chemicals will settle over time and could accumulate in residential soils and home gardens within the Study Area. These particular chemicals were also carried through a multimedia assessment, where the following exposure pathways were considered:

- **Inhalation:** Inhalation of air impacted by vapours and particulate emitted from the Project-related sources were evaluated.
- **Incidental Ingestion of Soil and Dust**: Through typical indoor and outdoor activities, individuals may accidentally ingest soil and/or dust particles. Children are typically more susceptible to this exposure pathway, as they spend more time in contact with the ground, and are more likely to put soiled articles, such as toys or hands, into their mouths.
- **Incidental Inhalation of Indoor Dust**: Soils impacted by particles emitted from the Project-related sources were assumed to be carried indoors (*e.g.*, by wind, or human and/pet activities) and present as indoor suspended dust for inhalation by individuals living within the home.
- **Dermal Exposure to Soils and Dusts**: Dermal exposures of human receptors may occur in both indoor and outdoor environments, through direct dermal contact with chemically impacted soil and dust.
- **Ingestion of Locally Grown Produce**: Locally grown produce (such as vegetables and fruits grown in backyard gardens) may itself pose a source of exposure to some COCs. As chemicals are deposited from air-borne emissions, they may come into contact with leaves and fruit of crop plants. Deposition of chemicals onto soil may also result in an accumulation in plants through root uptake.

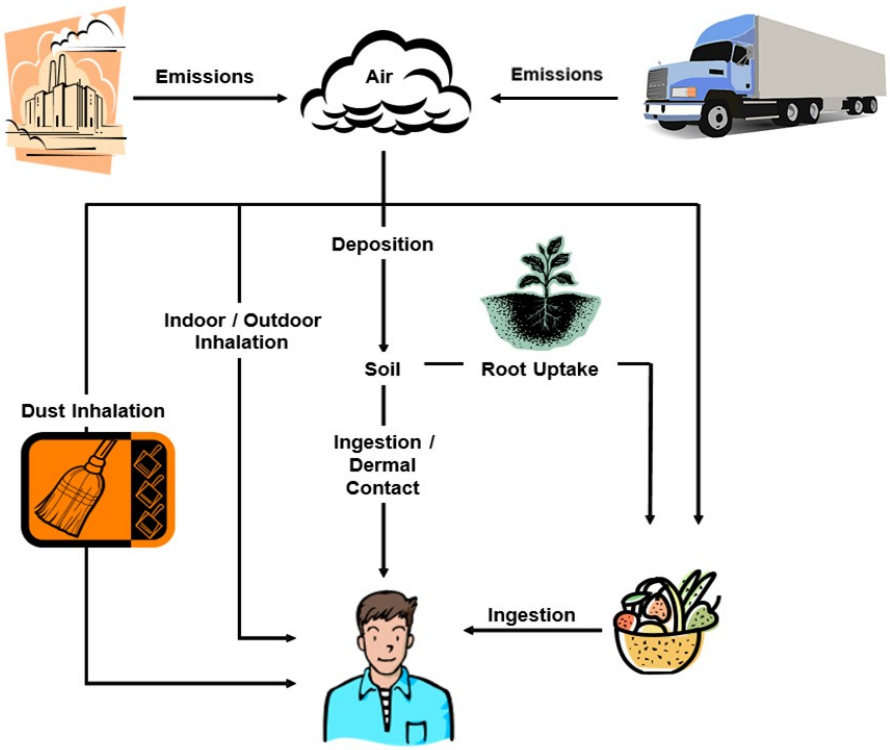


Figure ES-3 Residential Exposure Scenario

What Chemicals were evaluated in the HHRA?

A key element for both the air quality assessment and HHRA components of the HCTP EA was the development of a robust and defensible list of chemicals emitted from the various short-listed biosolids management alternatives under consideration. It should be noted the methodology ultimately used to identify COCs was developed in consultation with Toronto Public Health.

The City of Toronto routinely assesses the health impacts related to 30 key contaminants on their Priority Air Contaminants (PAC) list. In addition to these contaminants, a series of detailed screening steps were undertaken to add any additional chemicals of concern which may be emitted from any of the proposed biosolids treatment alternatives but were not on the original PAC list, and determine which of those COCs should be carried forward for a full multimedia pathway assessment. The table below provides a list of the chemicals evaluated in the HHRA by exposure scenario.

Table ES-2 Final COC Lists for each Exposure Assessment Scenario

Chemicals of Concern	Exposure Scenario											
	Base Case Existing Incinerators		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	I	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**
Acetaldehyde	●		●		●		●		●		●	
Acrolein	●		●		●		●		●		●	
Antimony	●	●	●	●								
Arsenic	●	●	●	●	●	●	●	●	●	●	●	●
Barium	●	●	●	●	●	●	●	●	●	●	●	●
Benzene	●		●		●		●		●		●	
Beryllium	●	●	●	●								
Boron	●	●	●	●								
1,3-Butadiene	●		●		●		●		●		●	
Cadmium	●	●	●	●	●	●	●	●	●	●	●	●
Carbon monoxide	●		●		●		●		●		●	
Carbon tetrachloride	●		●									
Chloroform	●		●									
Chromium	●	●	●	●	●	●	●	●	●	●	●	●
Cobalt	●	●	●	●	●	●	●	●	●	●	●	●
Copper	●	●	●	●	●	●	●	●	●	●	●	●
1,4-Dichlorobenzene	●		●						●		●	
1,2-Dichloroethane	●		●									
Dichloromethane	●		●									
Ethylene dibromide	●		●									
Formaldehyde	●		●		●		●		●		●	
Lead	●	●	●	●	●	●	●	●	●	●	●	●
Manganese	●	●	●	●	●	●	●	●	●	●	●	●
Mercury	●	●	●	●	●	●	●	●	●	●	●	●
Molybdenum	●	●	●	●	●	●	●	●	●	●	●	●
Nickel	●	●	●	●	●	●	●	●	●	●	●	●
Nitrogen Oxides	●		●		●		●		●		●	
Ozone	●		●		●		●		●		●	
$PM_{2.5}$	●		●		●		●		●		●	
PM_{10}	●		●		●		●		●		●	
Polychlorinated biphenyls (PCBs)	●	●	●	●								

Chemicals of Concern	Exposure Scenario											
	Base Case Existing Incinerators		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	I	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**
Polychlorinated dibenzo-p-dioxins and furans	●	●	●	●								
Polycyclic aromatic hydrocarbons (PAHs)	●	●	●	●	●	●	●	●	●	●	●	●
Selenium	●	●	●	●	●	●	●	●	●	●	●	●
Strontium	●	●	●	●								
Sulfur Dioxide	●		●		●		●		●		●	
Tetrachloroethylene	●		●									
Toluene	●		●						●		●	
Trichloroethylene	●		●									
Vinyl Chloride	●		●			●		●		●		●
Zinc	●	●	●	●	●	●	●	●	●	●	●	●

Note: I = Inhalation assessment; MM = multimedia assessment

How were Potential Risks Evaluated?

The risk characterization step integrates the exposure and hazard assessments to provide a conservative estimate of human health risk for the receptors assessed in the various exposure scenarios. Risk characterization involves comparing estimates of exposures (from the Exposure Assessment) with toxicity reference values (TRVs) published by various regulatory agencies (identified as part of the Hazard Assessment). This comparison (between predicted exposures and TRVs) can be expressed as a Concentration Ratio (CR) or Hazard Quotient (HQ) for non-carcinogenic chemicals and is calculated by dividing the predicted exposure by the regulatory TRV. In the case of carcinogenic chemicals, potential health risks are expressed as incremental lifetime cancer risks (ILCRs), and represent the incremental risk of an individual developing cancer over his or her lifetime due to exposures from a specific carcinogenic chemical.

Potential exposures to the criteria air contaminants (*i.e.*, carbon monoxide, nitrogen dioxide, ozone, particulate matter, and sulphur dioxide) were also evaluated to determine whether the incremental change in their average concentrations across the entire Study Area compared to the existing operating facility would result in any appreciable change (positive or negative) to various morbidity and mortality rates, based on the methodology employed by Toronto Public Health in their Local Air Quality (LAQ) reports.

What were the Assessment Results and Overall Conclusions?

The results of the assessment indicate that none of the assessed biosolids management alternatives would result in any unacceptable short- or long-term health risks, either from an inhalation, soil or vegetation exposure routes. Most predicted air concentrations were many orders of magnitude below their corresponding health-based reference benchmark (*i.e.*, typically between 3- and 12-orders of magnitude below). When one focuses in on the criteria air contaminants (*i.e.*, carbon monoxide, nitrogen dioxide, ozone, particulate matter, and sulphur dioxide), all of the proposed biosolids management alternatives resulted in a similar very small improvement in air quality across the Study Area compared to the existing base case scenario. These incremental changes in CAC concentrations were also evaluated for potential impacts on various morbidity and premature mortality rates across the Study Area using the methodology employed by the City in their LAQ reports. Results of this assessment indicate that each of the proposed biosolids treatment alternatives would result in a very small improvement in overall morbidity and mortality rates related to local air quality compared to the existing multiple hearth incinerator.

While the health impacts were negligible for all the proposed alternatives, there were differences in the potential levels of risk attributable to the various alternatives. While the proposed fluidized bed incineration alternative had slightly higher short-term risks than the off-site haulage alternatives, the longer term risks were mixed among the alternatives. Alternative 2 (*i.e.*, off-site haulage alternative) had slightly higher long-term risks, and the fluidized bed incinerator alternative had slightly higher risks from exposures to carcinogenic chemicals, exposure to criteria air contaminants and from multi-media exposures. On balance, all of these risks were orders of magnitude below levels that could potentially result in a health risk to the surrounding community.

When comparing the potential contribution of the various proposed biosolids management alternatives to the overall existing air quality within the Study Area, the assessment showed that the cumulative concentrations were dominated almost entirely by existing local background conditions. The various proposed biosolids management alternatives provided negligible contributions to the overall worst-case air quality conditions which was primarily dominated by vehicle emissions from Highway 401 and other major roadways within the Study Area. These findings are similar to the conclusions provided in the LAQ assessments conducted by the City in Wards near major transportation routes.

Even when the assessment focused on the local area closely surrounding the HCTP facility (*i.e.*, “near field”), the various alternatives still represented a very small to negligible contribution to the cumulative exposure, despite the further distance to Highway 401 as the dominant air quality impact within the Study Area.

In conclusion, the results of the HHRA indicate that none of the proposed biosolids management alternatives would result in any unacceptable health risks to the surrounding community. Furthermore, none of the project alternatives provide a significant contribution to short- or long-term cumulative concentrations in the Study Area. While each of the proposed options result in a marginal improvement in air quality compared to the existing multiple hearth incinerators, differences between the various proposed options are largely negligible from a health outcome point-of-view.

HIGHLAND CREEK TREATMENT PLANT (HCTP) CLASS EA
HUMAN HEALTH RISK ASSESSMENT (HHRA) REPORT

1.0 INTRODUCTION

The City of Toronto has four wastewater treatment plants, including the Highland Creek Treatment Plant (HCTP), with a rated capacity of 219,000 cubic metres per day (219 ML/d) that services approximately 500,000 people in the southeast portion of the City. The plant provides conventional activated sludge treatment and discharges treated effluent to Lake Ontario. Residue sludge from the wastewater treatment process is treated biologically by anaerobic digestion and mechanically processed to remove a significant portion of water. The resulting treated material, referred to as “biosolids”, is similar in appearance to a wet soil, and has high organic and nutrient content.

Approximately 40,000 cubic metres of dewatered biosolids are produced each year at the HCTP. Currently, the biosolids are processed in two multiple-hearth incinerators. The resulting inorganic, inert ash is stored on-site in lagoons. The lagoons are cleaned once per year and ash is hauled to the City’s Green Lane landfill site for disposal.

The existing multiple-hearth incinerators have been operating for approximately 38 years, and are approaching the end of their service life. In order to provide continued safe operation consistent within applicable regulatory standards, the City initiated a major maintenance and refurbishment program for the incinerators. This work is underway; however, this will extend the life of the equipment for a maximum of 10 years.

Over the period from 2003 to 2012, the City completed a Biosolids Master Plan (BMP), to provide direction on the future management of biosolids generated at its four wastewater treatment plants (including HCTP) to the year 2025. The BMP was undertaken in accordance with the Municipal Engineers Association (MEA) Class Environmental Assessment (Class EA) (October 2000, amended in 2007 & 2011) process, as defined by the Environmental Assessment Act.

The recommendation brought forward in 2009 from the BMP for the HCTP was to replace the aging multiple-hearth incinerators with new fluidized bed incineration equipment. While biosolids management plans were accepted by City Council for the other three plants, Council did not approve the BMP recommendation for the HCTP. Rather, City staff was directed to provide additional information on the biosolids management strategy proposed for the HCTP. In May 2011, after review and consideration of the additional information, Council directed that a biosolids beneficial use program with landfill as a contingency be implemented at the HCTP, requiring the construction of a new truck loading facility. Residents around the HCTP expressed concerns about this solution, as it would involve an increase in trucking through the primarily residential community.

The Ontario Ministry of the Environment and Climate Change (MOECC) indicated that to obtain acceptance of the preferred biosolids management strategy at the HCTP, the planning process needed to be consistent with the requirements for a Class EA, including analysis of alternative solutions and public consultation. To that end, a new Schedule B Class EA was initiated in April 2014, which involved the examination of all viable biosolids management alternatives to select a preferred solution that would provide long-term reliability for the HCTP.

The primary purpose of this project is to meet the requirements of the MEA Schedule B Class EA process to identify a preferred approach for managing the biosolids generated at the HCTP. To

address concerns with respect to potential human health impacts related to the management of biosolids, each of the potential management approaches were evaluated through the use of a Health Impact Assessment (HIA) framework. A key element of the proposed HIA was a quantitative evaluation of health risks related to potential exposures to chemicals released during the treatment or transportation of biosolids. The quantification of potential chemical health risks was conducted through the use of the human health risk assessment (HHRA) paradigm.

This report provides the detailed methodology and reports the results of the HHRA based on a comparison of predicted health impacts for each of the proposed biosolids treatment alternatives to that of the existing multiple hearth incineration process. Predicted emissions from the three biosolids treatment alternatives were also "added" to the existing background air quality conditions to get a sense of the cumulative risks for each alternative within the Study Area. These two points of reference were used to provide context to aid in the interpretation of the results. Toxicological summaries for each of the chemicals of concern are provided in Appendix A, while a worked example of the calculations conducted in the human health multi-pathway exposure model are provided in Appendix B.

The HHRA was conducted according to widely accepted risk assessment methodologies and guidance documents published and endorsed by regulatory agencies including the Ontario Ministry of the Environment and Climate Change (MOE 2005; 2011c), Health Canada (2009; 2010; 2012) and the United States Environmental Protection Agency (US EPA, 2005). Intrinsik consulted with Toronto Public Health (TPH) during the development of this HHRA and has made every effort to address the concerns and recommendations provided by TPH to ensure consistency with previous Local Air Quality (LAQ) assessments completed by the City and related health-based policies.

The HHRA has also undergone third party peer review by experts at both Toronto Public Health and Public Health Ontario.

2.0 REVIEW OF STUDY METHODOLOGY AND ANALYSIS

2.1 Risk Assessment Framework

In general, a human health risk assessment, or HHRA, is a scientific study that evaluates the potential for the occurrence of adverse health effects from exposures of people (receptors) to chemicals of concern (COCs) present in surrounding environmental media (*e.g.*, air, soil, sediment, surface water, groundwater, food, *etc*.), under existing or predicted exposure conditions. HHRA procedures are based on the fundamental dose-response principle of toxicology. The response of an individual to a chemical exposure typically increases in proportion to the chemical concentration in critical target tissues where adverse effects may occur. The concentrations of chemicals in the target tissues (the dose) are determined by the degree of exposure, which is proportional to the chemical concentrations in the environment where the receptor resides, works or visits.

All chemicals (anthropogenic and natural) have the potential to cause effects in people and the ecosystem. However, it is the chemical concentration, the route of exposure, and the inherent toxicity of the chemical that determines the level of effect and potential for unacceptable risk to the exposed receptor. As illustrated in the diagram to the right, if all three components are present (*i.e.,* where the three circles intersect), the possibility of adverse risk exists.

The prediction of an individual's exposure to specific chemicals in the environment and the potential risks resulting from such exposures can be determined through the completion of a quantitative HHRA. The current HHRA follows the standard HHRA framework (see Figure 2-1) that is composed of the following steps:

i) Problem formulation;

ii) Exposure assessment;

iii) Hazard assessment; and,

iv) Risk characterization.

Typically, where potential adverse impacts are predicted through risk characterization, an additional step providing risk management and recommendations for mitigation measures to address these concerns can be added, if necessary. This risk management step is an integral to the EA process, to ensure the mitigation of any predicted potential health risks in the surrounding community, should they be identified.

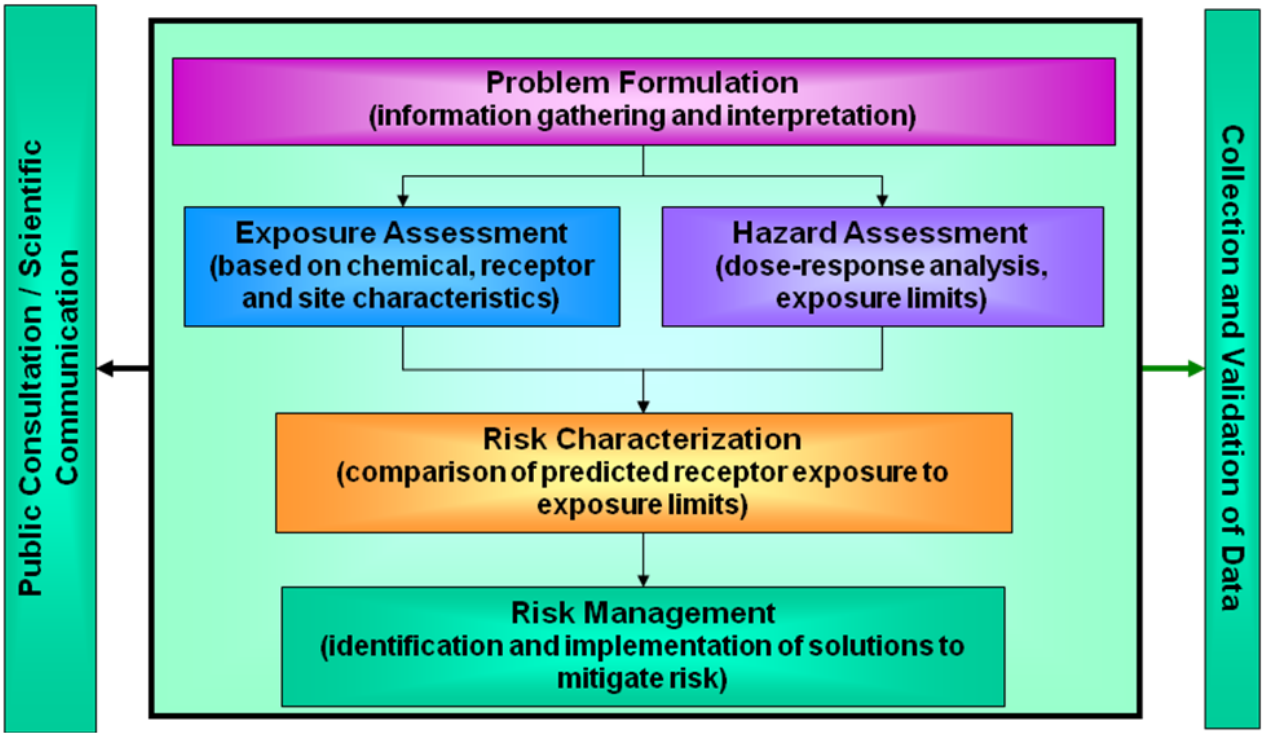


Figure 2-1 Overview of Standard HHRA Framework

2.1.1 Problem Formulation

The first step in the HHRA process is an information gathering and interpretation stage that plans and focuses the study on critical areas of concern for the Project. Problem formulation defines the nature and scope of the work to be conducted, permits practical boundaries to be placed on the overall scope of work and ensures that the assessment is directed at the key areas and issues of concern. This step is critical to the success of the HHRA as sound planning during the problem formulation step reduces the need for significant modifications once the HHRA has begun. The data gathered and evaluated in this step provides information into the physical layout and characteristics of the assessment area, possible exposure pathways, potential human receptors, COCs, and any other specific areas or issues of concern to be addressed.

The key tasks that comprise the problem formulation step of this HHRA include the following:

- **Site characterization**, which consists of a review of available project-specific data to identify factors affecting the availability of chemicals to potential receptors;
- **Chemical characterization**, which involves the identification of the COCs;
- **Receptor characterization** to identify "receptors of concern", which include those individuals with the greatest probability of exposure to chemicals from the proposed facility and those that have the greatest sensitivity to these chemicals; and,
- **Identification of exposure scenarios and pathways** takes into account chemical-specific parameters, such as solubility and volatility, characteristics of the site, such as physical geography, as well as the physiology and behaviour of the receptors.

The outcome of these tasks forms the basis of the approach taken in the HHRA.

2.1.2 Exposure Assessment

The exposure assessment evaluates data related to all chemicals, receptors and exposure pathways and routes identified during the problem formulation phase. As noted previously, the assessment of potential occurrences of adverse effects from chemicals is based on the dose-response concept that is fundamental to the responses of biological systems to chemicals (Filov *et al*., 1979; Amdur *et al.*, 1991). Since it is not usually practical to measure concentrations of chemicals at the actual site where the adverse response occurs within tissues and cells, these concentrations are estimated based on either the dose of the chemical that actually enters a receptor or, more commonly, by the concentrations in various environmental media that act as pathways for exposure. The degree of exposure of individuals to chemicals from the environment therefore depends on the interactions of a number of parameters, including:

- The concentrations of chemicals in various environmental media as determined by the magnitude of point sources as well as background or ambient concentrations;
- The characteristics of the chemicals of potential concern which affect environmental fate and persistence (*e.g.*, physical-chemical properties);
- The impact of site-specific characteristics, such as geology, geography and hydrogeology, on chemical behaviour;
- The physiological and behavioural characteristics of the receptors (*e.g.,* respiration rate, soils/dusts intake, time spent at various activities and in different environmental areas); and,
- The various physical, chemical and biological factors that determine the bioavailability of chemicals from various exposure pathways.

The primary objective of the exposure assessment was to predict, using a series of conservative assumptions, the rate of exposure of individuals living in the surrounding community (residential receptors) to the COCs through various exposure scenarios and pathways identified in the problem formulation step.

Given the nature of the project under assessment, and that the primary source of COCs to the environment is *via* emissions to the atmosphere from the proposed facility or vehicles transporting the biosolids to an off-site treatment or disposal location, the primary route of exposure for people is inhalation. However, for a subset of the COCs (*i.e.*, those considered persistent and/or bioaccumulative), there is the potential for deposition onto soils throughout the surrounding area, resulting in potential impacts to other exposure media (*e.g*., soil, house dust, locally grown produce, *etc*.). For these COCs, a multi-media assessment of potential risks related to oral and dermal exposures has been conducted, in addition to the inhalation assessment.

For the inhalation exposure assessment, specific rates of exposure were not calculated. Rather, human exposures has been conservatively assumed to be equal to ambient air concentrations (measured or modelled) of these substances (in $\mu g/m^3$). The inhalation assessment will evaluate health risks from short- and long-term exposures (*via* direct air inhalation only) for all of the COCs at each of the sensitive receptor locations in the surrounding community.

For the multi-media assessment, the rate of exposure of the selected receptors to the COCs *via* the various exposure scenarios, pathways, and routes identified in the problem formulation step is estimated. The overall objective is to predict, using a series of health-protective assumptions, the rate of exposure (in µg chemical/kg body weight/day) to the COCs *via* the oral and dermal

exposure routes identified in the problem formulation. As air exposures are evaluated as part of the inhalation assessment, the multi-media assessment will focus on exposures arising from the oral and dermal pathways.

In order to evaluate potential exposures, it is necessary to characterize the physiological and behavioral characteristics of each receptor group. Several published sources were considered in the selection of these parameters, including:

- Federal Contaminated Sites Risk Assessment in Canada. PART I: Guidance on Human Health Risk Preliminary Quantitative Risk Assessment (PQRA) (Health Canada, 2012);
- Rationale for the Development of Soil and Groundwater Standards for Use at Contaminated Sites in Ontario. Standards Development Branch, Ontario Ministry of the Environment. April 15th, 2011 (MOE, 2011a);
- Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates Environmental Inc. 1155-2720 Queensview Dr., Ottawa, Ontario (Richardson, 1997);
- The US EPA Exposure Factors Handbook, 2011 Edition (Final). US Environmental Protection Agency, Washington, DC, EPA/600/R-09/052F (US EPA, 2011);
- Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final. EPA/540//R/99/005. July, 2004 (US EPA, 2004); and,
- The US EPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (US EPA, 2005).

These sources have been used in numerous HHRAs and have been critically reviewed and accepted by regulatory agencies across Canada and the United States. The Compendium of Canadian Human Exposure Factors for Risk Assessment (Richardson, 1997), the MOE (2011a) Rationale document, and Health Canada (2012) all rely on data from published and reliable Canadian sources, such as Health Canada, Statistics Canada, and the Canadian Fitness and Lifestyles Research Institute. Where insufficient data were available to appropriately characterize relevant activity patterns and/or behavioral/physiological characteristics, other sources such as the US EPA Exposure Factors Handbook (US EPA, 2011) were used.

2.1.3 Hazard Assessment

The hazard assessment involves identifying and understanding potential health outcomes that can result from exposure to each of the COCs and the conditions under which the outcomes might be observed. The hazard, or toxicity, assessment methodology is based on the fundamental dose response principle. That is, the response of biological systems to chemical exposures increases in proportion to the concentration of a chemical in critical target tissues where adverse health outcomes may occur.

2.1.3.1 Dose-Response Approaches

Two basic and quite different chemical categories are commonly recognized by regulatory agencies, depending on the compound's mode of toxic action, and applied when estimating toxicological criteria for humans (FDA, 1982; US EPA, 1989). These are the *threshold approach* (or the no-observed-adverse-effect levels [NOAELs]/benchmark dose with extrapolation/uncertainty factor approach) typically used to evaluate non-carcinogens, and the *non-threshold approach* (or the mathematical model-unit risk estimation approach), typically used

for carcinogenic compounds. While there are other possible dose response relationships that could be used to describe the toxicological outcome related to exposure to a given chemical (*e.g.*, a J-shaped or an inverted U-shaped dose response such as would occur under hormesis conditions), the standard threshold and non-threshold approaches are the standard dose response relationships evaluated in HHRAs of this type.

Threshold Response Chemicals: For most effects, it is thought that there is a dose-response threshold below which no adverse effects would be expected to occur. Thresholds are generally assumed for non-carcinogenic effects because, for these types of effects, it is generally believed that homeostatic, compensating, and adaptive mechanisms must be overcome before toxicity is manifested. A NOAEL can be identified for threshold chemicals, which is the dose or amount of the chemical that results in no observable response in the most sensitive test species and test endpoint. The application of uncertainty or safety factors to the NOAEL provides an added level of protection, allowing for derivation of a *toxicity reference value* (TRV) or exposure limit that is expected to be safe to sensitive individuals following exposure for a prescribed period of time. Exposure limits derived for threshold-response chemicals are called reference concentrations (RfC), reference doses (RfD), acceptable daily intakes (ADI), tolerable daily intakes (TDI) or permissible daily intakes (PDI) and are generally derived by regulatory agencies such as Health Canada and the US EPA. These values indicate doses of chemicals that individuals can be exposed to on a daily basis over an entire lifetime without appreciable risk of the occurrence of adverse health effects.

Non-threshold Response Chemicals: This means that any exposure greater than zero is assumed to have a non-zero probability of causing some type of response or damage. This relationship is typically used for chemicals that can cause cancer by damaging genetic material. Under a “non-threshold” assumption, any exposure has some potential to cause damage, so it is necessary to define an “acceptable” level of risk associated with these types of exposures.

The acceptable level of risk is an issue of policy rather than a scientific decision (CCME, 2006), and is set by regulatory agencies as opposed to risk assessors. Regulatory agencies have typically employed acceptable incremental lifetime cancer risk (ILCR) levels (*i.e.*, over and above baseline) between 1-in-100,000 and 1-in-1,000,000. An ILCR represents the incremental risk of an individual within a given population developing cancer over his or her lifetime due to exposures from a specific carcinogenic compound.

- Health Canada has specified an ILCR of 1-in-100,000, which is considered “essentially negligible” (Health Canada, 2012).
- The Ontario MOECC considers an ILCR of 1-in-1,000,000 to be acceptable for human health risk assessments in the Province of Ontario. Toronto Public Health is in agreement with the use of a 1-in-1,000,000 ILCR benchmark for the City of Toronto, and encourages actions to reduce exposures when the risk is above one in one million.

ILCRs generally consider risks related to a particular Project (the Project alone, excluding any contribution from other background or pre-existing sources) in that the cancer risks are expressed on an incremental or additional basis as compared to cancer risks related to all sources. The current HHRA is being conducted as part of an EA process in the Province of Ontario, and specifically in the City of Toronto. As such, the ILCRs are reported relative to the Ontario acceptable ILCR of 1-in-1,000,000 (*i.e.*, one-in-one-million or 1×10^{-6}). This acceptable ILCR of 1-in-1,000,000 increases a person’s lifetime cancer risk from 0.400000 (based on the existing 40% lifetime probability of developing cancer in Canada) to 0.400001.

Similar to an ILCR, the lifetime cancer risk (LCR) is an additional measure used to assess cancer. Unlike ILCRs, LCRs include the consideration of cancer risks from all sources including the particular facility under consideration. As such, LCRs are expressed on a total or all sources basis. MOECC has indicated that it may be appropriate to consider cancer risks in this manner, which has been done in the current assessment. The Ontario MOECC does not recommend an acceptable LCR for exposure to carcinogens associated with background or existing baseline conditions and, therefore, the LCR values (for "baseline" and "cumulative sources") are typically provided for reference only.

2.1.3.2 Exposure Limit Terminology

The terminology used to define threshold and non-threshold exposure limits differs according to the source/media and type of exposure and often varies between regulatory jurisdictions. The following terms are used to describe exposure limits in the current assessment.

Reference concentration (RfC): The US EPA defines a reference concentration as *"…an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.*" It can be derived from a NOAEL, LOAEL, or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used." A reference concentration refers to the acceptable level of an airborne chemical for which the primary route of exposure is inhalation, and applies to either short- (*i.e.*, less than 24 hours) or long-term (*i.e.*, more than three months) exposure periods. The reference concentration is expressed as a concentration of the chemical in air (*i.e.,* micrograms per cubic metre, µg/m^3) and applies only to chemicals acting through a threshold mode of toxicological action.

For chemicals such as irritants and some combustion gases, short term or acute non-systemic toxicity is frequently observed at the points of entry into the body (*i.e.*, the respiratory tract, eyes, and skin, for air-borne contaminants). In these cases, because the toxicity is enacted simply by direct contact between the receptor and the contaminated medium, the concentration in the air to which the receptor is exposed is the important measure of exposure, rather than the internal dose associated with multiple exposure pathways. For chemicals with these characteristics, short term RfCs are used to characterize health risk, and are intended to be protective of the general population.

Reference dose (RfD): The US EPA defines a reference dose as *"…an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.*" It can be derived from a NOAEL, LOAEL, or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used." The reference dose is most commonly expressed in terms of the total intake of the chemical per unit of body weight (*i.e.,* micrograms per kilogram of body weight per day, µg/kg bw/day) and applies only to chemicals acting through a threshold mode of toxicological action.

Inhalation unit risk (IUR): The US EPA defines a unit risk value as "*…the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m^3 in air…"* The risks are referred to as "upper bound" because they are not likely to be underestimated and, in fact, may range from as low as zero to the upper bound value. A unit risk value of 3.0 x 10^{-5} per µg/m^3 would mean that under an upper worst-case estimate, three excess cancer cases would be expected to develop per one hundred

thousand (100,000) people, if all 100,000 people were exposed every day for a lifetime to 1 µg of the chemical per m^3 of air.

Cancer slope factor (SF): The US EPA defines a cancer slope factor (SF) as *"…[a]n upper bound, approximating a 95% confidence limit, on the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg-day, is generally reserved for use in the low-dose region of the dose-response relationship, that is, for exposures corresponding to risks less than 1 in 100.*"

2.1.3.3 Exposure Duration

The toxicity of a chemical has been observed to vary between acute (short term) and chronic (long term) exposure. Thus, it is important to differentiate TRVs based on duration of exposure. The two TRV durations used in the current HHRA can be described as follows:

- ***Acute:*** the amount or dose of a chemical that can be tolerated without evidence of adverse health effects on a short term basis. These benchmarks are routinely applied to conditions in which exposures extend from minutes through several hours or several days only (ATSDR, 2006). For the current HHRA, risks have been evaluated based upon a 24-hour exposure period, where a relevant acute TRV for that time period is available.
- ***Chronic:*** the amount of a chemical that is expected to be without effect, even when exposure occurs continuously or regularly over extended periods, possibly lasting for periods of at least a year, and possibly extending over an entire lifetime (ATSDR, 2006).

As it would be inappropriate to establish a generic hierarchy of source agencies by which to select TRVs given the breadth of COCs evaluated in a typical HHRA, when TRVs for a one of the COCs were available from multiple regulatory agencies, all of the TRVs have been reviewed and the professional judgment of experienced toxicologists be used to select the most appropriate TRV. To be consistent with assessments previously conducted by Toronto Public Health (*i.e.*, Local Air Quality reports), preference was given to TRVs established by California EPA, which are often the most conservative (health protective) TRVs available. Where more recent reviews from a credible regulatory agency were available, these were considered when selecting an appropriate benchmark for the current assessment. In each case, selection of the appropriate TRV was conducted in consultation with TPH.

The most critical considerations in selecting TRVs were the source (it must have been derived by a reputable agency), the data used to derive the benchmark, the date the TRV was derived (it must be as up to date as possible), and its relevance in terms of duration and route of exposure. Both MOE (2005, 2011a) and Health Canada (2010) provide lists of acceptable jurisdictions that maybe be used to determine toxicity reference values. The TRVs employed in the HHRA have been obtained from regulatory agencies such as:

- Ontario Ministry of the Environment and Climate Change (MOECC);
- Health Canada;
- Canadian Council of the Ministers of the Environment (CCME);
- World Health Organization (WHO);
- United States Environmental Protection Agency – Integrated Risk Information System (US EPA IRIS);
- Texas Commission on Environmental Quality (TCEQ);
- California Environmental Protection Agency (Cal EPA); and,
- Agency for Toxic Substances and Disease Registry (ATSDR).

Details on potential health outcomes associated with the COC, along with the basis of the TRVs, are outlined in toxicity profiles provided in Appendix A of this report.

2.1.4 Risk Characterization

The final step of a risk assessment is risk characterization. This involves the estimation, description, and evaluation of risk associated with exposure to COCs by comparing the estimated exposure to the appropriate reference benchmark or TRV for a specific chemical or group of compounds. Risk characterization involves the comparison of estimated exposures (identified in the exposure assessment) with reference benchmarks or TRVs (identified during the hazard/toxicity assessment) to identify potential human health risks. This comparison is typically expressed as a Concentration Ratio (CR) or Hazard Quotient (HQ) for non-carcinogenic chemicals and is calculated by dividing the predicted exposure by the reference benchmark/TRV. In the case of direct acting non-threshold carcinogenic chemicals, potential risks are expressed as incremental lifetime cancer risks (ILCRs), and represents the incremental risk of an individual within a given population developing cancer over his or her lifetime due to exposures from a specific carcinogenic chemical of concern.

Separate assessments were completed for short term (acute) and long term (chronic) durations because the health outcomes produced by some COCs depend on the duration of exposure. It is important to distinguish between the health outcomes that might result from short-term exposures *versus* effects that may occur following long-term exposures. In the long-term exposure assessment, further distinction was made between inhalation alone (which included all emitted COCs) and multiple pathway exposures (*i.e.*, inhalation, oral and dermal together) since the pathway of exposure could also influence the potential health outcomes associated with each of the COCs.

In typical transportation risk assessments, the assessment of 1-hour acute exposures is generally evaluated to ensure potential short-term impacts on local air quality around a given corridor are considered. However, given the nature of the emission sources under consideration in the current assessment (*i.e.*, a biosolids incinerator/pelletizer or a minimal number of trucks travelling on nearby routes), it was agreed in consultation with TPH that it is unlikely that 1-hour exposures would be significant.

In recognition of the influence of these exposure variables, risk estimates were segregated into:

- Short-term inhalation (24-hour durations, or 8-hour durations in the case of carbon monoxide);
- Long-term inhalation (annual average durations); and,
- Long-term multi-media pathways (*i.e.,* oral and dermal exposures).

2.1.4.1 Concentration Ratios (CRs) and Hazard Quotients (HQs) for Non-Carcinogens

Concentration Ratios (CR)

CR values were used to evaluate the short- and long-term health risk from exposure to chemicals *via* inhalation. CR values have been calculated by dividing the predicted ground-level air concentration (for 24-hour or annual average exposure durations) by the appropriate toxicity reference value (*i.e.*, RfC), according to the following example equation:

$$CR_{duration} = \frac{[Air]_{duration}}{RfC_{duration}}$$

Where:

$CR_{duration}$	=	the duration-specific *CR* (unitless), calculated for 24-hour short-term and long-term durations, as appropriate
$[Air]_{duration}$	=	the predicted ground-level air concentration (µg/m^3) for the specific time duration
$RfC_{duration}$	=	the RfC (µg/m^3) for the specific time duration

For a COC expected to be present in a single environmental media, such as the case with many gases which occur only or predominately in ambient air, a benchmark representing the entire exposure limit (*i.e.*, a CR value of 1.0) is considered appropriate. Therefore, a CR value of 1.0 (*i.e.*, 100% of the exposure limit) was used as acceptable CR value in the inhalation assessment. Short- and long-term CR values less than the selected benchmark (*i.e.*, CR ≤1.0), indicate that predicted concentrations of COC in air were less than the applicable inhalation exposure limit (*e.g.*, RfC) and that adverse health effects would not be expected to occur.

When predicted risks are greater than the inhalation benchmark level (*i.e.*, CR > 1.0), this indicates the potential for adverse health outcomes may exist. This outcome is referred to as an "exceedance" (*i.e.*, the predicted ground-level air concentration is greater than, or exceeds, the corresponding inhalation exposure limit for that averaging period). Re-evaluation of such CR estimates is important since both the exposure estimates and the toxicological criteria are based on a series of conservative assumptions, particularly when considering the maximum "worst-case" exposure scenarios.

In general, interpretation of the CR values proceeded as follows:

<u>CR ≤1:</u>

Signifies that the estimated exposure is less than or equal to the TRV (*i.e.*, the assumed safe level of exposure). This situation is generally indicative of a negligible likelihood of inhalation health effects. Typically, a significant degree of conservatism (or protection) is incorporated during the derivation of a TRV and, therefore, if predicted exposures (under a worst case or highly conservative set of conditions) are less than a properly derived TRV, it can reasonably be concluded that predicted health risks are not of concern. An exception to this may be in the evaluation of certain criteria air contaminants where no threshold for effects has been identified.

<u>CR >1:</u>

Signifies that the exposure estimate exceeds the TRV. This suggests that the potential for an elevated level of risk may be present for a particular COC, and triggers an additional evaluation. The significance of a CR above 1 must be balanced against the degree of conservatism incorporated in the risk assessment (*e.g.,* an accounting of the number of assumptions used within the risk assessment that tend to overestimate, rather than underestimate, exposure and health risks).

Hazard Quotients (HQ)

Hazard Quotient (HQ) values were used to express risk resulting from long-term exposures to systemically acting, non-carcinogenic chemicals. This approach was used where the exposure to the chemical occurs through multiple pathways, and shows the additional risks related to the oral and dermal exposure pathways. HQ values were calculated by dividing the predicted exposure (*via* multiple pathways) by the appropriate toxicity reference value (RfD), according to the following example equation:

$$HQ = \frac{Exposure}{RfD}$$

Where:

HQ	=	the chronic Hazard Quotient (unitless), calculated for long-term exposures resulting from multiple pathways of exposure
Exposure	=	the long-term exposure estimate resulting from multiple pathways of exposure (µg/kg bodyweight/day
RfD	=	the chronic RfD (µg/kg bodyweight/day)

For long-term multi-media exposures, the CCME (2006) typically allocates 20% of the total exposure to any one environmental media during the derivation of its health-based soil quality criteria. This was based on the assumption that the source of exposure to a particular chemical may occur *via* five potential media: air, food, water, soil, and consumer products. A similar source attribution or allocation model has been adopted by the MOE (2011). This means that, in the absence of a multi-media assessment that takes into account multiple sources or media, the exposure limit should be apportioned for the single medium under consideration.

For the current assessment a benchmark of 0.2 was selected for the evaluation of the long-term multi-media assessment of Project alone emissions since not all potential exposure sources were considered (*i.e.*, the contribution of background sources of these chemicals will not be quantified in the multi-media assessment). HQ values that are less than 0.2 represent a situation in which Project-related exposures (*e.g.*, facility- or transportation-related emissions) account for less than 20% of the oral exposure limit (*e.g.*, oral RfD). As a result, no adverse health risks are expected to be associated with the estimated level of exposure. When predicted health risks resulting from Project alone emissions were greater than the benchmark level (*i.e.*, HQ > 0.2), this may indicate the potential for adverse health outcomes among the most sensitive members of the population and triggers an additional evaluation. Re-evaluation of such HQs is important since both the exposure estimates and the TRV are based on a series of conservative assumptions, particularly when considering the maximum “worst-case” exposure scenarios.

In general, interpretation of the HQ values proceeded as follows:

HQ ≤0.2:

Signifies that the estimated exposure is less than or equal to 20% of the oral exposure limit (*i.e.*, the assumed safe level of exposure). This is generally indicative of a negligible likelihood of adverse human health effects. Typically an added assurance of protection is provided by the significant degree of conservatism (or protection) used during the development of regulatory exposure limits and predicted exposure estimates.

HQ >0.2:

Signifies that an exposure estimate exceeds 20% of the of the oral exposure limit. This generally suggests that the potential for an elevated level of health risk may exist for the specific COC and triggers an additional re-evaluation. The significance of an HQ above 0.2 must be balanced against the high degree of conservatism incorporated in the risk assessment (*e.g.*, an accounting of the number of assumptions used within the risk assessment that tend to overestimate, rather than underestimate, exposure and health risks)

2.1.4.2 Incremental Lifetime Cancer Risks (ILCRs) for Carcinogens

ILCR estimates were used to evaluate the increased cancer risk resulting from a lifetime of exposure to genotoxic, typically non-threshold, carcinogenic chemicals. ILCR estimates provide the incremental lifetime cancer risk resulting from contributions from Project emissions to the surrounding community.

Direct Air Inhalation

For carcinogenic chemicals evaluated as part of the inhalation assessment, ILCR estimates resulting from direct air inhalation were calculated as follows:

$$ILCR = [Air]_{Facility} \times IUR$$

Where:

ILCR	=	the incremental (or additional) lifetime cancer risk (unitless)
$[Air]_{Facility}$	=	the predicted annual average ground-level air concentration (µg/m^3) for the specific chemical arising from facility emissions
IUR	=	the chemical-specific inhalation unit risk value (µg/m^3)$^{-1}$

Multi-Media Exposure

For carcinogenic chemicals evaluated as part of the multi-media assessment, ILCR estimates resulting from a lifetime of exposure through multiple pathways were calculated as follows:

$$ILCR = LADD \times CSF$$

Where:

ILCR	=	the incremental lifetime cancer risk (unitless)
LADD	=	the incremental Lifetime Average Daily Dose via multiple pathways resulting from facility emissions (µg/kg bodyweight/day)
CSF	=	the chemical-specific cancer slope factor (µg/kg bodyweight/day)$^{-1}$

The resulting estimated incremental lifetime cancer risk can be compared to an acceptable risk level of cancer to determine if predicted exposures pose an unacceptable health risk. In the Province of Ontario, the acceptable ILCR is one-in-one million (or 1-in-1,000,000).

In general, interpretation of the ILCR values proceeded as follows:

ILCR ≤ 1.0 x 10^{-6} (1E-06):

Signifies that the estimated exposure results in an incremental lifetime cancer risk less than or equal to 1-in-1,000,000 (*i.e.*, within the accepted level of risk set by MOECC; Health Canada sets the level of essentially negligible risk at 1-in-100,000). This shows that negligible health risks are predicted. Toronto Public Health encourages actions to reduce exposures when the risk is above one-in-one million. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the cancer-based unit risk and slope factor and the exposure estimate.

ILCR > 1.0 x 10^{-6} (1E-06):

Signifies the estimated exposure results in an incremental lifetime cancer risk greater than the MOECC acceptable regulatory-established cancer risk benchmark of 1-in-1,000,000. This suggests that the potential for an elevated level of risk above MOECC's acceptable ILCR (of 1-in-1,000,000) may be present for some COC, the significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment.

2.1.5 Chronic Morbidity and Mortality Risks

In recent air quality studies that evaluate potential risks on an airshed-wide bases, the City of Toronto has also completed an evaluation of health risks associated with multiple respiratory and cardiovascular outcomes leading to acute or chronic premature mortality. One such example is the health assessment for the cumulative air quality modelling study conducted by Toronto Public Health for Wards 5 and 6 including the South Etobicoke and Lakeshore neighbourhoods (TPH, 2014). At the request of TPH and to permit the comparison of potential emissions on an airshed-wide basis to other similar projects, this approach was applied to the current assessment.

As discussed in TPH (2014), the excess risk of premature mortality due to exposure to mixture groups of COCs related to these types of health outcomes can be estimated based on the set of concentration response function (CRF) coefficients endorsed by Health Canada for use in its Air Quality Benefits Assessment Tool (AQBAT). These CRF coefficients represent statistically derived estimates of the percent (%) excess health endpoint associated with a unit increase in the pollutant concentration (Health Canada, 2006).

Estimated percent excess per capita risk for each COC within this group can be calculated as follows:

$$R = (e^{([Air]_{Project} \times CRF)} - 1) * 100$$

Where:

R	=	the estimate of percent excess per capita risk for a one µg/m^3 increase in chemical concentration within the airshed (unitless)
$[Air]_{Project}$	=	the predicted annual average ground-level air concentration (µg/m^3) for the specific chemical arising from emissions of the proposed Project
CRF	=	the coefficient representing excess per capita risk associated with a unit increase of a specific chemical

The mixture group typically evaluated for these risks are the criteria air contaminant group (CACs).

2.1.6 Chemical Mixtures

Concurrent exposures to more than one chemical may result in toxicological interactions which produce health outcomes; this may also result in a combined toxicity which is equal to the sum of toxicities of the individual chemicals (additivity or independence), greater than the sum (synergism or potentiation) or less than the sum (antagonism). In general, toxicological interactions depend on the chemicals present, the levels of exposure to each, their mode of action and their concentrations. Most non-additive interactions can only be demonstrated at relatively high exposures, where clear adverse health outcomes are observed. Such interactions have not been observed or quantified at the relatively low rates of exposure typical of those associated with most environmental situations (NAS, 1983; Krewski and Thomas, 1992; US EPA, 2000; Health Canada, 2012).

Because chemical exposures rarely occur in isolation, the potential health outcomes associated with mixtures of the COCs were assessed in the HHRA. The interaction between chemicals can take many forms, with additive interactions being assumed for the HHRA (Health Canada, 2012). Additive interactions apply to chemicals that are structurally similar, act toxicologically through similar mechanisms or affect the same target tissue in the body (*i.e.,* share common health outcome) (Health Canada, 2012).

The evaluation of risks related to chemical exposures in mixtures is an emerging science. There are currently no accepted reference benchmarks or specific guidance (beyond those chemical groups that have established *toxicity equivalency factors* or TEFs) by which one could evaluate whether exposure to a given mixture could pose a health concern. While the MOECC has not developed specific guidance on chemical mixtures assessment beyond these chemical types, there is a requirement under the Provincial regulations to consider cumulative effects (*i.e*., the additive or synergistic effects of chemical mixtures) when conducting risk assessments. Since discussions on acceptable benchmarks for chemical mixtures are emerging, the ministry has recommended that as a minimum HQ's and ILCR are summed when toxicologically justified (*e.g.*, common modes of toxicological action) and when significant mixture interactions are identified (*i.e*., independent modes of action at any level of disposition) that they be qualitatively discussed (MOECC SDB, personal communication, 2010). It should be noted that this would be considered a conservative approach, as the ILCR represents the incremental risk of an individual within a given population developing cancer over his or her lifetime due to exposures from a *specific* carcinogenic chemical, and has historically not been intended for use in evaluating the risk from a mixture of COCs.

For the current assessment, an initial screening was completed through the summing of all carcinogens and non-carcinogenic COCs in separate groups for each Project Scenario, as consistent with previous air quality evaluations completed by the City of Toronto (*e.g.*, Wards 5 and 6 Air Quality Report, *etc*.).

3.0 PROBLEM FORMULATION

The current assessment followed standard risk assessment methods, and was conducted in compliance with the risk assessment procedures endorsed by regulatory agencies including Health Canada, the CCME, and the US EPA, as well as guidance provided by the MOECC.

3.1 Overview of Problem

The HCTP is one of four wastewater treatment plants operated by the City of Toronto with a rated capacity of 219,000 cubic metres per day (219 ML/d) that services about 500,000 people in the southeast portion of the City. The plant provides conventional activated sludge treatment and discharges treated effluent to Lake Ontario. Residue sludge from the wastewater treatment process is treated biologically by anaerobic digestion and mechanically processed to removal a significant portion of water. The resulting treated material, referred to as *biosolids*, is similar in appearance to a wet soil, and has high organic and nutrient content.

Approximately 40,000 cubic metres of dewatered biosolids are produced each year at the HCTP. Currently, the biosolids are incinerated in two multiple-hearth incinerators. The resulting inorganic, inert ash is stored on-site in lagoons. The lagoons are cleaned once per year and ash is hauled to the City's Green Lane landfill site for disposal.

The HCTP is equipped with two multiple hearth incinerators that began operating in 1979, and have now been in operation for about 38 years. Each incinerator was designed to provide a capacity of 270 tonnes (72.7 dry tonnes) of biosolids per day; however they have never operated at this capacity due to inherent equipment limitations. The actual capacity of each incinerator is estimated at 130 tonnes (35 dry tonnes) per day. Gases leaving the multiple-hearth incinerator are drawn through a wet tray tower scrubber as water flows counter current through the tower. The effluent ash slurry is directed to the ash lagoons. Cleaned incinerator gases pass through an induced draft fan and are dispersed by a 76 m stack. The emissions are tested once per year, and fully comply with all regulatory requirements (CIMA, 2014a).

The Highland Creek Treatment Plant is equipped with two ash lagoons, each with a capacity of approximately 4,200 cubic metres, to store ash slurry. A water cover is maintained on the ash, and excess water is discharged with the plant effluent. Both lagoons are emptied once per year. This involves removing most of the water, and dredging the wet ash solids from the lagoon, into trucks. Ash removed from the lagoons over approximately one week per year, is currently hauled to the City of Toronto's Green Lane Landfill for disposal (CIMA, 2014a).

Currently, an average of 110 wet tonnes of biosolids is generated daily, or a total of 40,150 wet tonnes per year. Biosolids management facilities would be designed for the rated plant capacity, which will be reached well after the year 2032. At the rated plant capacity, an estimated 147.4 wet tonnes of biosolids will be generated daily, or a total of 53,900 wet tonnes per year. The existing HCTP biosolids incinerators are older technology, and a major maintenance program has been implemented to extend their service life so that they can continue to meet regulatory requirements for a maximum period of 10 years. After that time, a new biosolids management approach will be required with capacity to manage 148 wet tonnes per day (54,000 tonnes per year), which is the quantity of biosolids that would be generated at the rated capacity of the Highland Creek Treatment Plant (CIMA, 2014a).

Figure 3-1 provides an aerial overview of the existing Highland Creek Treatment Plant.

Figure 3-1 Aerial View of the Highland Creek Treatment Plant (CIMA, 2014a)

3.1.1 Selection of Assessed Biosolids Management Alternatives

The primary purpose of this project is to meet the requirements of the MEA Schedule B Class EA process to identify a preferred approach for managing the biosolids generated at the HCTP. Based on the initial review conducted as part of this process, the following short-listed biosolids management alternatives have been selected for evaluation through the EA, including the HHRA:

1. On-site fluidized bed incineration and off-site ash management;
2. Transporting biosolids off-site for further management; and,
3. On-site processing of biosolids into pellets (a fertilizer product) and transporting pellets off-site for further management.

To address concerns with respect to potential human health impacts related to the management of biosolids, each of the potential management approaches have been evaluated through the use of an HIA framework. A key element of the HIA is a quantitative evaluation of health risks related to potential exposures to chemicals released during the treatment or transportation of biosolids. The quantification of potential chemical health risks was conducted through the use of the standard HHRA paradigm. Each of these three biosolids treatment alternatives were compared to predicted health risks related to the existing conditions arising from the operation of the current HCTP multiple hearth incinerators.

Based on these short-listed biosolids management alternatives, Table 3-1 provides a list of the project alternatives that were evaluated in both the Air Quality Assessment and the HHRA report.

Table 3-1 List of Evaluated Project Scenarios based on Short-Listed Biosolids Treatment Alternatives	
Project Scenario	**Description**
Base Case	Existing multiple hearth incineration (*i.e.*, baseline, current conditions)
Alternative 1	New fluidized bed incineration
Alternative 2a	Off-site haulage of biosolids along Haul Route 1
Alternative 2b	Off-site haulage of biosolids along Haul Route 4
Alternative 3a	On-site pelletization plus off-site haulage along Haul Route 1
Alternative 3b	On-site pelletization plus off-site haulage along Haul Route 4

3.2 Site Characterization

The City of Toronto Highland Creek Treatment Plant (HCTP) is located on 51 Beechgrove Drive, at the mouth of Highland Creek, in the southeastern Scarborough community of West Hill in Toronto's Ward 44. The treatment plant serves an area of approximately 15,250 hectares (37,682 acres) with boundaries of Warden Avenue to the west, Steeles Avenue to the north, Rouge River to the east and Lake Ontario to the south (CIMA, 2014a).

The EA and HHRA study area has been defined as the area within the boundaries of Toronto's Wards 43 and 44 (Figure 3-2), and was selected as it would be the local area around the plant that could potentially be affected by activities associated with managing biosolids at the treatment plant, or the transport of biosolids from the treatment plant for management off-site. These two Wards include a population of 115,370 persons based on the 2011 census (CIMA, 2014a). The study area impacts due to transporting biosolids materials (if haulage by truck is selected the preferred transport mode) extends only to Highway 401 because of the large number of users of this Highway, the potential incremental air quality impacts from the small number of additional trucks from the Highland Creek Treatment Plant would be negligible (CIMA, 2014a).

Ward 43 is bounded by Morningside Avenue to the east, Lake Ontario to the south, Hill Crescent, Markham Road and Scarborough Golf Club Road to the west and Highway 401 to the north. Ward 44 is bordered by Morningside Avenue to the west, the Rouge River to the east, Highway 401 and Sheppard Avenue East to the north and Lake Ontario to the south. Ward 43 has a physical area of approximately 16 square kilometres, while Ward 44 has an area of 26 square kilometres, for a combined surface area of 42 square kilometers in the study area (CIMA, 2014a).

Biosolids are nutrient rich and are used around the world as a fertilizer product. Depending on the source of the biosolids there are likely trace amounts of various contaminants present. While it is recognized that the area potentially affected by transport and management of biosolids or processed biosolids products off-site is broad, potentially extending for several hundred kilometers in any direction, depending on the management or disposal destination, the broad area was not considered within the study area for this Class EA study, because the transport, management and marketing or disposal of these materials is fully regulated provincially or federally depending on its end use destination. In the event that biosolids are managed off-site, it would be the responsibility of the contractor retained by the City of Toronto to meet the regulatory requirements for the transport and management of the material. As such, assessment of potential health risks related to the off-site management and disposition of biosolids materials is beyond the scope of the current HHRA.

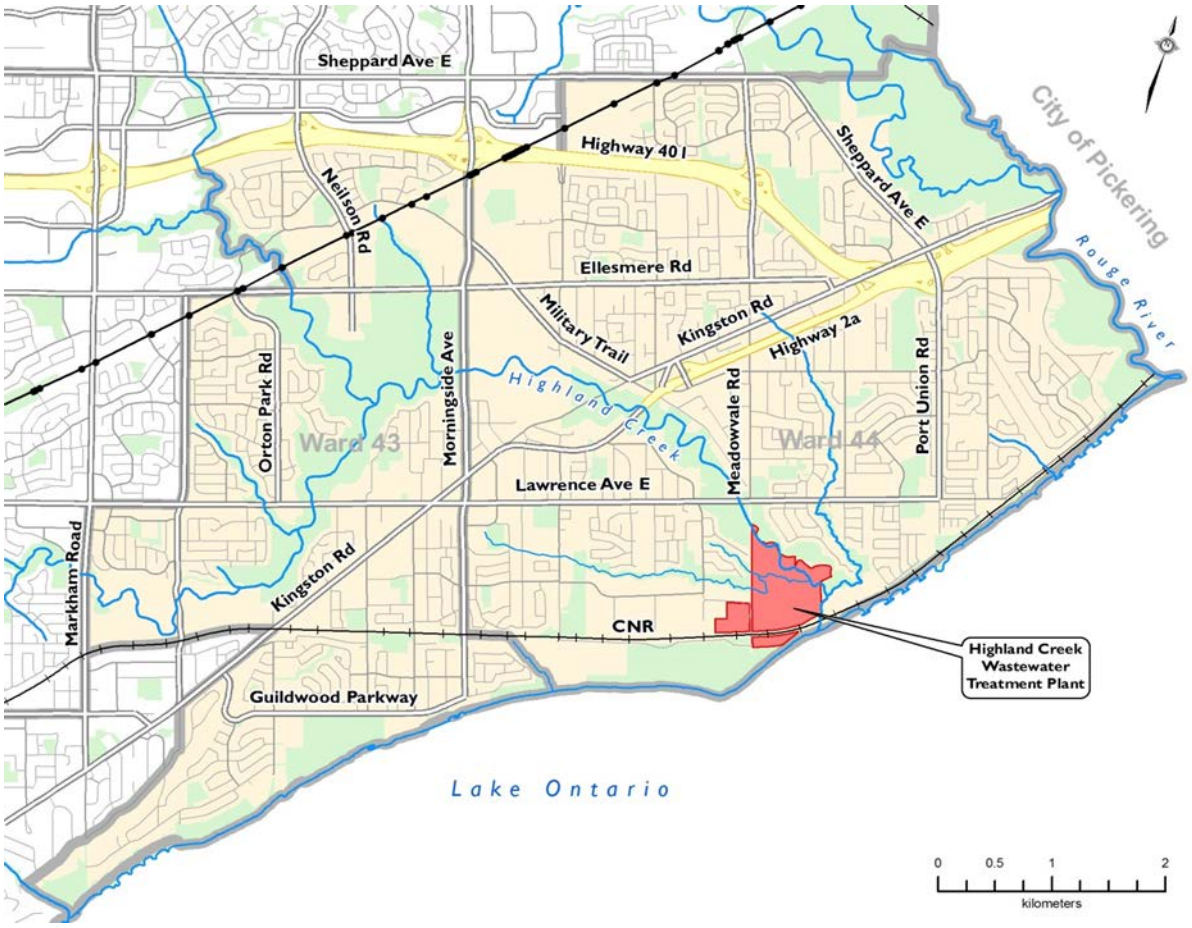


Figure 3-2 Study Area for the HCTP EA for Biosolids Management (CIMA, 2014a)

The predominant land uses within the study area are residential and open space areas, shown in yellow and green, respectively, on Figure 3-3. Open space land designations include zones of natural and recreational uses, golf courses and other areas such as marinas and cemeteries. Most of the open space land designated areas are local parks and ravines located in the vicinity of the tributaries of the Highland Creek watershed and the Lake Ontario waterfront (CIMA, 2014a).

Other land uses within the study area include:

- Approximately 69 ha of employment/industrial designated lands (shown in purple on Figure 3-3), located on the south area of Ward 44, immediately to the west of the Highland Creek Treatment Plant, on the north and south sides of the Canadian National Railroad (CNR);
- A number of small commercial and institutional areas spread across the study area (shown in pink and blue on Figure 3-3); and,
- A wide utility corridor traverses the north boundaries of the study area in a northeast to southwest direction (shown in grey in Figure 3-3). This hydro corridor includes existing electricity supply infrastructure.

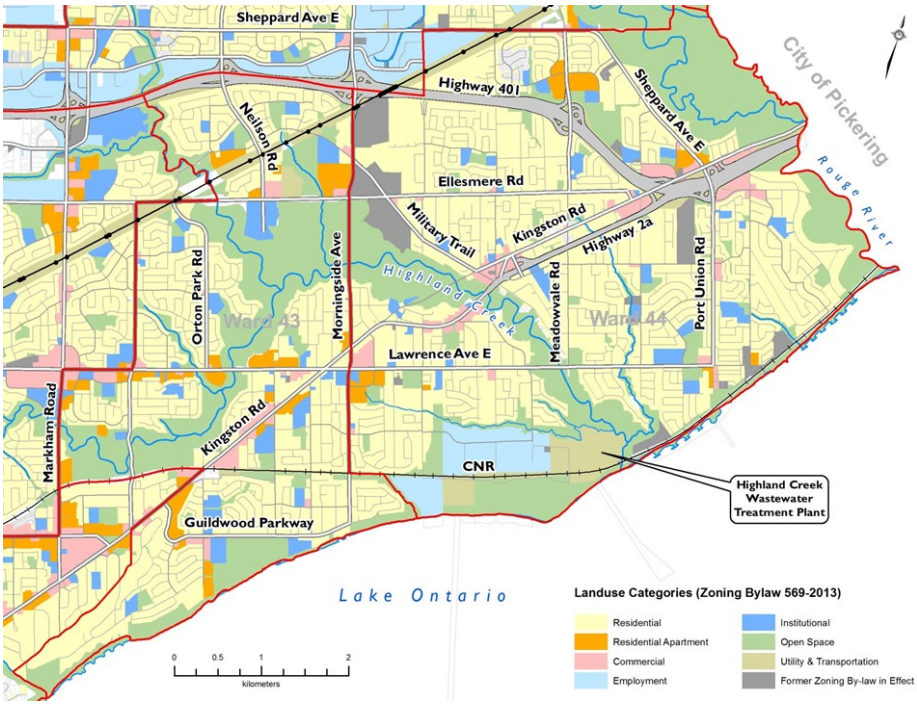


Figure 3-3 General Land Uses in EA Study Area (CIMA, 2014a)

A number of neighbourhoods are located within the limits of both Wards including Guildwood, Morningside, West Hill, Highland Creek and Centennial Scarborough, as well as portions of the Scarborough Village, Woburn and Rouge neighbourhoods. Further information on the demographics of these neighbourhoods can be found in Technical Memo 1 – Study Area Description, Current Biosolids Management, Future Needs and Problem/Opportunity Statement (CIMA, 2014a).

3.2.1 Proposed Locations for Sensitive Receptors

Relying on predicted ground-level air concentrations at the maximum point of impingement (MPOI) from a Project emission source to evaluate human health risks, particularly long-term risks, is considered a very conservative (*i.e.*, protective) approach. By definition, predicted ground-level air concentrations at all other locations are lower than those predicted at the MPOI. As such, the standard risk assessment approach is to also evaluate exposures and potential health risks at several specific sensitive receptor locations beyond the MPOI in the community surrounding the Project-specific emission sources.

As noted previously, the area surrounding the HCTP is composed of mixed industrial, parkland, and residential uses. To assess potential risks related to the projected emissions from the either on-site emission sources or transportation route emission sources for off-site management, the project team selected key sensitive locations representative of the surrounding community.

Typically generic sensitive receptors are modelled based upon guidance provided in Section 30 (relating to upper risk thresholds) of Ontario Regulation 419/05. A sensitive receptor is defined as:

- A senior citizen's residence or long-term care facility;
- A health care facility;
- A child care facility;

- An educational facility; or,
- A dwelling.

There are numerous long term care homes, child care centres, schools, places of worship, libraries, residences and a hospital in the study area. There are a number of open spaces within the study area providing natural and recreational uses, including parks, recreational centres, swimming pools, bikeways, opportunities for trails, *etc*. The most significant parks within the study area include: Morningside Park; Colonel Danforth Park; East Point Park; and, the Scarborough Golf and Country Club (CIMA, 2014a).

So as to avoid identifying specific residential properties within the HHRA, the entire Study Area was broken down into a grid of exposure areas where similar exposure conditions would be expected. This allowed the evaluation of any trends of potential exposures and related health risks associated with emissions arising from the various short-listed biosolids management alternatives. Each grid area is up to 1 km² in size, depending on where it is located (as the wards are not completely square).

Figures 3-4 and 3-5 provide an overview of the individual receptor grid locations within the Study Area evaluating the emission impacts from proposed facility-based and haul route sources, respectively.

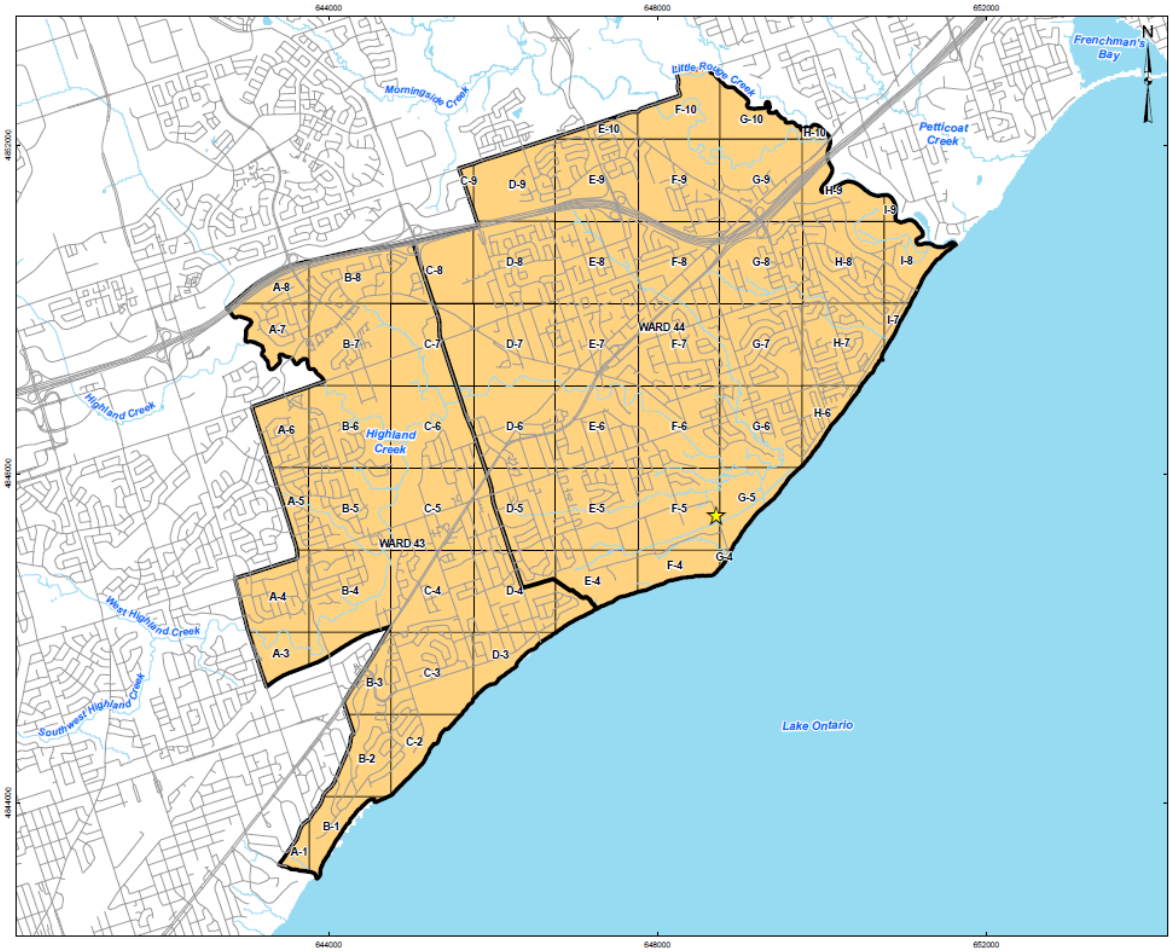


Figure 3-4 Facility Emission Receptor Grid Locations within Study Area

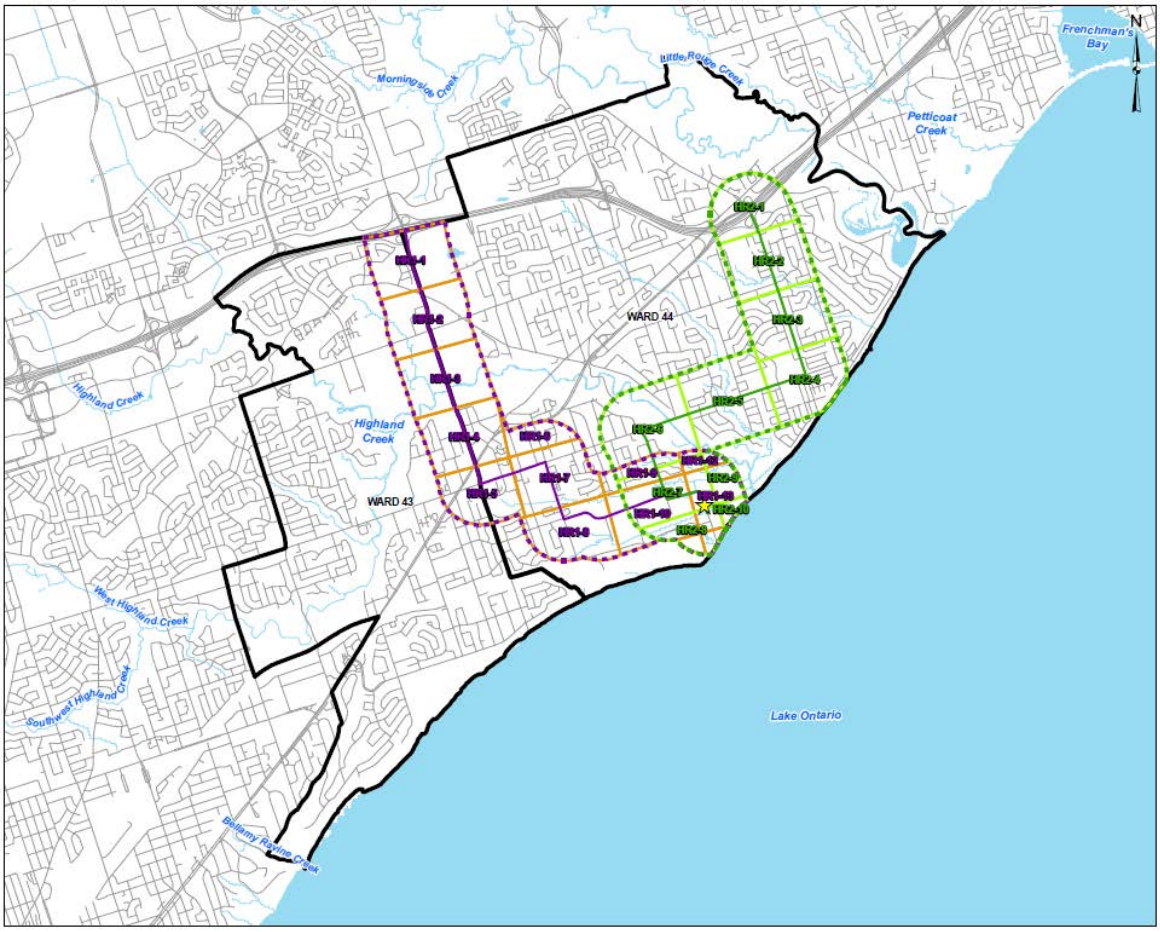


Figure 3-5 Haul Route Receptor Grid Locations within Study Area

Truck transport from the HCTP site introduces risks associated with traffic safety, as well as noise and other negative effects in the community. In light of these potential impacts, an evaluation of alternative feasible routes within the study area was completed to identify the best truck routes for hauling biosolids (or processed biosolids product) from the HCTP, and entering the plant (empty) to pick up the material. A total of six potential routes from the HCTP to the nearest Highway 401 intersections were evaluated based on fifteen criteria related to safety, operations and community impact (CIMA, 2014b).

As a result of the evaluation, the two highest ranked options were from the plant *via* Coronation, Manse, Lawrence and Morningside (*i.e.*, Haul Route 1, or HR1) and from the plant *via* Beechgrove Drive, Lawrence Avenue and Port Union Road (*i.e.*, Haul Route 4, or HR4). The receptor grid locations for HR1 and HR4 are labelled as purple and green, respectively, in Figure 3-5 above. Refer to the TM-3: Transportation Mode and Route Analysis report (CIMA, 2014b) for further details on haul route selection.

For the purpose of the current assessment, and to ensure a conservative approach to evaluating risk, a residential scenario was considered in each of the receptor grid locations outlined in Figures 3-4 and 3-5 based on the maximum ground-level air concentrations predicted for those locations. These worst-case exposures were used in the HHRA to estimate potential health risks related to individuals living within that grid area.

3.3 Identification of Chemical of Concern

A key element for both the air quality assessment and HHRA components of the HCTP EA was the development of a robust and defensible list of chemicals emitted from the various short-listed biosolids management alternatives under consideration. It should be noted the methodology ultimately used to identify COCs was developed in consultation with Toronto Public Health (TPH) through a series of project meetings.

With respect to the current expected short-listed biosolids management alternatives, it was expected that potential COCs for assessment would arise from three specific emission scenarios:

1. Emissions from a thermal destruction process (either the existing or replacement facility) with transportation emissions related to off-site disposal of ash;
2. Emissions from transportation of the treated biosolids to an off-site management or disposal location; or,
3. Emissions from processing on-site (*e.g.*, pelletizing) coupled with emissions from transportation of the enhanced product to an off-site location.

A cumulative air impact assessment was completed for each of the short-listed biosolids management alternatives to define the impacts of the City's Priority Air Contaminants (PACs) on local community (Wards 43 and 44) receptor points under a range of scenarios. These 30 original PACs include the following:

Table 3-2 List of Priority Air Contaminants (PACs) evaluated by the City of Toronto

Acetaldehyde	1,2-Dichloroethane	PM2.5
Acrolein	Dichloromethane	Tetrachloroethylene
Benzene	Ethylene dibromide	Toluene
1,3-Butadiene	Formaldehyde	Trichloroethylene
Cadmium	Lead	Vinyl Chloride
Carbon tetrachloride	Manganese	Carbon Monoxide
Chloroform	Mercury	PM10
~~Chloromethane~~ [a]	Nickel compounds	Sulfur Dioxide
Chromium	Nitrogen Oxides	VOC (anthropogenic/biogenic)
1,4-Dichlorobenzene	PAHs (as B[a]Ps)	Ozone

[a] Toronto Public Health subsequently has removed chloromethane from the City's PAC list, and as such was not automatically included in the HHRA. However, chloromethane was considered in the follow-up screening steps for potential inclusion in the HHRA.

The PAC list created by the City of Toronto was established with a view to identifying a suite of air contaminants that were of priority in Toronto based on estimates of exposure and toxicity. The list was intended to capture key pollutants from a city-wide perspective. Therefore, assessing which pollutants should be evaluated in association with a specific facility requires additional consideration of the pollutants that may be unique to the facility in question.

To ensure all relevant chemicals were evaluated in the HHRA, additional COCs related to thermal destruction or transportation emissions were considered. This was completed by evaluating the long list of chemicals potentially emitted from both emission sources. The HCTP 2013 source testing report provides measurements of 214 discrete compounds at the stack exit. Table 3-3 compares the 214 compounds and the 30 PACs targeted broadly by the City with those compounds measured in annual stack testing conducted at both the Ashbridges Bay and GE Booth Lakeview Treatment Plants (TP) (*i.e.*, comparable incineration facilities to HCTP).

Table 3-3 List of Contaminants Measured during the Stack Emissions Testing at Various Operating Treatment Facilities

Grouping	Contaminant	Contaminant Monitored in Stack Emissions Testing [a]		
		Existing HCTP	Ashbridges Bay TP	GE Booth Lakeview TP [b]
Particulates	TSP	●		●
	PM_{10}	●		
	$PM_{2.5}$	●	●	
Ammonia	Ammonia	●		
Combustion gases	Carbon Dioxide	●		
	Carbon Monoxide	●	●	
	Nitric Oxide	●	●	●
	Nitrogen Dioxide	●		
	Oxygen	●		
	Sulphur Dioxide	●	●	●
	Total Hydrocarbons	●		●
	Hydrogen Chloride			●
	Hydrogen Fluoride			●
Metals	Aluminum	●	●	●
	Antimony	●	●	●
	Arsenic	●	●	●
	Barium	●	●	●
	Beryllium	●	●	●
	Bismuth	●	●	●
	Boron	●	●	●
	Cadmium	●	●	●
	Calcium	●	●	●
	Chromium	●	●	●
	Cobalt	●	●	●
	Copper	●	●	●
	Iron	●	●	●
	Lead	●	●	●
	Lithium	●	●	●
	Magnesium	●	●	●
	Manganese	●	●	●
	Mercury	●	●	●
	Molybdenum	●	●	●
	Nickel	●	●	●
	Phosphorus	●	●	●
	Potassium	●	●	●
	Selenium	●	●	●
	Silicon	●	●	●
	Silver	●	●	●
	Sodium	●	●	●
	Strontium	●	●	●
	Sulphur	●		
	Tellurium	●	●	●
	Tin	●	●	●
	Titanium	●	●	●
	Vanadium	●	●	●
	Zinc	●	●	●

Table 3-3 List of Contaminants Measured during the Stack Emissions Testing at Various Operating Treatment Facilities

Grouping	Contaminant	Contaminant Monitored in Stack Emissions Testing [a]		
		Existing HCTP	Ashbridges Bay TP	GE Booth Lakeview TP [b]
Dioxins	Tetrachlorodi benzo-p-dioxins	●	●	●
	Pentachlorodibenzo-p-dioxins			
	Hexachlorodibenzo-p-dioxins			
	Heptachlorodibenzo-p-dioxins			
	Octachlorodibenzo-p-dioxin			
Furans	Tetrachlorodibenzofurans			
	Pentachlorodibenzofurans			
	Hexachlorodibenzofurans			
	Heptachlorodibenzofurans			
	Octachlorodibenzofuran			
Specific Isomers	2378-tetrachlorodibenzo-p-dioxin	●	●	
	12378-pentachlorodibenzo-p-dioxin	●	●	
	123478-hexachlorodibenzo-p-dioxin	●		
	123678-hexachlorodibenzo-p-dioxin	●	●	
	123789-hexachlorodibenzo-p-dioxin	●	●	
	1234678-heptachlorodibenzo-p-dioxin	●	●	
	Octachlorodibenzo-p-dioxin	●	●	
	2378-tetrachlorodibenzofuran	●	●	
	12378-pentachlorodibenzofuran	●	●	
	23478-pentachlorodibenzofuran	●	●	
	123478-hexachlorodibenzofuran	●	●	
	123678-hexachlorodibenzofuran	●	●	
	234678-hexachlorodibenzofuran	●	●	
	123789-hexachlorodibenzofuran	●	●	
	1234678-heptachlorodibenzofuran	●	●	
	1234789-heptachlorodibenzofuran	●	●	
	Octachlorodibenzofuran	●	●	
	PCB 81	●		
	PCB 77	●		
	PCB 123	●		
	PCB 118	●		
	PCB 114	●		
	PCB 105	●		
	PCB 126	●		
	PCB 167	●		
	PCB 156/157	●		
	PCB 169	●		
	PCB 189	●		
	Total Dioxins & Furans Only	●	●	●
Polychlorinated biphenyls	Dichlorinated biphenyls	●	●	●
	Trichlorinated biphenyls			
	Tetrachlorinated biphenyls			
	Pentachlorinated biphenyls			
	Hexachlorinated biphenyls			
	Heptachlorinated biphenyls			
	Octachlorinated biphenyls			
	Nonachlorinated biphenyls			
	Decachlorinated biphenyl			
Chlorobenzenes	1,3-Dichlorobenzene	●	●	
	1,4-Dichlorobenzene	●	●	
	1,2-Dichlorobenzene	●	●	
	Total Dichlorobenzene	●		
	1,3,5-trichlorobenzene	●	●	
	1,2,4-trichlorobenzene	●	●	
	1,2,3-trichlorobenzene	●	●	
	Total Trichlorobenzene	●		

Table 3-3 List of Contaminants Measured during the Stack Emissions Testing at Various Operating Treatment Facilities

Grouping	Contaminant	Contaminant Monitored in Stack Emissions Testing [a]		
		Existing HCTP	Ashbridges Bay TP	GE Booth Lakeview TP [b]
	1,2,3,5- & 1,2,4,5-tetrachlorobenzenes	●		
	1,2,3,4-tetrachlorobenzene	●	●	
	Total Tetrachlorobenzene	●		
	Pentachlorobenzene	●	●	
	Hexachlorobenzene	●	●	●
	Total Chlorobenzenes	●		
Chlorophenol Isomer and Congener Group	2,6-dichlorophenol	●	●	
	2,4 & 2,5-dichlorophenol	●	●	
	3,5-dichlorophenol	●	●	
	2,3-dichlorophenol	●	●	
	3,4-dichlorophenol	●	●	
	Total Dichlorophenols	●		
	2,4,6-trichlorophenol	●	●	
	2,3,6-trichlorophenol	●	●	
	2,3,5-trichlorophenol	●	●	
	2,4,5-trichlorophenol	●	●	
	2,3,4-trichlorophenol	●	●	
	3,4,5-trichlorophenol	●	●	
	Total Trichlorophenols	●		
	2,3,5,6/2,3,4,6-tetrachlorophenol	●	●	
	2,3,4,5-tetrachlorophenol	●	●	
	Total Tetrachlorophenols	●		
	Pentachlorophenol	●		
	Total Chlorophenols	●		
PAHs	Acenaphthene	●	●	●
	Acenaphthylene			
	Anthracene			
	Benzo(a)anthracene			
	Benzo(a)fluorine			
	Benzo(a)pyrene			
	Benzo(b)anthracene			
	Benzo(b)flouranthene			
	Benzo(b)fluorine			
	Benzo(e)pyrene			
	Benzo(g,h,i)perylene			
	Benzo(j)fluoranthene			
	Benzo(k)fluoranthene			
	Biphenyl			
	2-Chloronaphthalene			
	Chrysene/Triphenylene			
	Coronene			
	Dibenz(a,j)acridine			
	Dibenzo(a,e)pyrene			
	Dibenzo(a,h/a,c)anthracene			
	Dibenzo(a,i)pyrene			
	9,10-Dimethylanthracene			
	7,12-Dimethylbenzo(a)anthracene			
	7H-Dibenzo(c, g)carbazole			
	Fluoranthene			
	Fluorene			
	Indeno(l,2,3-cd)pyrene			
	2-Methylanthracene			
	3-Methylcholanthrene			
	1-Methylnaphthalene			
	2-Methylnaphthalene			
	1-Methylphenanthrene			
	9-Methylphenanthrene			
	Naphthalene			

Table 3-3 List of Contaminants Measured during the Stack Emissions Testing at Various Operating Treatment Facilities

Grouping	Contaminant	Contaminant Monitored in Stack Emissions Testing [a]		
		Existing HCTP	Ashbridges Bay TP	GE Booth Lakeview TP [b]
	Perylene			
	Phenanthrene			
	Picene			
	Pyrene			
	Quinoline			
	m-Terphenyl			
	o-Terphenyl			
	p-Terphenyl			
	Tetralin			
VOCs	Acetone			●
	Acetic Acid		●	
	Benzene	●	●	●
	Bis(2-ethylhexyl)phthalate		●	
	Bromodichlorometha ne	●		●
	Bromoethane		●	
	Bromomethane	●		●
	Bromoform			●
	2-Butanone	●		●
	Butylbenzyl phthalate		●	
	Butyric acid		●	
	Carbon disulfide	●	●	
	Carbon tetrachloride		●	●
	Chlorobenzene	●	●	
	Chlorodibromomethane	●		●
	Chloroethane	●		
	Chloroethene	●		
	Chloroform		●	●
	Chloromethane	●	●	
	1,2-Dibromoethane	●	●	
	Dibromomethane	●		
	1,1-Dichloroethane	●	●	●
	1,2-Dichloroethane	●	●	●
	1,1-Dichloroethene	●	●	
	cis,1,2-Dichloroethene	●	●	
	trans,1,2-Dichloroethene	●		●
	Dichlorodifluoromethane	●	●	●
	Dichloromethane	●	●	
	1,2-Dichloropropane	●	●	●
	cis,1,3-Dichloropropene	●	●	
	trans,1,3-Dichloropropene	●		
	1,2-dichlorotetrafluoroethane	●	●	
	Diethylphthalate		●	
	Dimethyl disulphide		●	
	Di-n-butyl phthalate		●	
	Di-n-octyl phthalate		●	
	Diphenyl ether		●	
	Ethyl benzene	●	●	●
	Ethylene dibromide			●
	Ethyl Mercaptan/dimethyl sulfide		●	
	Formic acid		●	
	Hexachlorobutadiene		●	
	2-Hexa none	●		
	Hydrogen sulphide		●	
	Hydrogen sulphide/carbonyl sulphide		●	
	Indole		●	

Table 3-3 List of Contaminants Measured during the Stack Emissions Testing at Various Operating Treatment Facilities

Grouping	Contaminant	Contaminant Monitored in Stack Emissions Testing [a]		
		Existing HCTP	Ashbridges Bay TP	GE Booth Lakeview TP [b]
	Lodomethane	●		
	Isopropylbenzene	●		●
	Lactic acid		●	
	Methyl mercaptan		●	
	Methyl phenol		●	
	Nitrobenzene		●	
	m-cresol		●	
	4-Methyl-2-Pentanone	●		
	Nitrobenzene		●	
	p-cresol		●	
	Pentachlorophenol		●	
	Phenol		●	
	2-Propanone	●		
	Propionic acid		●	
	Propyl mercaptan/methyl ethyl sulfide		●	
	Styrene	●	●	●
	1,1,1,2-Tetrachloroethane	●		
	1,1,2,2-Tetrachloroethane	●	●	
	Tetrachloroethene	●	●	●
	Tetrachloromethane	●		
	Toluene	●	●	●
	Tribromomethane	●		
	1,1,1-Trichloroethane	●	●	●
	1,1,2-Trichloroethane	●	●	
	Trichloroethene	●	●	●
	Trichlorofluoromethane	●	●	●
	Trichloromethane	●		
	1,2,3-Trichloropropane	●		
	Trichlorotrifluoroethane	●	●	●
	1,2,4-Trimethylbenzene	●	●	
	1,3,5-Trimethylbenzene	●	●	●
	Vinyl Acetate	●		
	Vinyl Chloride	●	●	●
	M&P-Xylene	●		●
	Methylene chloride			●
	O-Xylene	●		●
	Xylene	●	●	

Note: Chemicals highlighted with blue shading indicate those COCs which are included in the City of Toronto's Priority Air Contaminants (PAC) list.

[a] Stack emissions testing reports: Existing HCTP (ORTECH, 2013); Ashbridges Bay TP (ETGA, 2005); and, GE Booth Lakeview TP (AMEC, 2014).

[b] Stack monitoring and emissions data from the GE Booth Lakeview treatment plant was used in the City of Hamilton Biosolids Incinerator HHRA.

It is important to note that while these 214 contaminants are part of the existing facilities' routine stack monitoring program, many of these are emitted at negligible concentrations or are of low potential health concern based on their toxicological nature. To address this, the standard risk assessment approach is to conduct a detailed screening whereby the list of chemicals is reduced to those chemicals that are the most significant contributors to the predicted human health risk.

3.3.1 Chemical Screening

The following provide an overview of the screening approaches used to select the COCs evaluated for inhalation and multi-pathway (*i.e.*, inhalation, oral and dermal) exposures in the HHRA.

3.3.1.1 Inhalation Exposures

On-Site Treatment Alternatives

In order to identify COCs for the inhalation of ambient air, discrete screening exercises conducted for recent assessments for two similar recent incineration facilities were leveraged to augment the existing list of PACs. These two assessments included:

1. Evaluation of Air Emissions from the City of Toronto Ashbridges Bay Treatment Plant (ETGA, 2005); and,
2. Human health risk assessment of the proposed City of Hamilton Biosolids Incinerator (Intrinsik, 2010).

Each of these two assessments undertook a detailed chemical screening approach, which were reviewed and approved by either the MOECC and/or TPH. The emissions from both of these facilities would be considered comparable to that which would be expected from the existing HCTP incinerators, as well as any proposed future replacement thermal destruction technologies (should that alternative be selected), given they are all processing similar waste streams (*i.e.*, biosolids), assuming similar air pollution control (APC) systems are in place.

The detailed chemicals screening approach used in the Ashbridges Bay Treatment Plant (ABTP) assessment was based on two different methods. The first method is a two-part risk-based scoring system developed at the School of Public Health at the University of California at Berkeley and endorsed by the Environmental Defense Fund (EDF, 2003) to identify environmental releases of chemicals that are likely to pose the greatest risk to human health. This approach adjusts the concentration of a chemical that is released using a weighting factor (*i.e.,* a chemical's "toxic equivalency potential" or TEP), so that chemical releases can be compared on a common scale that takes into account differences in toxicity and exposure potential, for both carcinogens and non-carcinogens separately (ETGA, 2005).

The second method ranks chemicals using four different criteria: i) toxicity; ii) emissions; iii) persistence; and, iv) bioaccumulation. These two ranking methods were used in the ABTP assessment to select the final list of chemicals evaluated.

For comparison purposes, the following table provides the final list of 17 COCs evaluated in the ABTP 2005 Air Quality Study.

Table 3-4 Final List of Chemicals of Concern Evaluated in the ABTP 2005 Air Quality Study

Chemical of Concern	On City PAC List?
Arsenic	
Benzene	Yes
Benzo[a]pyrene	Yes
Bis(2-ethylhexyl)phthalate	
Cadmium	Yes

Table 3-4 Final List of Chemicals of Concern Evaluated in the ABTP 2005 Air Quality Study	
Chemical of Concern	**On City PAC List?**
Di-n-octyl phthalate	
Hexachlorobutadiene	
Hydrogen Chloride	
Lead	Yes
Mercury	Yes
Nitrogen Oxides	Yes
$PM_{2.5}$	Yes
PCBs (>= 4 Cl)	
Sulphur Dioxide	Yes
Total Dioxins and Furans (as 2,3,7,8-TCDD-eq)	
Total PAHs	Yes
Vinyl Chloride	Yes

In the case of the HHRA conducted as part of the City of Hamilton Biosolids Incinerator EA, a screening process was carried out using estimated emission rates of the initial list of 148 chemicals derived from emissions data for the GE Booth Lakeview Treatment Plant (AMEC, 2014, provides the latest emissions information for this facility). Detailed screening of chemicals to select those chemicals of concern which pose the greatest concern from both a quantity and toxicological relevance point-of-view is a standard approach in risk assessment to ensure the most relevant COCs are selected for consideration. This evaluation was conducted by evaluating the relative potency through two different approaches: i) based on emission factors at the incinerator stack; and, ii) based on ground-level air concentrations at the maximum point of impingement.

A relative potency was estimated for each chemical based on a comparison of inhalation toxicity reference values (TRVs) to the chemical-specific emission rates or ground-level air concentration, depending on the approach. The chemicals that had the highest relative potency (up to 99.9% cumulative) were retained as COCs for each approach. As such, those chemicals that presented the greatest cumulative toxicological potential to result in human health effects were retained, while those with a lower degree of relevance based on their toxicity and emission rate were not considered further in the detailed assessment. Therefore, if it is demonstrated that those chemicals with the greatest toxicological potential do not pose a risk, those emitted at much lower concentrations and have lesser toxicity would also not pose a risk. However, chemicals identified as having significant public concern were added to the list of COCs (*e.g.* dioxins, lead, tetrachloroethylene, hydrogen chloride). As such, a total of twenty-four (24) of the original 148 emitted chemicals were retained as COCs in the inhalation exposure assessment, based on the key chemicals identified through each screening approach.

As such, rather than repeat these two rather detailed screening processes, those chemicals that screened on either the ABTP or City of Hamilton incinerator assessments but weren't already on the City of Toronto's PAC list, were added to the list of COCs for the current HHRA.

Table 3-5 provides the full list of COCs considered in this HHRA based upon the results of the above analysis step, including the source by which it was added to the overall COC list. While the VOC (anthropogenic/biogenic) group is listed on the City's PAC list, as there is no toxicological benchmark available specifically for this broad group category, and the HHRA is evaluating a significant list of additional individual VOCs beyond those listed in the PAC list, the VOC (anthropogenic/biogenic) group was not carried forward in the HHRA.

Table 3-5 List of COC for the Inhalation Evaluation of the On-Site Treatment Alternatives

Chemical (45 total)	City of Toronto PAC List	Ashbridges Bay Air Quality Study	Hamilton Biosolids Incinerator HHRA
Acetaldehyde	●		●
Acrolein	●		●
Antimony			●
Arsenic		●	●
Barium			●
Benzene	●	●	●
Beryllium			●
Bis(2-ethylhexyl)phthalate [a]		●	●
Boron			●
1,3-Butadiene	●		●
Cadmium	●	●	●
Carbon monoxide	●		●
Carbon tetrachloride	●		
Chloroform	●		●
Chromium	●		●
Cobalt			●
Copper			●
1,4-Dichlorobenzene	●		
1,2-Dichloroethane	●		
Dichloromethane	●		
Di-n-octyl phthalate [a]		●	
Ethylene dibromide	●		
Formaldehyde	●		●
Hexachlorobutadiene [a]		●	
Hydrogen chloride [a]		●	●
Lead	●	●	●
Manganese	●		●
Mercury	●	●	●
Molybdenum			●
Nickel	●		●
Nitrogen oxides	●	●	●
Ozone	●		
PM2.5	●	●	●
PM10	●		●
Polychlorinated biphenyls (PCBs)		●	
Polychlorinated dibenzo-p-dioxins and furans		●	●
Polycyclic aromatic hydrocarbons (PAHs)	●	●	●
Selenium			●
Strontium			●
Sulfur Dioxide	●	●	●
Tetrachloroethylene	●		
Toluene	●		
Trichloroethylene	●		
Vinyl Chloride	●	●	
Zinc			●

[a] Eliminated due to a lack of emissions data for this chemical. See discussion below.

Though bis(2-ethylhexl)phthalate, di-n-octyl phthalate, hexachlorobutadiene, and hydrogen chloride were added to the initial list because they were evaluated in either the Ashbridges Bay or Hamilton Biosolids Incinerator HHRAs due to public concern, no emissions data could be located to evaluate potential risks either for the current HCTP incinerators or the proposed new fluidized bed incinerators. As such, these four chemicals were not carried forward further in the

current assessment. It should be noted that no risks were reported for these four COCs in either of the Ashbridges Bay or Hamilton Biosolids Incinerator HHRAs; therefore, this decision will have no impact on the conclusions of this HHRA.

Based on this three-tier screening process, a total of 41 COCs were selected to be carried forward for assessment in the on-site scenarios.

Transportation Alternatives

As a number of the short-listed biosolids management alternatives involve off-site treatment and/or disposal, one of the primary sources of concern for the current assessment is the potential increase in road traffic (*i.*e., diesel trucks) leaving the HCTP and travelling along nearby access roads and arteries to reach Highway 401. As such, one must understand the typical contaminants emitted by vehicle engines to ensure the COC list also addresses potential exposures for these scenarios. The emission source of highest potential concern would be diesel trucks, particularly given diesel exhaust was recently (June 2012) reclassifying by the World Health Organization (WHO) and International Agency for Research on Cancer (IARC) as a Group 1 carcinogen (*i.e.*, a known human carcinogen).

Based upon the primary components present in the exhaust from the vehicle fleets expected to be used to transport biosolids from the HCTP facility, the following contaminants were also selected as COC for the current assessment. These are the typical COCs evaluated in transportation human health assessments of this type (US EPA, 2002).

Table 3-6 List of COC for the Inhalation Evaluation of the Transportation Alternatives

Criteria Air Contaminants (CACs)	**Volatile Organic Contaminants (VOCs)**
• Carbon monoxide (CO) • Oxides of nitrogen (NO_x) • Ozone (O_3) • Inhalable coarse particulate matter (PM_{10}) • Fine particulate matter ($PM_{2.5}$) • Sulphur dioxide (SO_2)	• 1,3-Butadiene • Acetaldehyde • Acrolein • Benzene • Formaldehyde
Polycyclic Aromatic Hydrocarbons (PAHs)	
• Benzo[a]pyrene (as a surrogate for the carcinogenic PAH group)	

In addition to these core COCs typically evaluated in transportation assessments, for completeness, metals where emission factors were available were also considered for the various Transportation alternatives.

It should be noted that the final list of COCs selected for evaluation for the on-site treatment alternatives include all of the COCs selected for the off-site transportation alternatives. As such, no additional chemicals were added by the Transportation Alternatives to the overall COC list evaluated in the current assessment.

3.3.1.2 Multi-Pathway Exposures

Due to the physical-chemical properties of the individual evaluated chemicals, not all COCs emitted from the proposed facility will persist or accumulate in the environment. To identify the COCs that were considered in the multi-pathway risk assessment, the physical-chemical properties of each of the COCs were compared to accepted national and international criteria for the classification of persistent and bio-accumulative substances (Rodan *et al.,* 1999; Environment Canada, 2006).

The multimedia/multi-pathway screening approach used in the current assessment was adapted based upon the methodology presented in the 2005 US EPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities document (US EPA, 2005), and is the standard approach in these types of assessments. The approach accounts for soil loss over time through both degradation and volatilization.

The characterization of persistence and bio-accumulation is provided in detail within Environment Canada's Existing Substances Program and the Health Canada and Environment Canada's Domestic Substances List Categorization, under the *Canadian Environmental Protection Act* (CEPA).

Persistence refers to the length of time a chemical resides in the environment and is measured by its half-life. This is the time required for the quantity of a chemical to diminish or degrade to half of its original amount within a particular environment or medium. For the purposes of this assessment, a chemical was considered persistent if its half-life in soil was greater than or equal to (≥) six months (182 days). The appropriate rate constants (or half-lives) for each of the potential COCs were taken from sources such as US EPA (2005) and Lymann *et al*. (1990), or obtained using EpiSuite from the US EPA (EpiSuite, 2007).

Bio-accumulation is a general term used to describe the process by which chemicals are accumulated in an organism directly from exposure to water, soil, or through consumption of food containing the substances. A chemical's potential to bio-accumulate is related to its octanol-water partition coefficient (K_{ow}). The K_{ow} refers to the ratio of distribution of a substance in octanol compared to that in water. For the purposes of this assessment, a chemical was considered bio-accumulative if its Log K_{ow} was greater than or equal to five.

Therefore, COCs retained for full multi-pathway assessment had:

- A half-life in soil greater than or equal to six months; and/or,
- An octanol-water partition coefficient (Log K_{ow}) greater than or equal to five.

The rationale behind this exercise was that if a chemical released to the air does not meet either of these criteria, only a limited opportunity exists for human exposure *via* secondary exposure pathways (*i.e.,* those other than inhalation), as the potential for that chemical to persist and/or accumulate in the environment is negligible. However, if a chemical meets one or both of these criteria, sufficient opportunity could be present for long term exposure.

Table 3-7 provides the full list of COCs proposed for multimedia assessment based on the results of the persistence screening step.

Table 3-7 List of COC for the Multi-pathway Evaluation		
Chemical of Concern	**Octanol-Water Partition Coefficient Screen [a]**	**Half-Life in Soil Screen [b]**
Acetaldehyde		
Acrolein		
Antimony		●
Arsenic		●
Barium		●
Benzene		
Beryllium	●	
Boron		●
1,3-Butadiene		
Cadmium		●
Carbon monoxide		
Carbon tetrachloride		
Chloroform		
Chromium		●
Cobalt		●
Copper		●
1,4-Dichlorobenzene		
1,2-Dichloroethane		
Dichloromethane		
Ethylene dibromide		
Formaldehyde		
Lead		●
Manganese		●
Mercury		●
Molybdenum		●
Nickel		●
Nitrogen oxides		
Ozone		
PM2.5		
PM10		
Polychlorinated biphenyls (PCBs)	●	
Polychlorinated dibenzo-p-dioxins and furans	●	●
Polycyclic aromatic hydrocarbons (PAHs)	●	●
Selenium		●
Strontium		●
Sulfur Dioxide		
Tetrachloroethylene		
Toluene		
Trichloroethylene		
Vinyl Chloride		
Zinc		●

[a] Flagged chemicals have an octanol-water partition coefficient (log Kow) greater than or equal to five.
[b] Flagged chemicals have a half-life in soil greater than or equal to six months (182 days).

Therefore, 21 of the COCs were carried forward through the multimedia assessment of health risk for the on-site treatment alternatives.

3.3.1.3 Final List of Selected Chemicals of Concern

Table 3-8 provides the list of the selected COCs emitted from the on-site treatment and off-site transportation components for each Alternative, and indicates whether the assessment was inhalation only or also include a multi-media evaluation.

Table 3-8 Final List of COCs for Base Case and all Alternatives

Chemicals of Concern	On-site Treatment Component [a]		Transportation Component [b]	
	Inhalation	Multi-media	Inhalation	Multi-media
Acetaldehyde	●		●	
Acrolein	●		●	
Antimony	●	●		
Arsenic	●	●	●	●
Barium	●	●	●	●
Benzene	●		●	
Beryllium	●	●		
Boron	●	●		
1,3-Butadiene	●		●	
Cadmium	●	●	●	●
Carbon monoxide	●		●	
Carbon tetrachloride	●			
Chloroform	●			
Chromium	●	●	●	●
Cobalt	●	●	●	●
Copper	●	●	●	●
1,4-Dichlorobenzene	●			
1,2-Dichloroethane	●			
Dichloromethane	●			
Ethylene dibromide	●			
Formaldehyde	●		●	
Lead	●	●	●	●
Manganese	●	●	●	●
Mercury	●	●	●	●
Molybdenum	●	●	●	●
Nickel compounds	●	●	●	●
Nitrogen Oxides	●		●	
Ozone	●		●	
$PM_{2.5}$	●		●	
PM_{10}	●		●	
Polychlorinated biphenyls (PCBs)	●	●		
Polychlorinated dibenzo-p-dioxins and furans	●	●		
Polycyclic aromatic hydrocarbons (PAHs)	●	●	●	●
Selenium	●	●	●	●
Strontium	●	●		
Sulfur Dioxide	●		●	
Tetrachloroethylene	●			
Toluene	●			
Trichloroethylene	●			
Vinyl Chloride	●			
Zinc	●	●	●	●

[a] On-site treatment components include the thermal treatments involved with incineration for the Base Case and Alternative 1, and for the pelletization involved with Alternative 3.

[b] Transportation components include emissions from diesel vehicles used to transport either bottom ash (*i.e.*, Base Case and Alternative 1), treated biosolids (*i.e.*, Alternative 2), or pellets (*i.e.*, Alternative 3).

3.4 Identification and Selection of Human Receptors

A human receptor is a hypothetical person (*e.g.*, infant, toddler, child, adolescent, adult) who resides and/or works in the area being investigated and is, or could potentially be, exposed to the chemicals identified as being of potential concern. General physical and behavioural characteristics specific to the receptor type (*e.g.*, body weight, breathing rate, food consumption rate, *etc*.) were used to determine the amount of chemical exposure received by each receptor as part of the multi-media assessment. The potential risks associated with chemicals of concern will be different depending on the receptor chosen for evaluation.

The HHRA must be sufficiently comprehensive to ensure inclusion of those receptors with the greatest potential for exposure to COCs, and those who have the greatest sensitivity, or potential for developing adverse health outcomes from these exposures. With this in mind, the selection of hypothetical, reasonable "worst-case" receptors, with somewhat exaggerated life style habits, were used to ensure a conservative (*i.e*., protective) assessment.

For the current assessment, only one specific group of sensitive receptors was evaluated – the residential receptor. Due to the residency time at a given receptor location (*i.e.*, conservatively assumed to be present 24-hours per day and 365 days per year), this group is considered to have the highest potential exposure and resultant health risk from chemicals emitted from the Project. Due to this conservatism, this receptor group will also account for those sensitive individuals who may be present at other land uses throughout the Study Area (*e.g.*, hospitals, daycares, schools, retirement homes, *etc.*).

As per Health Canada (2012) guidance, the *residential receptor* was assumed to be represented by five discrete life stages:

1. Infant (birth to 6 months of age);
2. Preschool child/toddler (7 months to 4 years of age);
3. Child (5 to 11 years of age);
4. Adolescent (12 to 19 years of age); and,
5. Adult (≥ 20 years of age, assuming an 80 year lifespan).

The residential receptor was assumed to be born in Toronto with the facility operating, and conservatively assumed to live at that location for their entire lifetime (*i.e.*, 80 years). The individual was assumed to be exposed *via* inhalation of ambient air to emissions from the proposed facility or project-related transportation source (and other nearby significant sources). The resident was also be assumed to be exposed to COCs through contact with contaminated soil or home grown produce impacted by the deposition of the emitted COCs onto surface soils in the surrounding community. Predicted soil concentrations were conservatively assumed to be the maximum concentration that would be present after the facility's lifetime of deposition, taking into account degradation and soil loss over that time (US EPA, 2005).

For the assessment of inhalation risks, as a straight comparison between predicted short term (*i.e*., 24-hour exposure durations) and long term (*i.e.*, annual average exposures) air concentrations and the corresponding regulatory RfC is made, the resulting CR value is receptor-independent (*i.e.*, the same value is calculated for all receptor types).

In the case of the multi-pathway assessment, exposures *via* the inhalation, oral and dermal pathways to the select COCs were evaluated for the most sensitive receptor groups living in the surrounding community – preschool children. In the case of carcinogenic COCs, potential

incremental lifetime cancer risks were evaluated for a lifetime composite receptor, which combined predicted risks each of the life stages described above to produce an overall lifetime composite risk value.

3.5 Identification of Exposure Scenarios and Pathways

3.5.1 Exposure Scenarios

As noted previously, five new potential treatment alternatives were evaluated in comparison to the existing base-case multiple hearth incineration operations. Table 3-9 provides an overview of each of these alternatives and the COCs that were evaluated for both the inhalation and multimedia evaluation in the HHRA.

Incineration in both the Base Case and Alternative 1 scenarios involves the production of residual ash as a by-product of the combustion process. As noted previously, the resulting inorganic, inert ash is stored on-site in lagoons. The lagoons are cleaned once per year and ash is hauled to the City's Green Lane landfill site for disposal during a two week period annually. For the purpose of the current assessment, predictions of ground-level air concentrations throughout the Study Area for both the Base Case and Alternative 1 scenarios also included the contribution of emissions from truck traffic during this two week period. Both Alternatives 2 and 3 include emissions from truck traffic throughout the year *via* either Haulage Routes 1 or 4 depending on the specific Alternative scenario (*i.e.*, "a" *versus* "b", respectively).

As the pelletization process in Alternatives 3a and 3b does not involve the incineration of biosolids, rather it is a heating process with natural gas to remove excess water, only emissions of the COCs on the City's PAC list were evaluated for the inhalation assessment (rather than the full incinerator COC list), and only PAHs for the multimedia assessment.

It should be noted that the HHRA did not quantitatively evaluate an operational upset scenario, where the facility may malfunction or not work as intended. In the case of the fluidized bed incinerator alternative, start-up and shut-down operations would use natural gas to control the fluidized bed temperature, so there would be no increased risk to emissions. Furthermore, these start-up and shut-down conditions would be infrequent, during scheduled maintenance periods. Because there is continuous monitoring, there would be minimal risk due to air quality parameters as a result of incomplete combustion if there were operational temperature issues, because temperature would be maintained with natural gas supplement. Particulate emissions will also be continuously monitored after the air pollution control system, so any problem detected would result in incinerator shut-down for maintenance. Given there would be two completely independent incinerator trains under this Alternative option, each with full capacity, if one is down for maintenance, the other train can operate to continue biosolids treatment.

With respect to all of the remaining alternatives, risk of failure (*i.e.*, spills, process performance, *etc*.) is addressed in the Class EA evaluation but not the HHRA and HIA as a whole. This is because any such event would be very infrequent and due to monitoring, standard operating procedures and controls, short-lived so that they would not present a significant health risk.

Table 3-9 Final List of COCs for each Exposure Assessment Scenario

Chemicals of Concern	Exposure Scenario											
	Base Case Existing Incinerators		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	I	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**	**I**	**MM**
Acetaldehyde	●		●		●		●		●		●	
Acrolein	●		●		●		●		●		●	
Antimony	●	●	●	●								
Arsenic	●	●	●	●	●	●	●	●	●	●	●	●
Barium	●	●	●	●	●	●	●	●	●	●	●	●
Benzene	●		●		●		●		●		●	
Beryllium	●	●	●	●								
Boron	●	●	●	●								
1,3-Butadiene	●		●		●		●		●		●	
Cadmium	●	●	●	●	●	●	●	●	●	●	●	●
Carbon monoxide	●		●		●		●		●		●	
Carbon tetrachloride	●		●									
Chloroform	●		●									
Chromium	●	●	●	●	●	●	●	●	●	●	●	●
Cobalt	●	●	●	●	●	●	●	●	●	●	●	●
Copper	●	●	●	●	●	●	●	●	●	●	●	●
1,4-Dichlorobenzene	●		●						●		●	
1,2-Dichloroethane	●		●									
Dichloromethane	●		●									
Ethylene dibromide	●		●									
Formaldehyde	●		●		●		●		●		●	
Lead	●	●	●	●	●	●	●	●	●	●	●	●
Manganese	●	●	●	●	●	●	●	●	●	●	●	●
Mercury	●	●	●	●	●	●	●	●	●	●	●	●
Molybdenum	●	●	●	●	●	●	●	●	●	●	●	●
Nickel	●	●	●	●	●	●	●	●	●	●	●	●
Nitrogen Oxides	●		●		●		●		●		●	
Ozone	●		●		●		●		●		●	
$PM_{2.5}$	●		●		●		●		●		●	
PM_{10}	●		●		●		●		●		●	
Polychlorinated biphenyls (PCBs)	●	●	●	●								
Polychlorinated dibenzo-p-dioxins and furans	●	●	●	●								
Polycyclic aromatic hydrocarbons (PAHs)	●	●	●	●	●	●	●	●	●	●	●	●
Selenium	●	●	●	●	●	●	●	●	●	●	●	●
Strontium	●	●	●	●								
Sulfur Dioxide	●		●		●		●		●		●	
Tetrachloroethylene	●		●									
Toluene	●		●						●		●	
Trichloroethylene	●		●									
Vinyl Chloride	●		●			●		●		●		●
Zinc	●	●	●	●	●	●	●	●	●	●	●	●

Note: I = Inhalation assessment; MM = multimedia assessment

For each of these scenarios, two specific exposure conditions were evaluated:

- *Project Alone* exposures; and, 2) *Cumulative* exposures.

The *Project Alone* assessment evaluates the potential health impact related to the predicted ground-level air concentrations of each of the COCs contributed by each of the proposed biosolids management alternatives to off-site residential locations in the surrounding community.

The *Cumulative* assessment evaluates the potential health impact related to the predicted ground-level air concentrations of each of the COCs contributed by the proposed biosolids management alternative **plus** the existing background ambient concentrations (minus the existing multiple hearth incinerator) of the COC based on the Study Team's modelling of local air quality within the Study Area. It should be noted, that the local background concentrations are only available for the 30 COCs on the City's Priority Air Contaminants list. For those COCs or averaging periods for which no background air quality data is available, evaluation of the cumulative scenario will not be possible, and only the Project Alone scenario was completed.

The maximum ground-level air concentrations predicted under the cumulative assessment may not necessarily represent the worst-case Project contribution, as the worst-case local background contribution rarely occurs at the same time as the worst-case project scenario contribution given local traffic and meteorological conditions.

See the Cumulative Air Quality Report (Golder, 2015) for further details on the air quality modelling.

3.5.2 Exposure Pathways

The primary exposure pathway evaluated in the HHRA was the inhalation of the COCs by individuals living, working or playing in the surrounding community.

For those COCs evaluated by the multi-pathway assessment (*i.e.*, inhalation, oral and dermal exposures), the following additional exposure pathways were considered concurrently:

- **Inhalation:** Inhalation of air impacted by vapours and particulate emitted from the Project-related sources were evaluated.
- **Incidental Ingestion of Soil and Dust**: Through typical indoor and outdoor activities, individuals may accidentally ingest soil and/or dust particles. Children are typically more susceptible to this exposure pathway, as they spend more time in contact with the ground, and are more likely to put soiled articles, such as toys or hands, into their mouths.
- **Incidental Inhalation of Indoor Dust**: Soils impacted by particles emitted from the Project-related sources were assumed to be carried indoors (*e.g.*, by wind, or human and/pet activities) and present as indoor suspended dust for inhalation by individuals living within the home.
- **Dermal Exposure to Soils and Dusts**: Dermal exposures of human receptors may occur in both indoor and outdoor environments, through direct dermal contact with chemically impacted soil and dust.
- **Ingestion of Locally Grown Produce**: Locally grown produce (such as vegetables and fruits grown in backyard gardens) may itself pose a source of exposure to some COCs. As chemicals are deposited from air-borne emissions, they may come into contact with leaves and fruit of crop plants. Deposition of chemicals onto soil may also result in an accumulation in plants through root uptake.

Figure 3-6 provides an overview of the residential exposure scenario, while Figure 3-7 illustrates the Conceptual Site Model (CSM) used in the assessment, and provides an overview of the sources of COCs and the exposure pathways associated with these sources.

As noted in the CSM, for the sake of conservatism, each of the potential pathways and exposure assumptions typically associated with a residential scenario were evaluated at all sensitive receptor locations. For example, when considering multimedia exposures (*i.e.*, non-inhalation), individuals at each of the assessed receptor locations were assumed to spend 24 hours per day, 7 days per week, for 50 weeks per year at this location. This is obviously an overestimation of potential exposures for the schools or other similar sensitive receptor locations (*e.g.*, retirement homes, parks, *etc*.), as well as individuals exposed while at their workplace.

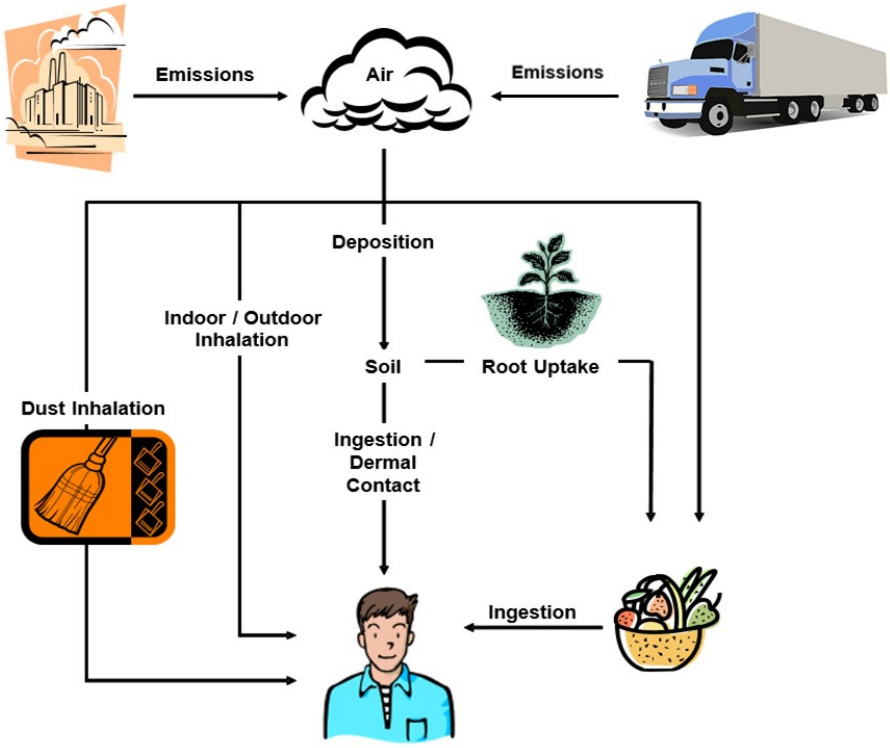


Figure 3-6 Residential Exposure Scenario

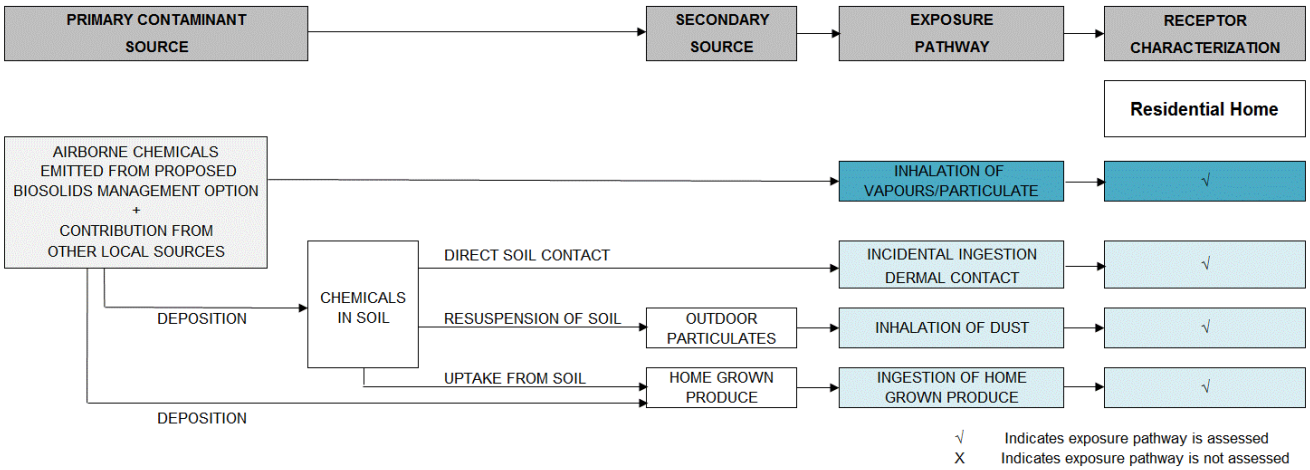


Figure 3-7 Conceptual Site Model (CSM) for Assessment

4.0 EXPOSURE ASSESSMENT

The magnitude of exposure of human receptors to chemicals in the environment typically depends on the interactions of a number of parameters, including:

- The concentrations of chemicals in various environmental media (as determined by the quantities of chemicals entering the environment from various sources, their persistence, fate and behaviour in these media, and the normal ambient, or background concentrations that exist independent of a specific source);
- The physical-chemical characteristics of the chemicals of concern, which affect their environmental fate, transport, behaviour and persistence, and determine the degree or extent by which chemicals can be absorbed into the body;
- The influence of site-specific environmental characteristics, such as geology, soil type, topography, hydrology, hydrogeology, local meteorology and climatology, *etc*., on a chemical's fate, transport and behaviour within environmental media;
- The physiological and behavioural characteristics of the receptors (*e.g.,* respiration rate, soils/dusts intake rate, food ingestion rates, time spent at various activities and in different areas); and,
- The various exposure pathways for the transfer of the chemicals from the different environmental media to humans (*e.g.,* inhalation of indoor and outdoor air, soil particles and dusts; ingestion of food items, water, soils/dusts; skin penetration of various chemicals from dermal contact with soil/dust, water, sediments).

Exposure estimation in the multi-pathway assessment portion of the HHRA was conducted through the use of an integrated environmental risk assessment model developed by the Study Team. The model is spreadsheet based (Microsoft Excel™) but has a number of more advanced add-ons or features. Models of this type have been used on hundreds of peer-reviewed HHRAs in Canada, including those conducted for contaminated sites, landfills, smelters, refineries, incinerators, and a variety of other industrial facilities. The current model version incorporates the techniques and procedures for exposure modelling developed by various regulatory agencies and published scientific literature sources. Refer to Appendix B for a full description (*i.e.*, worked example) of the equations and parameters used in the HHRA.

4.1 Estimation of Ambient Ground-Level Air Concentrations

Ground-level air concentrations for each of the COC at all sensitive receptor grid locations within the Study Area was estimated by the Air Quality Assessment team for use in the HHRA (Golder, 2015). Table 4-1 provides the worst-case Project Alone ground-level air concentrations for each of the COC and relevant averaging periods for each of the Project scenarios. Table 4-2 provides the worst-case Cumulative (*i.e.*, local background conditions plus any Project-specific contributions) ground-level air concentrations for each of the COC and relevant averaging periods for each of the Project scenarios. For comparison purposes, the local background component of the cumulative air concentration is also provided.

It should be noted that predicted concentrations for the Base Case scenario are based on the results of stack monitoring conducted at the existing HCTP incinerator, while the predicted concentrations for Alternative 1 (*i.e.*, a new fluidized bed incinerator) are based on the results of stack monitoring conducted at a comparable fluidized bed incinerator at the GE Booth Lakeview Waste Water Treatment Facility in Mississauga, Ontario. An exception to this was for inorganics evaluated under Alternative 1.

As the incineration process does not create inorganics, there is the potential for discrepancies in inorganic emissions predicted between the Base Case and Alternative 1 scenarios due to differences in inorganic content within the waste streams treated at the respective facilities.

In other words, the Sewer Use Bylaws in either of the City of Toronto or the Region of Peel/City of Mississauga, and their respective enforcement activities, can have a significant impact on the contaminant loads present within biosolids treated in their municipal biosolids management facilities. Moreover, it is expected that the City of Toronto and Peel Region likely have different industries present in their regions. The upstream inputs into the sewer system have a direct impact on the presence and quantity of various contaminants in the biosolids. The type and effectiveness of their respective air pollution control systems, in particular any baghouse units, can also impact on the relative emissions of inorganics from a given facility. Finally, the combustion process used by the existing multiple hearth incinerators requires the input of a significant amount of natural gas to maintain sufficiently high combustion temperatures compared to that required for the more modern fluidized bed incineration alternative. As a result, this can have an impact on the emissions of criteria air contaminants (*e.g.*, higher NOx emissions from the multiple hearth incinerator) when comparing the two incineration methods.

To address the potential inaccuracies in inorganic content due to use of Region of Peel data to represent City of Toronto data, inorganic emissions data from the existing multiple hearth incinerators was used and adjusted by the fractional difference in particulate emissions between the HCTP multiple hearth incinerators and the fluidized bed incinerators at the G.E. Booth facility. This ensures that the increased efficiency of the air pollution control systems used in a modern fluidized bed incinerators are accounted for, while more accurately representing the typical inorganic load present in City of Toronto biosolids.

The predicted ground level air concentrations for both the Base Case and Alternative 1 scenarios include emissions related to the transportation of incineration bottom ash once per year to an approved disposal site. In these cases, emissions for both HR1 and HR4 were assessed. For conservatism, the highest emissions from either of the routes were added to the existing incineration treatment component.

As can be noted in Table 4-2, under worst-case predictions, cumulative air concentrations are dominated by local background conditions with little or no contribution from any of the Project sources. The few Base Case or Alternative cumulative concentrations that were higher than background alone were highlighted in gray. For those COCs associated with vehicle emissions, it is likely that the worst case air concentrations occur when the wind direction blows south to southeast across Highway 401 over the Study Area. This would result in most of the Project-related emissions to actually be blown southward, away from the Study Area, over the nearby Lake.

To evaluate whether maximum concentrations at receptor grid locations closest to the HCTP differed from those observed throughout the Study Area, and concentration information was extracted for a subset of the Study Area dataset (*i.e.*, E-4 to E-6, F-4 to F-6, G-5 to G-6, and H-6). Figure 4-1 shows the location of the “Near Field” area surrounding the HCTP facility.

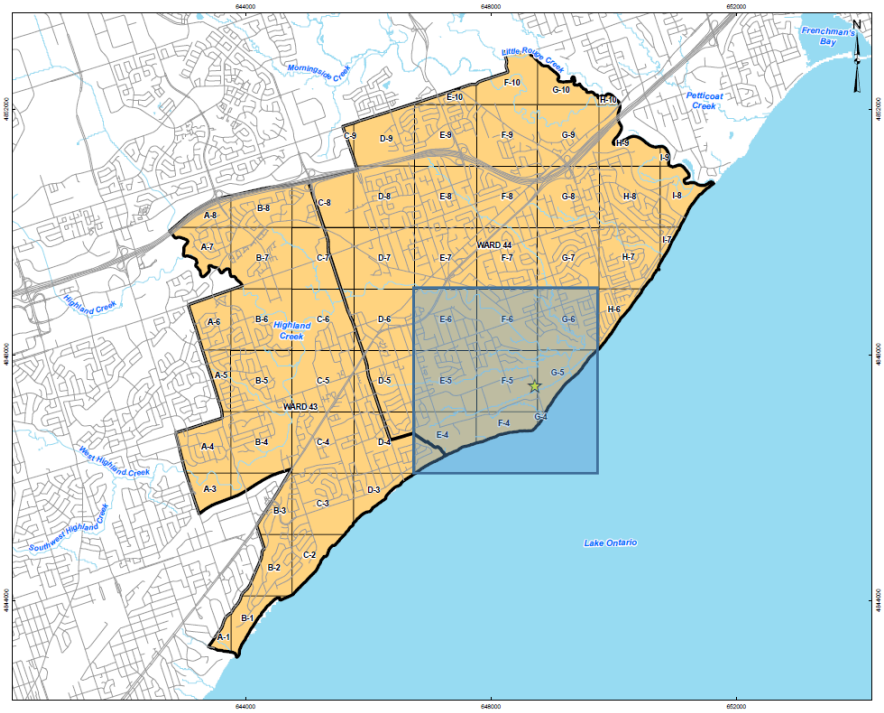


Figure 4-1 Location of the Near Field Area within the Study Area

Table 4-3 provides a comparison of predicted maximum cumulative ground-level air concentrations for the Near Field area *versus* the entire Study Area. Results of this comparison does indicate that maximum concentrations in the area surrounding the HCTP facility are slightly less than observed throughout the remainder of the Study Area, and that even in close proximity, the alternatives still represent a very small to negligible contribution to the cumulative exposure. This likely further demonstrates the far-reaching influence of vehicle emissions from Highway 401 and other major roadways on local air quality within the Study Area, despite improved air quality as one moves towards the lake.

Table 4-1 Summary of Predicted Maximum Project Alone Ground-Level Air Concentrations

Chemical of Concern	Predicted Maximum PROJECT ALONE Ground-Level Air Concentrations (µg/m³)					
	Base Case Existing Multiple Hearth Incineration [a]	**Alternative 1** New Fluidized Bed Incineration [a]	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
8-Hour Concentrations						
Criteria Air Contaminants						
Carbon Monoxide (CO)	18.0	0.35	0.35	0.37	0.39	0.40
24-Hour Concentrations						
Criteria Air Contaminants						
Nitrogen Dioxide (NO_2)	6.6	0.20	0.20	0.20	0.29	0.29
Fine Particulate ($PM_{2.5}$)	0.38	0.037	0.019	0.016	0.022	0.022
Respirable Particulate (PM_{10})	0.47	0.037	0.021	0.017	0.022	0.022
Sulphur Dioxide (SO_2)	1.0	0.37	0.0012	0.0013	0.0017	0.0018
Inorganics						
Antimony	4.6E-05	4.2E-06	NA	NA	NA	NA
Arsenic	3.8E-04	3.4E-05	5.7E-07	4.0E-07	3.9E-07	5.7E-07
Barium	1.2E-05	8.8E-06	1.3E-05	8.8E-06	8.8E-06	1.3E-05
Beryllium	9.9E-07	8.9E-08	NA	NA	NA	NA
Boron	1.1E-04	9.5E-06	NA	NA	NA	NA
Cadmium	1.8E-04	1.7E-05	2.2E-06	2.2E-06	3.2E-06	3.2E-06
Chromium (total)	1.5E-04	1.4E-05	2.8E-06	2.8E-06	4.0E-06	4.0E-06
Chromium (VI) [a]	2.8E-05	2.6E-06	9.5E-07	9.5E-07	1.4E-06	1.4E-06
Cobalt	2.2E-06	2.3E-07	2.4E-07	1.7E-07	1.7E-07	2.4E-07
Copper	1.3E-03	1.2E-04	2.5E-06	1.7E-06	1.7E-06	2.5E-06
Lead	1.2E-03	1.1E-04	9.8E-07	9.8E-07	1.4E-06	1.4E-06
Manganese	2.7E-05	2.5E-06	7.6E-07	7.7E-07	1.1E-06	1.1E-06
Mercury (inorganic) [b]	6.4E-04	2.9E-04	5.2E-07	5.2E-07	7.5E-07	7.5E-07
Molybdenum	2.1E-04	1.9E-05	3.2E-06	2.2E-06	2.2E-06	3.2E-06
Nickel	6.2E-05	6.4E-06	4.2E-06	4.2E-06	6.1E-06	6.1E-06
Selenium	3.5E-04	3.2E-05	6.9E-08	4.8E-08	4.8E-08	6.9E-08
Strontium	1.0E-05	9.1E-07	NA	NA	NA	NA
Zinc	1.4E-02	1.3E-03	NA	5.8E-05	5.8E-05	8.4E-05
Volatile Organic Chemicals (VOCs)						
Acetaldehyde	3.1E-05	3.1E-05	1.2E-03	9.1E-04	4.0E-04	3.2E-04
Acrolein	3.7E-05	3.7E-05	2.1E-04	1.7E-04	7.3E-05	5.8E-05
Benzene	1.3E-03	4.2E-06	2.5E-04	2.0E-04	8.7E-05	6.8E-05
Butadiene, 1,3-	8.7E-07	8.7E-07	8.6E-05	6.8E-05	3.0E-05	2.3E-05
Carbon tetrachloride	NA	4.0E-08	NA	NA	NA	NA
Chloroform	NA	4.0E-08	NA	NA	NA	NA
Dichlorobenzene, 1,4-	3.8E-05	2.4E-06	2.4E-06	2.4E-06	3.5E-06	3.5E-06
Dichloroethane, 1,2-	4.0E-05	4.0E-08	NA	NA	NA	NA

Table 4-1 Summary of Predicted Maximum Project Alone Ground-Level Air Concentrations

Chemical of Concern	Predicted Maximum PROJECT ALONE Ground-Level Air Concentrations (µg/m³)					
	Base Case Existing Multiple Hearth Incineration [a]	**Alternative 1** New Fluidized Bed Incineration [a]	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Dichloromethane	1.1E-03	NA	NA	NA	NA	NA
Ethylene dibromide	7.7E-05	8.0E-08	NA	NA	NA	NA
Formaldehyde	1.5E-04	1.5E-04	1.5E-04	1.5E-04	2.1E-04	2.1E-04
Tetrachloroethylene	NA	NA	NA	NA	NA	NA
Toluene	5.1E-04	6.6E-06	1.8E-04	1.4E-04	6.3E-05	5.0E-05
Trichloroethylene	NA	NA	NA	NA	NA	NA
Vinyl chloride	7.7E-05	8.0E-08	NA	NA	NA	NA
Polychlorinated biphenyls (PCBs)						
PCBs (total)	2.4E-06	7.1E-11	NA	NA	NA	NA
Polychlorinated dibenzo-p-dioxins and Polychlorinated dibenzofurans (PCDD/PCDF)						
PCDD/F as Toxic Equivalents (TEQ)	9.7E-10	7.1E-11	NA	NA	NA	NA
Annual Average Concentrations						
Criteria Air Contaminants						
Nitrogen Dioxide (NO_2)	0.26	0.019	0.019	0.023	0.021	0.022
Fine Particulate ($PM_{2.5}$)	0.015	0.0016	0.0058	0.0041	0.0021	0.0021
Respirable Particulate (PM_{10})	0.019	0.002	0.0063	0.0045	0.0022	0.0021
Sulphur Dioxide (SO_2)	0.040	0.015	0.00024	0.00018	0.00013	0.00015
Inorganics						
Antimony	1.8E-06	1.7E-07	NA	NA	NA	NA
Arsenic	1.5E-05	1.4E-06	3.9E-08	3.0E-08	2.7E-08	3.9E-08
Barium	8.6E-07	8.2E-07	9.2E-07	8.2E-07	8.2E-07	9.2E-07
Beryllium	4.0E-08	3.6E-09	NA	NA	NA	NA
Boron	4.2E-06	3.8E-07	NA	NA	NA	NA
Cadmium	7.3E-06	7.0E-07	2.0E-07	2.0E-07	2.3E-07	2.3E-07
Chromium (total)	5.9E-06	5.8E-07	2.6E-07	2.6E-07	2.9E-07	2.9E-07
Chromium (VI) [c]	1.1E-06	1.1E-07	8.8E-08	8.8E-08	9.9E-08	9.9E-08
Cobalt	9.1E-08	1.6E-08	1.8E-08	1.6E-08	1.6E-08	1.8E-08
Copper	5.3E-05	4.8E-06	1.8E-07	1.6E-07	1.6E-07	1.8E-07
Lead	4.7E-05	4.2E-06	6.6E-08	6.6E-08	9.5E-08	9.5E-08
Manganese	1.1E-06	1.2E-07	7.4E-08	8.6E-08	8.0E-08	8.4E-08
Mercury (inorganic) [b]	2.6E-05	1.2E-05	4.8E-08	4.8E-08	5.4E-08	5.4E-08
Molybdenum	8.5E-06	8.0E-07	2.3E-07	2.0E-07	2.0E-07	2.3E-07
Nickel	2.5E-06	4.6E-07	3.9E-07	4.2E-07	4.4E-07	4.4E-07
Selenium	1.4E-05	1.3E-06	5.0E-09	4.5E-09	4.5E-09	5.0E-09
Strontium	4.1E-07	3.6E-08	NA	NA	NA	NA
Zinc	5.6E-04	5.1E-05	6.1E-06	5.4E-06	5.4E-06	6.1E-06

Table 4-1 Summary of Predicted Maximum Project Alone Ground-Level Air Concentrations

Chemical of Concern	Predicted Maximum PROJECT ALONE Ground-Level Air Concentrations (µg/m^3)					
	Base Case Existing Multiple Hearth Incineration [a]	**Alternative 1** New Fluidized Bed Incineration [a]	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Volatile Organic Chemicals (VOCs)						
Acetaldehyde	2.9E-06	2.9E-06	3.5E-04	2.4E-04	1.2E-04	8.4E-05
Acrolein	3.4E-06	3.4E-06	6.3E-05	4.4E-05	2.2E-05	1.6E-05
Benzene	5.0E-05	3.9E-07	7.5E-05	5.3E-05	2.6E-05	1.8E-05
Butadiene, 1,3-	8.1E-08	8.1E-08	2.6E-05	1.8E-05	8.9E-06	6.2E-06
Carbon tetrachloride	NA	1.6E-09	NA	NA	NA	NA
Chloroform	NA	1.6E-09	NA	NA	NA	NA
Dichlorobenzene, 1,4-	1.5E-06	2.2E-07	2.2E-07	2.2E-07	2.5E-07	2.5E-07
Dichloroethane, 1,2-	1.6E-06	1.6E-09	NA	NA	NA	NA
Dichloromethane	4.6E-05	NA	NA	NA	NA	NA
Ethylene dibromide	3.1E-06	3.2E-09	NA	NA	NA	NA
Formaldehyde	9.9E-06	9.9E-06	9.9E-06	9.9E-06	1.4E-05	1.4E-05
Tetrachloroethylene	NA	NA	NA	NA	NA	NA
Toluene	2.0E-05	4.6E-07	5.4E-05	3.8E-05	1.9E-05	1.3E-05
Trichloroethylene	NA	NA	NA	NA	NA	NA
Vinyl chloride	3.1E-06	3.2E-09	NA	NA	NA	NA
Polychlorinated biphenyls (PCBs)						
PCBs (total)	9.7E-08	2.8E-12	NA	NA	NA	NA
Polychlorinated dibenzo-p-dioxins and Polychlorinated dibenzofurans (PCDD/PCDF)						
PCDD/F as Toxic Equivalents (TEQ)	3.9E-11	2.8E-12	NA	NA	NA	NA
Polycyclic Aromatic Hydrocarbons (PAHs)						
Total PAHs [d]	3.8E-11	7.9E-09	7.6E-09	5.4E-09	2.6E-09	1.9E-09

Note: "NA" indicates that either this COC is not emitted under this particular Scenario, or emission factors could not be located.

[a] For both the Base Case and Alternative 1 scenarios, the additional contribution from the small number of trucks hauling residual bottom ash to a landfill two weeks annually was essentially the same given the number of significant figures in the overall total concentration. As such, only the worst-case total concentrations from the two candidate haul routes was presented.

[b] Estimated air concentrations of mercury assumes the use of a mercury scrubber as part of the air pollution control system planned for the proposed fluidized bed incinerator (*i.e.*, Alternative 1).

[c] For incineration scenarios, chromium VI was assumed to represent 19% of total chromium emitted based on the US EPA (2005) National Emissions Inventory Data and Documentation for a sewage sludge incineration facility, while for the remaining scenarios chromium VI was conservatively assumed to represent 34% of chromium emitted from diesel engines based on data presented in the US EPA MOVES model.

[d] For all evaluated alternatives, the Total PAH concentration was adjusted to be B[a]P-TEQ using a PAH speciation fingerprint for a diesel engine to avoid grossly overestimating potential risks (*i.e.*, assuming all emitted PAHs were toxicologically equivalent to benzo(a)pyrene. This permits a more accurate calculate of toxicological risk. Refer to Section 5.3.1 for further discussion.

Table 4-2 Summary of Predicted Maximum Cumulative Ground-Level Air Concentrations in Study Area

Chemical of Concern	Worst-Case Background Concentration	Predicted Maximum CUMULATIVE Ground-Level Air Concentrations (µg/m³)					
		Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
8-Hour Concentrations							
Criteria Air Contaminants							
Carbon Monoxide (CO)	1901	1901	1901	1901	1901	1901	1901
24-Hour Concentrations							
Criteria Air Contaminants							
Nitrogen Dioxide (NO_2)	36.7	36.7	36.7	36.7	36.7	36.7	36.7
Fine Particulate ($PM_{2.5}$)	52.6	52.6	52.6	52.6	52.6	52.6	52.6
Respirable Particulate (PM_{10})	124	124	124	124	124	124	124
Sulphur Dioxide (SO_2)	128	128	128	128	128	128	128
Inorganics							
Cadmium	0.00096	0.00096	0.00096	0.00096	0.00096	0.00096	0.00096
Chromium (total)	0.0090	0.0090	0.0090	0.0090	0.0090	0.0090	0.0090
Chromium (VI) [a]	0.0022	0.0022	0.0022	0.0022	0.0022	0.0022	0.0022
Lead	0.022	0.022	0.022	0.022	0.022	0.022	0.022
Manganese	0.029	0.029	0.029	0.029	0.029	0.029	0.029
Mercury (inorganic)	0.0012	0.0017	0.0014	0.0012	0.0012	0.0012	0.0012
Nickel	0.012	0.012	0.012	0.012	0.012	0.012	0.012
Volatile Organic Chemicals (VOCs)							
Acetaldehyde	1.3	1.3	1.3	1.3	1.3	1.3	1.3
Acrolein	0.14	0.14	0.14	0.14	0.14	0.14	0.14
Benzene	5.2	5.2	5.2	5.2	5.2	5.2	5.2
Butadiene, 1,3-	0.61	0.61	0.61	0.61	0.61	0.61	0.61
Carbon tetrachloride	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083
Chloroform	0.047	0.047	0.047	0.047	0.047	0.047	0.047
Dichlorobenzene, 1,4-	0.76	0.76	0.76	0.76	0.76	0.76	0.76
Dichloromethane	1.1	1.1	1.1	1.1	1.1	1.1	1.1
Ethylene dibromide	0.00016	0.00024	0.00016	0.00016	0.00016	0.00016	0.00016
Formaldehyde	4.5	4.5	4.5	4.5	4.5	4.5	4.5
Tetrachloroethylene	1.9	1.9	1.9	1.9	1.9	1.9	1.9
Toluene	27.4	27.4	27.4	27.4	27.4	27.4	27.4
Trichloroethylene	0.22	0.22	0.22	0.22	0.22	0.22	0.22
Vinyl chloride	0.0056	0.0057	0.0056	0.0056	0.0056	0.0056	0.0056
Annual Average Concentrations							
Criteria Air Contaminants							
Nitrogen Dioxide (NO_2)	8.1	8.2	8.1	8.1	8.1	8.1	8.1
Fine Particulate ($PM_{2.5}$)	7.9	7.9	7.9	7.9	7.9	7.9	7.9

Table 4-2 Summary of Predicted Maximum Cumulative Ground-Level Air Concentrations in Study Area

Chemical of Concern	Worst-Case Background Concentration	Predicted Maximum CUMULATIVE Ground-Level Air Concentrations (µg/m³)					
		Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Respirable Particulate (PM_{10})	25.9	25.9	25.9	25.9	25.9	25.9	25.9
Sulphur Dioxide (SO_2)	16.3	16.3	16.3	16.3	16.3	16.3	16.3
Inorganics							
Cadmium	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016
Chromium (total)	0.0012	0.0012	0.0012	0.0012	0.0012	0.0012	0.0012
Chromium (VI) [a]	0.00030	0.00030	0.00030	0.00030	0.00030	0.00030	0.00030
Lead	0.0034	0.0034	0.0034	0.0034	0.0034	0.0034	0.0034
Manganese	0.0034	0.0034	0.0034	0.0034	0.0034	0.0034	0.0034
Mercury (inorganic)	0.00015	0.00017	0.00015	0.00015	0.00015	0.00015	0.00015
Nickel	0.0015	0.0015	0.0015	0.0015	0.0015	0.0015	0.0015
Volatile Organic Chemicals (VOCs)							
Acetaldehyde	0.24	0.24	0.24	0.24	0.24	0.24	0.24
Acrolein	0.027	0.027	0.027	0.027	0.027	0.027	0.027
Benzene	1.1	1.1	1.1	1.1	1.1	1.1	1.1
Butadiene, 1,3-	0.13	0.13	0.13	0.13	0.13	0.13	0.13
Carbon tetrachloride	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5
Chloroform	0.0078	0.0078	0.0078	0.0078	0.0078	0.0078	0.0078
Dichlorobenzene, 1,4-	0.16	0.16	0.16	0.16	0.16	0.16	0.16
Dichloromethane	0.21	0.21	0.21	0.21	0.21	0.21	0.21
Ethylene dibromide	1.5E-5	1.8E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5
Formaldehyde	0.67	0.67	0.67	0.67	0.67	0.67	0.67
Tetrachloroethylene	0.39	0.39	0.39	0.39	0.39	0.39	0.39
Toluene	5.4	5.4	5.4	5.4	5.4	5.4	5.4
Trichloroethylene	0.032	0.032	0.032	0.032	0.032	0.032	0.032
Vinyl chloride [b]	0.00084	0.00085	0.00084	0.00084	0.00084	0.00084	0.00084
Polycyclic Aromatic Hydrocarbons (PAHs)							
Total PAHs [c]	0.00018	0.00018	0.00018	0.00018	0.00018	0.00018	0.00018

Note: Concentrations for the Base Case or Alternatives which showed a cumulative concentrations higher than just background concentrations where highlighted in gray.

[a] Air quality studies in Ontario have indicated that approximately 20-25% of the routinely monitored ambient airborne total chromium was in the hexavalent form and the PM_{10} size fractionation study suggested that the majority of the chromium VI was in the inhalable fraction (MOE, 2011b). Therefore, for all cumulative scenarios, chromium VI was conservatively assumed to represent 25% of the total chromium estimated to be present within the airshed.

[b] Based on information provided by the Air Quality Study (Technical Memo 5C), background concentrations of vinyl chloride is very likely underestimated due to a lack of city-wide release information.

[c] For all evaluated alternatives, the Total PAH concentration was adjusted to be B[a]P-TEQ using a PAH speciation fingerprint for a diesel engine to avoid grossly overestimating potential risks (*i.e.*, assuming all emitted PAHs were toxicologically equivalent to benzo(a)pyrene. This permits a more accurate calculate of toxicological risk. Refer to Section 5.3.1 for further discussion.

Table 4-3 Comparison of Predicted Maximum Cumulative Ground-Level Air Concentrations for Near Field *versus* Entire Study Area

Chemical of Concern	Predicted Maximum CUMULATIVE Ground-Level Air Concentrations (µg/m³)											
	Base Case Existing Multiple Hearth Incineration		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field
8-Hour Concentrations												
Criteria Air Contaminants												
Carbon Monoxide (CO)	1901	1711	1901	1711	1901	1711	1901	1711	1901	1711	1901	1711
24-Hour Concentrations												
Criteria Air Contaminants												
Nitrogen Dioxide (NO_2)	36.7	31.8	36.7	31.8	36.7	31.8	36.7	31.8	36.7	31.8	36.7	31.8
Fine Particulate ($PM_{2.5}$)	52.6	47.7	52.6	47.7	52.6	47.7	52.6	47.7	52.6	47.7	52.6	47.7
Respirable Particulate (PM_{10})	124	113	124	113	124	113	124	113	124	113	124	113
Sulphur Dioxide (SO_2)	128	127	128	127	128	127	128	127	128	127	128	127
Inorganics												
Cadmium	0.00096	0.00087	0.00096	0.00087	0.00096	0.00087	0.00096	0.00087	0.00096	0.00087	0.00096	0.00087
Chromium (total)	0.0090	0.0083	0.0090	0.0083	0.0090	0.0083	0.0090	0.0083	0.0090	0.0083	0.0090	0.0083
Chromium (VI)	0.0022	0.0021	0.0022	0.0021	0.0022	0.0021	0.0022	0.0021	0.0022	0.0021	0.0022	0.0021
Lead	0.022	0.017	0.022	0.017	0.022	0.017	0.022	0.017	0.022	0.017	0.022	0.017
Manganese	0.029	0.029	0.029	0.029	0.029	0.029	0.029	0.029	0.029	0.029	0.029	0.029
Mercury (inorganic)	0.0017	0.0017	0.0014	0.0014	0.0012	0.0012	0.0012	0.0012	0.0012	0.0012	0.0012	0.0012
Nickel	0.012	0.012	0.012	0.012	0.012	0.012	0.012	0.012	0.012	0.012	0.012	0.012
Volatile Organic Chemicals (VOCs)												
Acetaldehyde	1.3	1.2	1.3	1.2	1.3	1.2	1.3	1.2	1.3	1.2	1.3	1.2
Acrolein	0.14	0.13	0.14	0.13	0.14	0.13	0.14	0.13	0.14	0.13	0.14	0.13
Benzene	5.2	4.7	5.2	4.7	5.2	4.7	5.2	4.7	5.2	4.7	5.2	4.7
Butadiene, 1,3-	0.61	0.54	0.61	0.54	0.61	0.54	0.61	0.54	0.61	0.54	0.61	0.54
Carbon tetrachloride	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083	0.00083
Chloroform	0.047	0.044	0.047	0.044	0.047	0.044	0.047	0.044	0.047	0.044	0.047	0.044
Dichlorobenzene, 1,4-	0.76	0.65	0.76	0.65	0.76	0.65	0.76	0.65	0.76	0.65	0.76	0.65
Dichloromethane	1.1	1.0	1.1	1.0	1.1	1.0	1.1	1.0	1.1	1.0	1.1	1.0
Ethylene dibromide	0.00024	0.00024	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016	0.00016
Formaldehyde	4.5	3.6	4.5	3.6	4.5	3.6	4.5	3.6	4.5	3.6	4.5	3.6
Tetrachloroethylene	1.9	1.6	1.9	1.6	1.9	1.6	1.9	1.6	1.9	1.6	1.9	1.6
Toluene	27.4	24.0	27.4	24.0	27.4	24.0	27.4	24.0	27.4	24.0	27.4	24.0
Trichloroethylene	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22	0.22
Vinyl chloride	0.0057	0.0057	0.0056	0.0056	0.0056	0.0056	0.0056	0.0056	0.0056	0.0056	0.0056	0.0056

Table 4-3 Comparison of Predicted Maximum Cumulative Ground-Level Air Concentrations for Near Field *versus* Entire Study Area

Chemical of Concern	Predicted Maximum CUMULATIVE Ground-Level Air Concentrations (µg/m³)											
	Base Case Existing Multiple Hearth Incineration		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field	Study Area	Near Field
Annual Average Concentrations												
Criteria Air Contaminants												
Nitrogen Dioxide (NO_2)	8.2	6.3	8.1	6.2	8.1	6.2	8.1	6.2	8.1	6.2	8.1	6.2
Fine Particulate ($PM_{2.5}$)	7.9	6.6	7.9	6.5	7.9	6.5	7.9	6.5	7.9	6.5	7.9	6.5
Respirable Particulate (PM_{10})	25.9	20.3	25.9	20.3	25.9	20.3	25.9	20.3	25.9	20.3	25.9	20.3
Sulphur Dioxide (SO_2)	16.3	16.2	16.3	16.2	16.3	16.2	16.3	16.2	16.3	16.2	16.3	16.2
Inorganics												
Cadmium	0.00016	0.00014	0.00016	0.00014	0.00016	0.00014	0.00016	0.00014	0.00016	0.00014	0.00016	0.00014
Chromium (total)	0.0012	0.0011	0.0012	0.0011	0.0012	0.0011	0.0012	0.0011	0.0012	0.0011	0.0012	0.0011
Chromium (VI)	0.00030	0.00027	0.00030	0.00027	0.00030	0.00027	0.00030	0.00027	0.00030	0.00027	0.00030	0.00027
Lead	0.0034	0.0023	0.0034	0.0023	0.0034	0.0023	0.0034	0.0023	0.0034	0.0023	0.0034	0.0023
Manganese	0.0034	0.0033	0.0034	0.0033	0.0034	0.0033	0.0034	0.0033	0.0034	0.0033	0.0034	0.0033
Mercury (inorganic)	0.00017	0.00017	0.00015	0.00015	0.00015	0.00015	0.00015	0.00015	0.00015	0.00015	0.00015	0.00015
Nickel	0.0015	0.0014	0.0015	0.0014	0.0015	0.0014	0.0015	0.0014	0.0015	0.0014	0.0015	0.0014
Volatile Organic Chemicals (VOCs)												
Acetaldehyde	0.24	0.20	0.24	0.20	0.24	0.20	0.24	0.20	0.24	0.20	0.24	0.20
Acrolein	0.027	0.021	0.027	0.021	0.027	0.021	0.027	0.021	0.027	0.021	0.027	0.021
Benzene	1.1	0.8	1.1	0.8	1.1	0.8	1.1	0.8	1.1	0.8	1.1	0.8
Butadiene, 1,3-	0.13	0.10	0.13	0.10	0.13	0.10	0.13	0.10	0.13	0.10	0.13	0.10
Carbon tetrachloride	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5	9.8E-5
Chloroform	0.0078	0.0070	0.0078	0.0070	0.0078	0.0070	0.0078	0.0070	0.0078	0.0070	0.0078	0.0070
Dichlorobenzene, 1,4-	0.16	0.13	0.16	0.13	0.16	0.13	0.16	0.13	0.16	0.13	0.16	0.13
Dichloromethane	0.21	0.18	0.21	0.18	0.21	0.18	0.21	0.18	0.21	0.18	0.21	0.18
Ethylene dibromide	1.8E-5	1.8E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5	1.5E-5
Formaldehyde	0.67	0.50	0.67	0.50	0.67	0.50	0.67	0.50	0.67	0.50	0.67	0.50
Tetrachloroethylene	0.39	0.29	0.39	0.29	0.39	0.29	0.39	0.29	0.39	0.29	0.39	0.29
Toluene	5.4	4.2	5.4	4.2	5.4	4.2	5.4	4.2	5.4	4.2	5.4	4.2
Trichloroethylene	0.032	0.032	0.032	0.032	0.032	0.032	0.032	0.032	0.032	0.032	0.032	0.032
Vinyl chloride	0.00085	0.00085	0.00084	0.00084	0.00084	0.00084	0.00084	0.00084	0.00084	0.00084	0.00084	0.00084
Polycyclic Aromatic Hydrocarbons (PAHs)												
Total PAHs	0.00018	0.00015	0.00018	0.00015	0.00018	0.00015	0.00018	0.00015	0.00018	0.00015	0.00018	0.00015

4.2 Estimation of Soil and Home Garden Produce Concentrations

Another important element of exposure related to the emissions for the proposed biosolids treatment alternatives is the potential deposition of airborne particulate-bound (and sometimes gaseous) contaminants from the atmosphere onto ground-level surfaces (such as soil, home gardens, *etc*.) in the surrounding community. Deposition (both dry and wet) can be affected by a variety of different factors, the most important of which tend to be the characteristics of the atmosphere (*e.g.*, wind speed, temperature, atmospheric stability, *etc*.), the nature of the surface (*e.g.*, its surface roughness, porosity, *etc*.), and the properties of the depositing species (*e.g.*, reactivity, diameter and shape, solubility, *etc*.). This process can be achieved through "dry" deposition where the particles or gas molecules impact upon a surface, or through "wet" deposition where rain or other precipitation scavenges particles and gas molecules from the air and deposits them on surfaces.

To address this particular exposure route, total deposition into the environment (*e.g*., soil) was estimated in total, wet, and dry deposition per year at each sensitive receptor location by the air quality assessment team. This data was then used to predict exposure concentrations in soil and indoor dust, as well as garden produce consumed by people living within the Study Area. To capture the potential range of exposures, soil and garden produce concentrations were calculated based on the worst-case and average deposition rates across the entire Study Area. Dust represents re-suspended dust from surface soil.

Tables 4-4 through 4-9 and 4-10 through 4-15 provide a summary of predicted Project Alone soil and home garden COC concentrations assuming the predicted annual average air concentration and long-term deposition at the worst-case receptor location and on average across the entire Study Area for each of the six evaluated scenarios. These calculated media concentrations were then used to predict overall multimedia exposures to sensitive individuals across the Study Area.

Table 4-4 Summary of Predicted Project Alone Concentrations for Environmental Media – Base Case (Existing Multiple Hearth Incineration) at Worst-Case Receptor Location

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	1.85E-06	4.22E-01	6.48E-01	4.93E-07	6.57E-04	2.92E-04	2.53E-04	7.34E-04
Arsenic	1.50E-05	3.42E+00	5.25E+00	3.99E-06	3.31E-03	6.30E-04	5.46E-04	3.93E-03
Barium	8.57E-07	9.26E-02	1.42E-01	1.08E-07	1.45E-04	3.20E-05	2.77E-05	1.62E-04
Beryllium	3.96E-08	9.05E-03	1.39E-02	1.06E-08	7.98E-06	3.13E-07	2.71E-07	9.63E-06
Boron	4.23E-06	9.65E-01	1.48E+00	1.13E-06	5.64E-02	5.56E-02	3.85E-02	4.54E-02
Cadmium	7.34E-06	1.67E+00	2.56E+00	1.95E-06	6.18E-03	2.46E-03	2.13E-03	6.48E-03
Chromium	5.95E-06	1.35E+00	2.07E+00	1.57E-06	1.26E-03	1.40E-04	1.21E-04	1.50E-03
Cobalt	9.09E-08	2.01E-02	3.09E-02	2.35E-08	2.13E-05	4.75E-06	2.81E-06	2.34E-05
Copper	5.29E-05	1.21E+01	1.85E+01	1.41E-05	8.98E-02	7.99E-02	6.02E-02	8.16E-02
Lead	4.67E-05	1.07E+01	1.64E+01	1.24E-05	1.21E-02	2.21E-03	1.91E-03	1.40E-02
Manganese	1.07E-06	2.41E-01	3.70E-01	2.82E-07	7.54E-04	5.56E-04	2.41E-04	5.20E-04
Mercury	2.55E-05	5.83E+00	4.21E+00	3.20E-06	2.73E-03	2.27E-03	1.97E-03	3.13E-03
Molybdenum	8.49E-06	1.93E+00	2.96E+00	2.25E-06	6.37E-03	4.78E-03	2.31E-03	4.61E-03
Nickel	2.54E-06	5.63E-01	8.64E-01	6.57E-07	5.84E-04	1.04E-04	8.99E-05	6.86E-04
Selenium	1.41E-05	3.22E+00	4.94E+00	3.75E-06	4.09E-03	1.63E-03	1.41E-03	4.68E-03
Strontium	4.05E-07	9.25E-02	1.42E-01	1.08E-07	1.81E-03	1.73E-03	4.62E-04	6.25E-04
Zinc	5.58E-04	1.27E+02	1.95E+02	1.49E-04	3.89E-01	2.64E+00	2.29E+00	4.12E-01
Organic Parameters								
Polychlorinated biphenyls (PCBs)	9.71E-08	2.22E-02	1.88E-02	1.43E-08	2.02E-05	2.70E-04	2.34E-06	2.42E-05
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	3.87E-11	8.85E-06	7.50E-06	5.70E-12	7.79E-09	1.16E-07	1.00E-09	9.41E-09
Polycyclic aromatic hydrocarbons (PAHs)	1.98E-09	4.43E-04	3.07E-05	2.34E-11	3.70E-07	2.79E-08	2.42E-10	4.51E-07

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.
[b] Annual deposition rates resulting from the Base Case Scenario were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.
[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).
[d] Dust represents re-suspended dust from surface soil.

Table 4-5 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 1 (New Fluidized Bed Incineration) at Worst-Case Receptor Location

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	1.66E-07	3.80E-02	5.83E-02	4.43E-08	5.92E-05	2.63E-05	2.28E-05	6.61E-05
Arsenic	1.35E-06	3.08E-01	4.72E-01	3.59E-07	2.98E-04	5.67E-05	4.91E-05	3.54E-04
Barium	8.20E-07	8.43E-03	1.29E-02	9.84E-09	1.32E-05	2.91E-06	2.52E-06	1.47E-05
Beryllium	3.57E-09	8.14E-04	1.25E-03	9.50E-10	7.18E-07	2.81E-08	2.44E-08	8.67E-07
Boron	3.80E-07	8.69E-02	1.33E-01	1.01E-07	5.07E-03	5.00E-03	3.47E-03	4.09E-03
Cadmium	6.96E-07	1.50E-01	2.31E-01	1.75E-07	5.56E-04	2.21E-04	1.92E-04	5.83E-04
Chromium	5.80E-07	1.21E-01	1.86E-01	1.42E-07	1.13E-04	1.26E-05	1.09E-05	1.35E-04
Cobalt	1.64E-08	1.81E-03	2.78E-03	2.11E-09	1.92E-06	4.28E-07	2.53E-07	2.11E-06
Copper	4.78E-06	1.09E+00	1.67E+00	1.27E-06	8.08E-03	7.19E-03	5.42E-03	7.34E-03
Lead	4.22E-06	9.59E-01	1.47E+00	1.12E-06	1.09E-03	1.99E-04	1.72E-04	1.26E-03
Manganese	1.17E-07	2.17E-02	3.34E-02	2.53E-08	6.79E-05	5.00E-05	2.17E-05	4.68E-05
Mercury	2.55E-05	5.83E+00	4.21E+00	3.20E-06	2.73E-03	2.27E-03	1.97E-03	3.13E-03
Molybdenum	7.99E-07	1.74E-01	2.67E-01	2.03E-07	5.73E-04	4.30E-04	2.08E-04	4.15E-04
Nickel	4.56E-07	5.07E-02	7.79E-02	5.92E-08	5.26E-05	9.34E-06	8.10E-06	6.18E-05
Selenium	1.27E-06	2.90E-01	4.45E-01	3.38E-07	3.68E-04	1.47E-04	1.27E-04	4.21E-04
Strontium	3.65E-08	8.32E-03	1.28E-02	9.71E-09	1.63E-04	1.56E-04	4.15E-05	5.63E-05
Zinc	5.11E-05	1.15E+01	1.76E+01	1.34E-05	3.50E-02	2.37E-01	2.06E-01	3.71E-02
Organic Parameters								
Polychlorinated biphenyls (PCBs)	2.81E-12	6.43E-07	5.45E-07	4.14E-13	5.84E-10	7.83E-09	6.79E-11	7.01E-10
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	2.81E-12	6.43E-07	5.45E-07	4.14E-13	5.66E-10	8.41E-09	7.29E-11	6.83E-10
Polycyclic aromatic hydrocarbons (PAHs)	4.15E-07	9.49E-02	6.59E-03	5.01E-09	7.94E-05	5.98E-06	5.18E-08	9.67E-05

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.
[b] Annual deposition rates resulting from Alternative 1 were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.
[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).
[d] Dust represents re-suspended dust from surface soil.

Table 4-6 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 2a (Off-Site Haulage along HR1) at Worst-Case Receptor Location

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	3.93E-08	1.80E-04	2.77E-04	2.10E-10	1.75E-07	3.32E-08	2.88E-08	2.08E-07
Barium	9.20E-07	3.96E-03	6.09E-03	4.63E-09	6.20E-06	1.37E-06	1.19E-06	6.92E-06
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	2.04E-07	9.91E-04	1.52E-03	1.16E-09	3.67E-06	1.46E-06	1.27E-06	3.85E-06
Chromium	2.60E-07	1.26E-03	1.94E-03	1.47E-09	1.18E-06	1.31E-07	1.13E-07	1.41E-06
Cobalt	1.76E-08	7.57E-05	1.16E-04	8.83E-11	8.01E-08	1.79E-08	1.06E-08	8.83E-08
Copper	1.78E-07	7.66E-04	1.18E-03	8.94E-10	5.70E-06	5.07E-06	3.82E-06	5.18E-06
Lead	6.62E-08	4.50E-04	6.91E-04	5.25E-10	5.11E-07	9.33E-08	8.08E-08	5.93E-07
Manganese	7.36E-08	3.43E-04	5.26E-04	4.00E-10	1.07E-06	7.90E-07	3.42E-07	7.40E-07
Mercury	4.82E-08	2.34E-04	1.69E-04	1.29E-10	1.43E-07	9.14E-08	7.92E-08	1.59E-07
Molybdenum	2.30E-07	9.91E-04	1.52E-03	1.16E-09	3.27E-06	2.45E-06	1.19E-06	2.37E-06
Nickel	3.95E-07	1.89E-03	2.91E-03	2.21E-09	1.96E-06	3.49E-07	3.02E-07	2.31E-06
Selenium	5.02E-09	2.16E-05	3.32E-05	2.52E-11	2.75E-08	1.10E-08	9.50E-09	3.14E-08
Strontium	-	-	-	-	-	-	-	-
Zinc	6.06E-06	2.61E-02	4.01E-02	3.05E-08	7.99E-05	5.42E-04	4.69E-04	8.46E-05
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	4.01E-07	2.78E-05	1.93E-06	1.47E-12	5.63E-08	1.75E-09	1.52E-11	6.46E-08

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 2a were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-7 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 2b (Off-Site Haulage along HR4) at Worst-Case Receptor Location

Chemical of Concern	*Ground-level Air Concentration*[a]	*Total Deposition Rate*[b]	*Soil*[c]	*Dust*[d]	*Exposed Produce*	*Protected Produce*	*Root Vegetables*	*Fruits*
	µg/m³	*mg/m²/yr*	*mg/kg dw*	*mg/kg dw*	*mg/kg ww*	*mg/kg ww*	*mg/kg ww*	*mg/kg ww*
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	2.99E-08	1.80E-04	2.76E-04	2.10E-10	1.74E-07	3.32E-08	2.87E-08	2.07E-07
Barium	8.16E-07	3.96E-03	6.09E-03	4.63E-09	6.20E-06	1.37E-06	1.19E-06	6.92E-06
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	2.04E-07	9.91E-04	1.52E-03	1.16E-09	3.67E-06	1.46E-06	1.27E-06	3.85E-06
Chromium	2.60E-07	1.26E-03	1.94E-03	1.47E-09	1.18E-06	1.31E-07	1.13E-07	1.41E-06
Cobalt	1.56E-08	7.57E-05	1.16E-04	8.83E-11	8.01E-08	1.79E-08	1.06E-08	8.83E-08
Copper	1.58E-07	7.66E-04	1.18E-03	8.94E-10	5.70E-06	5.07E-06	3.82E-06	5.18E-06
Lead	6.62E-08	4.50E-04	6.91E-04	5.25E-10	5.11E-07	9.33E-08	8.08E-08	5.93E-07
Manganese	8.62E-08	3.43E-04	5.26E-04	4.00E-10	1.07E-06	7.90E-07	3.42E-07	7.39E-07
Mercury	4.82E-08	2.34E-04	1.69E-04	1.29E-10	1.43E-07	9.14E-08	7.92E-08	1.59E-07
Molybdenum	2.04E-07	9.91E-04	1.52E-03	1.16E-09	3.27E-06	2.45E-06	1.19E-06	2.37E-06
Nickel	4.16E-07	1.89E-03	2.91E-03	2.21E-09	1.96E-06	3.49E-07	3.02E-07	2.31E-06
Selenium	4.45E-09	2.16E-05	3.32E-05	2.52E-11	2.75E-08	1.10E-08	9.50E-09	3.14E-08
Strontium	-	-	-	-	-	-	-	-
Zinc	5.38E-06	2.61E-02	4.01E-02	3.05E-08	7.99E-05	5.42E-04	4.69E-04	8.46E-05
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	2.82E-07	2.12E-05	1.47E-06	1.12E-12	4.17E-08	1.33E-09	1.16E-11	4.80E-08

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 2b were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-8 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 3a (On-Site Pelletization plus Haulage along HR1) at Worst-Case Receptor Location

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	2.70E-08	2.63E-04	4.03E-04	3.07E-10	2.54E-07	4.84E-08	4.20E-08	3.02E-07
Barium	8.16E-07	5.79E-03	8.88E-03	6.75E-09	9.05E-06	2.00E-06	1.73E-06	1.01E-05
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	2.30E-07	1.45E-03	2.22E-03	1.69E-09	5.35E-06	2.13E-06	1.85E-06	5.62E-06
Chromium	2.93E-07	1.84E-03	2.83E-03	2.15E-09	1.72E-06	1.91E-07	1.65E-07	2.06E-06
Cobalt	1.56E-08	1.10E-04	1.70E-04	1.29E-10	1.17E-07	2.61E-08	1.54E-08	1.29E-07
Copper	1.58E-07	1.12E-03	1.72E-03	1.30E-09	8.32E-06	7.40E-06	5.58E-06	7.56E-06
Lead	9.45E-08	6.57E-04	1.01E-03	7.66E-10	7.46E-07	1.36E-07	1.18E-07	8.66E-07
Manganese	8.05E-08	5.00E-04	7.67E-04	5.83E-10	1.56E-06	1.15E-06	4.99E-07	1.08E-06
Mercury	5.43E-08	3.42E-04	2.47E-04	1.88E-10	1.98E-07	1.33E-07	1.16E-07	2.21E-07
Molybdenum	2.04E-07	1.45E-03	2.22E-03	1.69E-09	4.77E-06	3.58E-06	1.73E-06	3.45E-06
Nickel	4.41E-07	2.76E-03	4.24E-03	3.22E-09	2.86E-06	5.09E-07	4.41E-07	3.37E-06
Selenium	4.45E-09	3.16E-05	4.85E-05	3.68E-11	4.01E-08	1.60E-08	1.39E-08	4.59E-08
Strontium	-	-	-	-	-	-	-	-
Zinc	5.38E-06	3.81E-02	5.86E-02	4.45E-08	1.17E-04	7.90E-04	6.85E-04	1.24E-04
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	1.39E-07	1.08E-05	7.52E-07	5.72E-13	2.05E-08	6.82E-10	5.91E-12	2.36E-08

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 3a were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-9 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 3b (On-Site Pelletization plus Haulage along HR4) at Worst-Case Receptor Location

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	3.90E-08	2.63E-04	4.03E-04	3.07E-10	2.54E-07	4.84E-08	4.20E-08	3.02E-07
Barium	9.20E-07	5.79E-03	8.88E-03	6.75E-09	9.05E-06	2.00E-06	1.73E-06	1.01E-05
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	2.30E-07	1.45E-03	2.22E-03	1.69E-09	5.35E-06	2.13E-06	1.85E-06	5.62E-06
Chromium	2.93E-07	1.84E-03	2.83E-03	2.15E-09	1.72E-06	1.91E-07	1.65E-07	2.06E-06
Cobalt	1.76E-08	1.10E-04	1.70E-04	1.29E-10	1.17E-07	2.61E-08	1.54E-08	1.29E-07
Copper	1.78E-07	1.12E-03	1.72E-03	1.30E-09	8.32E-06	7.40E-06	5.58E-06	7.56E-06
Lead	9.45E-08	6.57E-04	1.01E-03	7.66E-10	7.46E-07	1.36E-07	1.18E-07	8.66E-07
Manganese	8.36E-08	5.00E-04	7.67E-04	5.83E-10	1.56E-06	1.15E-06	4.99E-07	1.08E-06
Mercury	5.43E-08	3.42E-04	2.47E-04	1.88E-10	1.98E-07	1.33E-07	1.16E-07	2.21E-07
Molybdenum	2.30E-07	1.45E-03	2.22E-03	1.69E-09	4.77E-06	3.58E-06	1.73E-06	3.45E-06
Nickel	4.45E-07	2.76E-03	4.24E-03	3.22E-09	2.86E-06	5.09E-07	4.41E-07	3.37E-06
Selenium	5.02E-09	3.16E-05	4.85E-05	3.68E-11	4.01E-08	1.60E-08	1.39E-08	4.59E-08
Strontium	-	-	-	-	-	-	-	-
Zinc	6.06E-06	3.81E-02	5.86E-02	4.45E-08	1.17E-04	7.90E-04	6.85E-04	1.24E-04
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	9.77E-08	8.54E-06	5.93E-07	4.51E-13	1.54E-08	5.38E-10	4.66E-12	1.78E-08

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 3b were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-10 Summary of Predicted Project Alone Concentrations for Environmental Media – Base Case (Existing Multiple Hearth Incineration) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	4.91E-07	9.25E-04	1.42E-03	1.08E-09	1.44E-06	6.39E-07	5.54E-07	1.61E-06
Arsenic	3.98E-06	7.49E-03	1.15E-02	8.74E-09	7.27E-06	1.38E-06	1.20E-06	8.64E-06
Barium	1.46E-07	2.20E-04	3.38E-04	2.57E-10	3.45E-07	7.61E-08	6.60E-08	3.86E-07
Beryllium	1.05E-08	1.98E-05	3.04E-05	2.31E-11	1.75E-08	6.85E-10	5.94E-10	2.12E-08
Boron	1.12E-06	2.11E-03	3.25E-03	2.47E-09	1.24E-04	1.22E-04	8.44E-05	9.95E-05
Cadmium	1.95E-06	3.66E-03	5.62E-03	4.27E-09	1.36E-05	5.40E-06	4.68E-06	1.42E-05
Chromium	1.58E-06	2.96E-03	4.54E-03	3.45E-09	2.77E-06	3.07E-07	2.66E-07	3.31E-06
Cobalt	2.41E-08	4.44E-05	6.82E-05	5.18E-11	4.71E-08	1.05E-08	6.20E-09	5.19E-08
Copper	1.40E-05	2.64E-02	4.06E-02	3.08E-08	1.97E-04	1.75E-04	1.32E-04	1.79E-04
Lead	1.24E-05	2.34E-02	3.59E-02	2.72E-08	2.66E-05	4.84E-06	4.19E-06	3.08E-05
Manganese	2.84E-07	5.30E-04	8.14E-04	6.19E-10	1.66E-06	1.22E-06	5.29E-07	1.14E-06
Mercury	6.78E-06	1.28E-02	9.23E-03	7.01E-09	1.08E-05	4.98E-06	4.32E-06	1.17E-05
Molybdenum	2.25E-06	4.23E-03	6.50E-03	4.94E-09	1.40E-05	1.05E-05	5.07E-06	1.01E-05
Nickel	6.73E-07	1.24E-03	1.91E-03	1.45E-09	1.29E-06	2.29E-07	1.98E-07	1.52E-06
Selenium	3.74E-06	7.05E-03	1.08E-02	8.23E-09	8.98E-06	3.57E-06	3.10E-06	1.03E-05
Strontium	1.08E-07	2.03E-04	3.11E-04	2.36E-10	3.96E-06	3.79E-06	1.01E-06	1.37E-06
Zinc	1.48E-04	2.79E-01	4.28E-01	3.26E-07	8.53E-04	5.78E-03	5.01E-03	9.04E-04
Organic Parameters								
Polychlorinated biphenyls (PCBs)	2.58E-08	4.99E-05	4.23E-05	3.21E-11	4.62E-08	6.08E-07	5.27E-09	5.55E-08
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	1.03E-11	2.00E-08	1.69E-08	1.29E-14	1.85E-11	2.61E-10	2.26E-12	2.22E-11
Polycyclic aromatic hydrocarbons (PAHs)	5.25E-10	1.01E-06	7.00E-08	5.32E-14	8.88E-10	6.35E-11	5.51E-13	1.08E-09

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.
[b] Annual deposition rates resulting from the Base Case Scenario were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.
[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).
[d] Dust represents re-suspended dust from surface soil.

Table 4-11 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 1 (New Fluidized Bed Incineration) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	4.42E-08	8.33E-05	1.28E-04	9.72E-11	1.30E-07	5.75E-08	4.99E-08	1.45E-07
Arsenic	3.61E-07	6.75E-04	1.04E-03	7.88E-10	6.55E-07	1.24E-07	1.08E-07	7.79E-07
Barium	4.84E-08	3.60E-05	5.53E-05	4.20E-11	5.64E-08	1.24E-08	1.08E-08	6.30E-08
Beryllium	9.47E-10	1.78E-06	2.74E-06	2.08E-12	1.58E-09	6.16E-11	5.34E-11	1.90E-09
Boron	1.01E-07	1.90E-04	2.92E-04	2.22E-10	1.11E-05	1.10E-05	7.60E-06	8.96E-06
Cadmium	1.84E-07	3.34E-04	5.12E-04	3.89E-10	1.24E-06	4.92E-07	4.26E-07	1.30E-06
Chromium	1.53E-07	2.71E-04	4.17E-04	3.17E-10	2.54E-07	2.81E-08	2.44E-08	3.04E-07
Cobalt	2.84E-09	4.30E-06	6.61E-06	5.02E-12	4.57E-09	1.02E-09	6.01E-10	5.03E-09
Copper	1.27E-06	2.38E-03	3.66E-03	2.78E-09	1.77E-05	1.58E-05	1.19E-05	1.61E-05
Lead	1.12E-06	2.10E-03	3.23E-03	2.45E-09	2.39E-06	4.36E-07	3.78E-07	2.78E-06
Manganese	3.12E-08	4.92E-05	7.55E-05	5.74E-11	1.54E-07	1.13E-07	4.91E-08	1.06E-07
Mercury	6.78E-06	1.28E-02	9.23E-03	7.01E-09	1.08E-05	4.98E-06	4.32E-06	1.17E-05
Molybdenum	2.12E-07	3.85E-04	5.91E-04	4.49E-10	1.27E-06	9.53E-07	4.61E-07	9.20E-07
Nickel	8.18E-08	1.20E-04	1.84E-04	1.40E-10	1.24E-07	2.20E-08	1.91E-08	1.46E-07
Selenium	3.37E-07	6.35E-04	9.74E-04	7.40E-10	8.08E-07	3.21E-07	2.79E-07	9.24E-07
Strontium	9.68E-09	1.82E-05	2.80E-05	2.13E-11	3.56E-07	3.41E-07	9.10E-08	1.23E-07
Zinc	1.36E-05	2.52E-02	3.87E-02	2.94E-08	7.71E-05	5.23E-04	4.53E-04	8.17E-05
Organic Parameters								
Polychlorinated biphenyls (PCBs)	7.47E-13	1.45E-09	1.23E-09	9.31E-16	1.34E-12	1.76E-11	1.53E-13	1.61E-12
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	7.47E-13	1.45E-09	1.23E-09	9.34E-16	1.34E-12	1.90E-11	1.64E-13	1.61E-12
Polycyclic aromatic hydrocarbons (PAHs)	1.10E-07	2.15E-04	1.49E-05	1.13E-11	1.89E-07	1.35E-08	1.17E-10	2.29E-07

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.
[b] Annual deposition rates resulting from Alternative 1 were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.
[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).
[d] Dust represents re-suspended dust from surface soil.

Table 4-12 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 2a (Off-Site Haulage along HR1) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	3.00E-09	8.43E-07	1.29E-06	9.84E-13	8.21E-10	1.55E-10	1.35E-10	9.76E-10
Barium	5.45E-08	1.78E-05	2.73E-05	2.07E-11	2.79E-08	6.14E-09	5.32E-09	3.11E-08
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	9.68E-09	4.44E-06	6.82E-06	5.18E-12	1.65E-08	6.55E-09	5.67E-09	1.73E-08
Chromium	1.23E-08	5.65E-06	8.68E-06	6.60E-12	5.31E-09	5.86E-10	5.08E-10	6.35E-09
Cobalt	1.04E-09	3.39E-07	5.21E-07	3.96E-13	3.60E-10	8.01E-11	4.74E-11	3.97E-10
Copper	1.05E-08	3.43E-06	5.27E-06	4.00E-12	2.56E-08	2.27E-08	1.71E-08	2.32E-08
Lead	4.11E-09	2.00E-06	3.08E-06	2.34E-12	2.28E-09	4.15E-10	3.60E-10	2.65E-09
Manganese	4.50E-09	1.61E-06	2.47E-06	1.88E-12	5.04E-09	3.71E-09	1.61E-09	3.48E-09
Mercury	2.29E-09	1.05E-06	7.58E-07	5.76E-13	2.13E-09	4.09E-10	3.55E-10	2.20E-09
Molybdenum	1.36E-08	4.44E-06	6.82E-06	5.18E-12	1.47E-08	1.10E-08	5.32E-09	1.06E-08
Nickel	2.05E-08	8.61E-06	1.32E-05	1.00E-11	8.96E-09	1.59E-09	1.37E-09	1.05E-08
Selenium	2.97E-10	9.69E-08	1.49E-07	1.13E-13	1.24E-10	4.91E-11	4.26E-11	1.41E-10
Strontium	-	-	-	-	-	-	-	-
Zinc	3.59E-07	1.17E-04	1.80E-04	1.37E-10	3.58E-07	2.43E-06	2.10E-06	3.80E-07
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	7.99E-09	4.07E-07	2.83E-08	2.15E-14	9.27E-10	2.57E-11	2.22E-13	1.05E-09

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 2a were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-13 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 2b (Off-Site Haulage along HR4) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	2.52E-09	8.02E-07	1.23E-06	9.36E-13	7.80E-10	1.48E-10	1.28E-10	9.27E-10
Barium	3.87E-08	1.78E-05	2.73E-05	2.07E-11	2.79E-08	6.14E-09	5.32E-09	3.11E-08
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	9.68E-09	4.44E-06	6.82E-06	5.18E-12	1.65E-08	6.55E-09	5.67E-09	1.73E-08
Chromium	1.23E-08	5.65E-06	8.68E-06	6.60E-12	5.31E-09	5.86E-10	5.08E-10	6.35E-09
Cobalt	7.39E-10	3.39E-07	5.21E-07	3.96E-13	3.60E-10	8.01E-11	4.74E-11	3.97E-10
Copper	7.48E-09	3.43E-06	5.27E-06	4.00E-12	2.56E-08	2.27E-08	1.71E-08	2.32E-08
Lead	4.11E-09	2.03E-06	3.11E-06	2.36E-12	2.31E-09	4.20E-10	3.64E-10	2.68E-09
Manganese	4.92E-09	1.57E-06	2.42E-06	1.84E-12	4.92E-09	3.62E-09	1.57E-09	3.40E-09
Mercury	2.29E-09	1.05E-06	7.58E-07	5.76E-13	2.13E-09	4.09E-10	3.55E-10	2.20E-09
Molybdenum	9.68E-09	4.44E-06	6.82E-06	5.18E-12	1.47E-08	1.10E-08	5.32E-09	1.06E-08
Nickel	2.12E-08	8.55E-06	1.31E-05	9.97E-12	8.90E-09	1.57E-09	1.36E-09	1.05E-08
Selenium	2.11E-10	9.69E-08	1.49E-07	1.13E-13	1.24E-10	4.91E-11	4.26E-11	1.41E-10
Strontium	-	-	-	-	-	-	-	-
Zinc	2.55E-07	1.17E-04	1.80E-04	1.37E-10	3.58E-07	2.43E-06	2.10E-06	3.80E-07
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	1.09E-08	6.04E-07	4.19E-08	3.19E-14	1.33E-09	3.81E-11	3.30E-13	1.52E-09

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 2b were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-14 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 3a (On-Site Pelletization plus Haulage along HR1) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	1.87E-09	1.18E-06	1.82E-06	1.38E-12	1.15E-09	2.18E-10	1.89E-10	1.37E-09
Barium	3.87E-08	2.58E-05	3.96E-05	3.01E-11	4.05E-08	8.92E-09	7.73E-09	4.52E-08
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	1.36E-08	6.46E-06	9.91E-06	7.53E-12	2.39E-08	9.51E-09	8.25E-09	2.51E-08
Chromium	1.73E-08	8.22E-06	1.26E-05	9.59E-12	7.71E-09	8.51E-10	7.38E-10	9.22E-09
Cobalt	7.39E-10	4.93E-07	7.57E-07	5.75E-13	5.24E-10	1.16E-10	6.89E-11	5.77E-10
Copper	7.48E-09	4.99E-06	7.66E-06	5.82E-12	3.72E-08	3.30E-08	2.49E-08	3.38E-08
Lead	5.90E-09	2.92E-06	4.48E-06	3.41E-12	3.33E-09	6.05E-10	5.24E-10	3.86E-09
Manganese	5.11E-09	2.26E-06	3.46E-06	2.63E-12	7.06E-09	5.20E-09	2.25E-09	4.88E-09
Mercury	3.22E-09	1.53E-06	1.10E-06	8.38E-13	3.02E-09	5.95E-10	5.16E-10	3.12E-09
Molybdenum	9.68E-09	6.46E-06	9.91E-06	7.53E-12	2.13E-08	1.60E-08	7.73E-09	1.54E-08
Nickel	2.67E-08	1.24E-05	1.90E-05	1.44E-11	1.29E-08	2.28E-09	1.97E-09	1.51E-08
Selenium	2.11E-10	1.41E-07	2.16E-07	1.64E-13	1.80E-10	7.14E-11	6.18E-11	2.05E-10
Strontium	-	-	-	-	-	-	-	-
Zinc	2.55E-07	1.70E-04	2.61E-04	1.99E-10	5.21E-07	3.53E-06	3.06E-06	5.52E-07
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	2.78E-09	1.47E-07	1.02E-08	7.77E-15	3.27E-10	9.28E-12	8.04E-14	3.71E-10

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 3a were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

Table 4-15 Summary of Predicted Project Alone Concentrations for Environmental Media – Alternative 3b (On-Site Pelletization plus Haulage along HR4) on Average across the Study Area

Chemical of Concern	Ground-level Air Concentration[a]	Total Deposition Rate[b]	Soil[c]	Dust[d]	Exposed Produce	Protected Produce	Root Vegetables	Fruits
	µg/m³	mg/m²/yr	mg/kg dw	mg/kg dw	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Inorganic Parameters								
Antimony	-	-	-	-	-	-	-	-
Arsenic	2.66E-09	1.17E-06	1.79E-06	1.36E-12	1.14E-09	2.15E-10	1.86E-10	1.35E-09
Barium	5.45E-08	2.58E-05	3.96E-05	3.01E-11	4.05E-08	8.92E-09	7.73E-09	4.52E-08
Beryllium	-	-	-	-	-	-	-	-
Boron	-	-	-	-	-	-	-	-
Cadmium	1.36E-08	6.46E-06	9.91E-06	7.53E-12	2.39E-08	9.51E-09	8.25E-09	2.51E-08
Chromium	1.73E-08	8.22E-06	1.26E-05	9.59E-12	7.71E-09	8.51E-10	7.38E-10	9.22E-09
Cobalt	1.04E-09	4.93E-07	7.57E-07	5.75E-13	5.24E-10	1.16E-10	6.89E-11	5.77E-10
Copper	1.05E-08	4.99E-06	7.66E-06	5.82E-12	3.72E-08	3.30E-08	2.49E-08	3.38E-08
Lead	5.90E-09	2.92E-06	4.48E-06	3.41E-12	3.33E-09	6.05E-10	5.24E-10	3.86E-09
Manganese	5.25E-09	2.26E-06	3.46E-06	2.63E-12	7.06E-09	5.20E-09	2.25E-09	4.88E-09
Mercury	3.22E-09	1.53E-06	1.10E-06	8.38E-13	3.02E-09	5.95E-10	5.16E-10	3.12E-09
Molybdenum	1.36E-08	6.46E-06	9.91E-06	7.53E-12	2.13E-08	1.60E-08	7.73E-09	1.54E-08
Nickel	2.70E-08	1.24E-05	1.90E-05	1.44E-11	1.29E-08	2.28E-09	1.97E-09	1.51E-08
Selenium	2.97E-10	1.41E-07	2.16E-07	1.64E-13	1.80E-10	7.14E-11	6.18E-11	2.05E-10
Strontium	-	-	-	-	-	-	-	-
Zinc	3.59E-07	1.70E-04	2.61E-04	1.99E-10	5.21E-07	3.53E-06	3.06E-06	5.52E-07
Organic Parameters								
Polycyclic aromatic hydrocarbons (PAHs)	3.79E-09	2.15E-07	1.50E-08	1.14E-14	4.68E-10	1.36E-11	1.18E-13	5.33E-10

Note: "-" values indicate COCs for which emissions data was unavailable for this Scenario.

[a] Ground-level air concentrations were used in the multi-media assessment to facilitate the prediction of volatile chemical concentrations in home garden produce.

[b] Annual deposition rates resulting from Alternative 3b were used to predict soil concentrations and deposition onto vegetation after 48 years of deposition.

[c] Surface soil concentrations used to evaluate the incidental soil ingestion pathway were predicted assuming a 2 cm mixing zone as per US EPA (2005).

[d] Dust represents re-suspended dust from surface soil.

4.3 Exposure Analysis of Particulate Matter

The size of the airborne particles to which people are exposed is one of the most important aspects in determining the potential for health risk resulting from PM exposure. Size is directly related to where particles will be deposited in specific parts of the respiratory tract. Particles larger than about 10 microns (*μm*) in aerodynamic diameter (>PM_{10}) are deposited almost exclusively in the nose, throat, and upper respiratory tract, and tend to be coughed out over a very short period of time. This size range is considered outside the inhalable range for people, since these particles are too large to be deposited in the lung. Health effects associated with particles greater than PM_{10} are considered less critical compared to fractions less than 10 microns in size since they are less likely to be absorbed into the body *via* inhalation. Fine and ultrafine particles (<2.5 μm), on the other hand, are small enough to reach the alveoli (air spaces) deep in the lungs. In general, it may be assumed that the smaller the particle, the greater the potential to reach respiratory structures such as alveoli where blood-gas exchange occurs. Inhaled fine and ultrafine particles can also carry adsorbed chemical pollutants to the deeper lung structures. Smaller particles tend to be present in greater numbers, and they possess a greater total surface area than larger particles of the same mass.

The potential impacts of human exposure to the respirable fraction of PM (*i.e.*, $PM_{2.5}$ and PM_{10}) were emphasized in the current HHRA, rather than the broader size fraction represented by total suspended particulate (*i.e.*, TSP, comprising particles ranging up to 44 μm in size). The inhalable fraction (*i.e.*, PM_{10}) is also widely used to evaluate potential health issues, since this size of particle primarily affects tissues in the upper airways, but can also travel deep into the lung. When both sets of data are available (PM_{10} and $PM_{2.5}$), the $PM_{2.5}$ data tends to carry more weight in determining the potential for health risks because of the large body of scientific literature characterizing both the epidemiological and toxicological properties of the finer size fraction. Furthermore, the $PM_{2.5}$ size fraction is typically the most relevant size fraction for vehicle exhaust emissions, and as such is particularly relevant for the transportation scenario.

4.3.1 Uncertainties Related to Ultrafine Particulate Matter (UFP)

The potential health impact of ultrafine particulate matter (*i.e.*, UFP or $PM_{0.1}$) is an emerging area of scientific enquiry. As combustion emission byproducts and produced through secondary atmospheric transformations, ambient UFPs have many potential environmental sources whose relative contributions to ambient concentrations vary with location, season, and time-of-day. However, in urban areas, particularly in proximity to major roads, motor vehicle exhaust can be identified as the major contributor to UFP concentrations. In particular, diesel vehicles have been found to contribute substantially, sometimes in disproportion to their numbers in the vehicle fleet (HEI, 2013).

The unique physical properties of UFPs, their interactions with tissues and cells, and their potential for easy movement within the body beyond the lungs have lead researchers to suspect that UFPs may have specific or enhanced toxicity relative to other particle size fractions and may contribute to effects beyond the respiratory system. However, the considerable body of research that has been conducted has not been able to definitively confirm this possibility (HEI, 2013). To date, toxicological studies in animals, controlled human exposure studies, and epidemiologic studies have not provided consistent findings on the effects of exposures to ambient levels of UFPs, particularly in human populations. Most importantly, the current scientific evidence does not support a conclusion that exposures to UFPs alone can account in substantial ways for the adverse effects that have been associated with other ambient pollutants, such as $PM_{2.5}$ (HEI, 2013).

Currently there are no established accepted reference benchmarks or standardized approaches to evaluation of the health impact related to exposures to this particulate matter fraction. As such, for the current assessment, the ultrafine fraction was considered as part of the evaluation of health impacts related to the $PM_{2.5}$ (*i.e.*, particulate matter less than 2.5 microns in size) group. However, the uncertainties related to both exposures and health impacts from UFPs, particularly as it pertains to combustion emissions from industrial facilities, is something that should flagged for further consideration in the future once additional scientific information on this particle size fraction becomes available.

Therefore, only the PM_{10} and $PM_{2.5}$ size fractions were overtly evaluated in the current assessment.

5.0 HAZARD ASSESSMENT

All chemicals have the potential to cause toxicological effects; however, it is the chemical concentration, the route of exposure, the duration of exposure, and the inherent toxicity of the chemical that determines the level of effect and hence the potential for adverse health effects. In this stage of the HHRA, toxicity reference values (TRVs) to be used to characterize health risks were selected for each COC.

When TRVs for a particular COC were available from multiple regulatory agencies, values were reviewed and the professional judgment of an experienced toxicologist and/or risk assessor was used to select the most appropriate TRV. A number of different considerations went into selecting a TRV for use in the HHRA, including:

- The source of the information. Is the TRV derived by a reputable regulatory agency?
- Is there sufficient documentation available concerning the derivation of the TRV (*e.g.,* study, endpoint, point of departure, uncertainty factors applied, *etc.*)?
- How current is the derivation of the TRV?
- How relevant is the TRV in terms of exposure route and duration of interest?

The TRVs employed in the current HHRA were obtained from reputable regulatory agencies including, but not limited to:

- Ontario Ministry of the Environment and Climate Change (MOECC);
- Health Canada;
- US EPA Integrated Risk Information System (US EPA IRIS);
- Agency for Toxic Substances and Disease Registry (ATSDR);
- Canadian Council of the Ministers of the Environment (CCME);
- World Health Organization (WHO);
- California Environmental Protection Agency (Cal EPA); and,
- Texas Commission on Environmental Quality (TCEQ).

For the current assessment, selection of TRVs was conducted in consultation with Toronto Public Health. In particular, TRVs used by TPH in previous assessments of air quality within the City of Toronto (*i.e.*, Local Air Quality Assessment, or LAQ, reports for various Wards) were given preference unless alternative, more recent or appropriate reference benchmarks were available.

A summary of the non-carcinogenic and carcinogenic TRVs used in both the inhalation and multi-media assessments are summarized in Tables 5-1 through 5-3. Refer to Appendix A for further details concerning each TRV considered and, where necessary, the rationale used to select the specific TRV.

5.1 Acute Toxicity Reference Values

The acute (*i.e.*, 24-hour exposure durations) non-carcinogenic inhalation TRVs for each of the COCs (where they were available), as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 5-1.

While the Ontario Ministry of the Environment and Climate Change has established a series of 24-hour ambient air quality criteria (AAQC), many of these are not based on acute toxicological endpoints and/or outcomes. Rather, in the case of a number of the COCs, these 24-hour AAQC are actually based on chronic toxicological outcomes requiring long-term exposures adjusted to a 24-hour averaging period for regulatory compliance and enforcement purposes.

In response to this issue, the MOECC recommends the following (J. Gilmore, personal communication, 2015):

> *"HHRAs should use appropriately supported human health based TRVs (scientifically sound and up to date) and should be linked to the duration of exposure (e.g., acute, sub-chronic or chronic effects on human health) against which an air concentration is assessed. It is noted that AAQCs may:*
>
> - *not differentiate between cancer and non-cancer effects*
> - *not be based on human health effects (e.g., environmental (e.g., ecological) or nuisance effects).*
> - *not differentiate as to whether they are based on an acute, sub-chronic or chronic health effect, let alone for a cancer or non-cancer effect. For example, the ministry uses meteorological conversion factors to adjust averaging times of AAQCs to facilitate the assessment of air quality (e.g., carcinogens are extrapolated from an annual AAQC to a 24 hour AAQC). AAQCs with 24-hour averaging time are usually based on protection in long-term continuous exposures and are not "acute" values (this is often misinterpreted).*
> - *not be based on current science.*
>
> *However, in the absence of a readily identifiable TRV, an AAQC may be used as long as the effect on which it is based is accurately described. For those AAQCs that are not directly based on human health, it is more appropriate to use concentration ratios (CRs) rather than hazard quotients since the exceedance of an AAQC may not reflect the potential for an adverse human health effect."*

Based on this guidance, only those 24-hour AAQC that are based on an acute effect toxicological endpoint were considered for the current assessment. Furthermore, inhalation pathways are evaluated using concentration ratios, while multi-media risks (*e.g.*, from oral and dermal exposures) are evaluated using hazard quotients.

Table 5-1 Summary of Acute-Duration Inhalation TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Inhalation TRVs (µg/m^3)			
	Duration	***Value***	***Critical Effect***	***Source***
Criteria Air Contaminants (CACs)				
Carbon monoxide	8-Hour	6,000	Carboxyhemoglobin blood level of less than 1%	Health Canada, 2006
Nitrogen dioxide (NO_2)	24-Hour	200	Respiratory tract irritation	MOE, 2012
Ozone	8-hour	100	Estimated 1–2% increase in daily mortality (based on findings of daily time series studies)	WHO, 2006
Respirable particulate matter ($PM_{2.5}$)	24-Hour	27	Respiratory tract irritation	CCME, 2012
Inhalable particulate matter (PM_{10})	24-Hour	50	Respiratory tract irritation	WHO, 2006a
Sulphur dioxide (SO_2)	24-Hour	275	Respiratory tract irritation	MOE, 2012

Table 5-1 Summary of Acute-Duration Inhalation TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Inhalation TRVs (µg/m³)			
	Duration	*Value*	*Critical Effect*	*Source*
Inorganics				
Antimony	24-Hour	25	Skin and eye irritation	MOE, 2012
Arsenic	24-Hour	0.3	Respiratory tract irritation, gastrointestinal effects, and central nervous system depression	MOE, 2012
Beryllium	24-Hour	0.01	Respiratory tract irritation and pulmonary effects	MOE, 2012
Boron	24-Hour	300	Increased nasal secretions (human)	ATSDR, 2010
Cadmium	24-Hour	0.03	Histological changes in the respiratory tract (rat, mouse)	ATSDR, 2012a
Chromium (total)	24-Hour	0.5	Respiratory effects (rodents)	MOE, 2011b
Chromium (VI)	24-hour	0.0007 (in TSP)	Respiratory irritation	MOE, 2012
Cobalt	24-Hour	0.1	Respiratory irritation	MOE, 2012
Manganese	24-Hour	0.4 (in TSP)	Adverse central nervous system effects (occupational exposure)	MOE, 2012
Selenium	24-Hour	10	Respiratory irritation	MOE, 2012
Strontium	24-Hour	120	Respiratory irritation based on particulate levels	MOE, 2012
Volatile Organic Compounds (VOCs)				
Acetaldehyde	24-Hour	500	Tissue damage	MOE, 2012
Acrolein	24-Hour	0.4	Eye irritation	MOE, 2012
Benzene	24-Hour	29	Reduces lymphocyte proliferation following mitogen stimulation	ATSDR, 2007a
Dichlorobenzene, 1,4-	24-Hour	95	Eye and respiratory system irritation	MOE, 2012
Dichloromethane	24-Hour	220	Central nervous system depression	MOE, 2012
Formaldehyde	24-Hour	65	Respiratory and eye irritation	MOE, 2012
Tetrachloroethylene	24-Hour	360	Central nervous system depression and respiratory system effects	MOE, 2012
Toluene	24-Hour	3,800	Neurological effects (human)	ATSDR, 2000
Trichloroethylene	24-Hour	12	Central nervous system, eye, and respiratory system effects	MOE, 2012
Vinyl chloride	24-Hour	1	Central nervous system depression	MOE, 2012

TSP Total suspended particulate

It should be noted that the typical regulatory approach in Canada to evaluating ambient air concentrations of the criteria air contaminants is through a comparison to Canada Wide Standards (CWS) or National Ambient Air Quality Objectives (NAAQOs). These standards and objectives typically provide the benchmark by which emissions from a proposed project are evaluated for acceptability, from both a federal and provincial compliance point-of-view. However, it should be noted that the NAAQOs for NOx and SO_2 are not specifically health risk-based. Many of these standards and objectives are dated (*i.e.*, established in 1974/5), do not include the most recent scientific health-based knowledge, and are impacted by policy decisions in their derivation. As such, any discussion on the effect of air pollution cannot rely on the attainment of such "standards" to guarantee that health within exposed population will be protected.

Ozone is a unique COC that is not actually emitted by any of the proposed biosolids treatment alternatives or the existing HCTP incineration facility. Ozone is formed through secondary reactions in the atmosphere based on the reaction of precursor chemicals (such as oxides of nitrogen and VOCs) with sunlight under certain meteorological conditions. As the primary health concern arising from ambient exposures to ozone are acute respiratory impacts leading to morbidity and mortality outcomes over multiple hours of exposure, the primary averaging period evaluated by regulatory agencies is an 8-hour exposure window.

Unfortunately, the air dispersion model used by the City of Toronto to develop airshed ambient concentrations of the various COC can only accurately estimate an annual average concentrations of ozone, and not a value for an 8-hour exposure period. However, given the very low concentrations of NO_x and VOCs predicted to be emitted by the various biosolids treatment alternatives, it is unlikely that the any of the alternatives would result in a significant increase in ambient ozone concentrations within the Study Area.

As such, for the current assessment, ozone will only be assessed as part of the CAC group evaluated for impacts on premature mortality rates within the Study Area (as discussed in Section 5.2.2).

5.2 Chronic Toxicity Reference Values

5.2.1 Inhalation Exposures

The chronic non-carcinogenic and carcinogenic inhalation TRVs for each of the COCs (where they were available), as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 5-2.

Table 5-2 Summary of Chronic-Duration Inhalation TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Inhalation TRVs (µg/m^3)				Carcinogenic Inhalation Unit Risk ((µg/m^3)$^{-1}$)		
	Duration	*Value*	*Critical Effect*	*Source*	*Value*	*Critical Effect*	*Source*
Criteria Air Contaminants (CACs)							
Nitrogen dioxide (NO_2)	Annual average	40	Respiratory effects	WHO, 2006a	NA	-	-
Respirable particulate matter ($PM_{2.5}$)	Annual average	8.8	Cardiopulmonary and lung cancer mortality increase (human)	CCME, 2012	NA	-	-
Inhalable particulate matter (PM_{10})	Annual average	20	Lowest levels at which total, cardiopulmonary and lung cancer mortality has been shown to increase (human)	WHO, 2006a	NA	-	-
Sulphur dioxide (SO_2)	Annual average	29	Respiratory inflammation (human)	Health Canada, 2006	NA	-	-
Inorganics							
Antimony	Chronic	0.2	Pulmonary toxicity, chronic interstitial inflammation (rat)	US EPA IRIS, 1995a	NA	-	-
Arsenic	Chronic	0.015	Decreased intellectual function (human)	Cal EPA, 2014	$6.4x10^{-3}$	Lung cancer (human)	Health Canada, 2010
Barium	Chronic	1	Cardiovascular effects (human)	RIVIM, 2001	NA	-	-
Beryllium	Chronic	0.007	Beryllium sensitization and progression to chronic beryllium disease (human)	Cal EPA, 2001	$2.4x10^{-3}$	Lung cancer (human)	US EPA IRIS, 1998a
Boron	Annual average	5 [a]	Respiratory effects	TCEQ, 2014	NA	-	-
Cadmium	Annual average	0.005	Proteinuria associated with proximal tubular dysfunction and lung cancer (human)	MOE, 2007	$9.8x10^{-3}$	Lung tumours (rat)	Health Canada, 2010
Chromium (total)	Chronic	0.14	Increased total lung and trachea weight (rat)	TCEQ, 2009a	$1.1x10^{-2}$	Increased incidence of lung cancer (human)	Health Canada, 2010
Chromium (VI)	Chronic	0.1	Lactate dehydrogenase in bronchialveolar lavage fluid (rat)	US EPA IRIS, 1998b	$7.6x10^{-2}$	Lung cancer (human)	Health Canada, 2010
Cobalt	Chronic	0.1	Respiratory symptoms and effects on lung function (human)	WHO, 2006b	NA	-	-
Copper	Annual average	1[a]	Respiratory and immunological effects	TCEQ, 2014	NA	-	-
Lead	Annual average	0.15	Protective of children and other at-risk populations	US EPA, 2008	$1.2x10^{-5}$	Kidney tumour incidence (rat)	Cal EPA, 2011

Table 5-2 Summary of Chronic-Duration Inhalation TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Inhalation TRVs (µg/m^3)				Carcinogenic Inhalation Unit Risk ((µg/m^3)$^{-1}$)		
	Duration	*Value*	*Critical Effect*	*Source*	*Value*	*Critical Effect*	*Source*
Manganese	Annual average	0.05	Impairment of neurobehavioral function (human)	US EPA IRIS, 1993a	NA	-	-
Mercury (inorganic)	Chronic	0.03	Hand tremors, cognitive effects (human)	Cal EPA, 2014	NA	-	-
Molybdenum	Annual average	3	Health-based	TCEQ, 2014	NA	-	-
Nickel	Chronic	0.014	Pathological changes to respiratory system and hematologic system (rat)	Cal EPA, 2014	2.6×10^{-4}	Lung and nasal cancer incidence (occupational exposure)	Cal EPA, 2011
Selenium	Annual average	0.2	Eye and upper respiratory tract irritation	TCEQ, 2014	NA	-	-
Strontium	Chronic	2 [a]	Respiratory inflammation	TCEQ, 2014	NA	-	-
Zinc	Annual average	2 [a]	Respiratory inflammation	TCEQ, 2014	NA	-	-
Volatile Organic Compounds (VOCs)							
Acetaldehyde	Chronic	140	Degeneration of olfactory epithelium (rat)	Cal EPA, 2014	2.7×10^{-6}	Nasal tumour incidence (rat)	Cal EPA, 2011
Acrolein	Chronic	0.02	Nasal lesions (rat)	US EPA IRIS, 2003a	NA	-	-
Benzene	Chronic	3	Statistically significant decreased counts of B-lymphocytes (human)	Cal EPA, 2014	2.9×10^{-5}	Leukemia incidence (occupational exposure)	Cal EPA, 2011
Butadiene, 1,3-	Chronic	2	Ovarian atrophy (rat)	US EPA IRIS, 2002	5.0×10^{-7}	Leukemia incidence data (human)	TCEQ, 2008
Carbon tetrachloride	Chronic	100	Fatty change in liver (rat, mouse)	US EPA IRIS, 2010	6.0×10^{-6}	Pheochromocytoma (mouse)	US EPA IRIS, 2010
Chloroform	Chronic	100	Hepatomegaly, toxic hepatitis, and hepatosteatosis (human)	ATSDR, 1997a	5.3×10^{-6}	Renal tumors (rat, mouse)	CAL EPA, 2011
Dichlorobenzene, 1,4-	Chronic	60	Incidences of nasal lesions (rat)	ATSDR, 2006	1.1×10^{-5}	Liver tumours (mouse)	Cal EPA, 2011
Dichloroethane, 1,2-	Chronic	400	Hepatotoxicity (rat)	Cal EPA, 2014	2.6×10^{-5}	Hemangiosarcomas (rat)	US EPA IRIS, 1991a
Dichloromethane	Chronic	400	COHb formation (human)	Cal EPA, 2014	1.0×10^{-6}	Lung tumors (mouse)	Cal EPA, 2011
Di-n-octyl phthalate	Chronic	NA	-	-	NA	-	-
Ethylene dibromide	Chronic	0.8	Reproductive effects (human)	Cal EPA, 2014	6.0×10^{-4}	Nasal cavity tumours, hemangiosarcomas, and mesotheliomas (rat)	US EPA IRIS, 2004a

Table 5-2 Summary of Chronic-Duration Inhalation TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Inhalation TRVs (µg/m^3)				Carcinogenic Inhalation Unit Risk ((µg/m^3)$^{-1}$)		
	Duration	*Value*	*Critical Effect*	*Source*	*Value*	*Critical Effect*	*Source*
Formaldehyde	Chronic	9	Nasal obstruction and discomfort, lower airway discomfort, eye irritation (human)	Cal EPA, 2014	$6.0x10^{-6}$	Nasal squamous carcinoma incidence (rat)	Cal EPA, 2011
Hexachlorobutadiene	Chronic	0.2	Health-based	TCEQ, 2014	$2.2x10^{-5}$	Renal tubular adenomas and adenocarcinomas (rat)	US EPA IRIS, 1991b
Tetrachloroethylene	Chronic	40	Neurotoxicity (human)	US EPA IRIS, 2012a	$2.6x10^{-7}$	Hepatocellular adenomas or carcinomas (mice)	US EPA IRIS, 2012a
Toluene	Chronic	5,000	Neurological effects in occupationally-exposed workers	US EPA IRIS, 2005a	NA	-	-
Trichloroethylene	Chronic	2	Decreased thymus weights and fetal heart malformations (mouse)	US EPA IRIS, 2011	$4.1x10^{-6}$	Renal cell carcinoma (human)	US EPA IRIS, 2011
Vinyl chloride	Annual average	60	Centrilobular hypertrophy in the liver (rat)	TCEQ, 2009b	$7.8x10^{-5}$	Increased tumor incidence (mice)	Cal EPA, 2011
Polychlorinated biphenyls (PCBs)							
PCBs (total)	Annual average	0.035	Systemic effects	MOE, 2012	$1.0x10^{-4}$	Liver adenomas and carcinomas (rat)	US EPA IRIS, 1997
Polychlorinated dibenzo-p-dioxins and Polychlorinated dibenzofurans (PCDD/PCDF)							
PCDD/F as Toxic Equivalents (TEQ)	Chronic	40 pg/m^3	Increased mortality, systemic effects, effects to numerous organs (rat)	Cal EPA, 2014	NA	-	-
Carcinogenic Polycyclic Aromatic Hydrocarbons (PAHs)							
PAHs as Benzo(a)pyrene Toxic Equivalents (BaP TEQ)	NA	-	-	-	$1.1x10^{-3}$	Respiratory tract tumour (hamster)	Cal EPA, 2011

NA Not available. No TRV or benchmark is available for this endpoint.
[a] In PM_{10}

5.2.2 Morbidity and Premature Mortality Risks

As discussed in Section 2.1.5.1, the City of Toronto has previously completed an evaluation of health risks associated with multiple respiratory and cardiovascular outcomes leading to chronic morbidity or premature mortality risks as part of their Local Air Quality (LAQ) reports on a number of City Wards. As discussed in TPH (2014), the excess risk of premature mortality due to exposure to mixture groups of COCs related to these types of health outcomes can be estimated based on the set of concentration response function (CRF) coefficients endorsed by Health Canada for use in its Air Quality Benefits Assessment Tool (AQBAT). These CRF coefficients represent statistically derived estimates of the percent (%) excess health endpoint associated with a unit increase in the pollutant concentration (Health Canada, 2006).

Table 5-3 provides a list of the key CRF values established by Health Canada which were used in the evaluation of potential changes in chronic morbidity and premature mortality rates within the Study Area relative to the existing base case conditions. These are the same CRF values used by the City of Toronto in previous LAQ reports (TPH, 2014) and are evaluated against long-term (*i.e.*, annual average) exposures to the CAC group.

Table 5-3 Summary of Concentration Response Functions (CRFs) for evaluation of acute and chronic CAC exposures within the Study Area

Criteria Air Contaminant	Incremental Risk CRF Coefficient ($(\mu g/m^3)^{-1}$)		
	Premature Mortality	Cardiovascular Hospital Admissions	Respiratory Hospital Admissions
Carbon monoxide	1.60×10^{-6}	-	-
Nitrogen dioxide (NO_2)	4.00×10^{-4}	-	-
Ozone	4.20×10^{-4}	-	-
Respirable particulate matter ($PM_{2.5}$)	6.76×10^{-3}	7.11×10^{-4}	7.54×10^{-4}
Sulphur dioxide (SO_2)	1.75×10^{-4}	-	-

Given the PM_{10} size fraction also include $PM_{2.5}$, to avoid double-counting when estimating cumulative risks, only $PM_{2.5}$ was included in the evaluation of chronic morbidity and mortality impacts across the Study Area.

5.2.3 Multi-Pathway Exposures

The chronic non-carcinogenic and carcinogenic oral/dermal TRVs, as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 5-3. Refer to the toxicological profile for each of the COCs provided in Appendix A of this report for a detailed discussion of the relevant background information supporting the selected TRV.

Table 5-4 Summary of Oral TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Oral/Dermal TRVs (µg/kg bw/d)				Carcinogenic Oral/Dermal Slope Factors ((µg/kg bw/d)$^{-1}$)			
	Exposure Limit		Critical Effect	Source	Exposure Limit		Critical Effect	Source
	Type	*Value*			*Type*	*Value*		
Inorganics								
Antimony	RfD	0.4	Longevity, blood glucose, and cholesterol levels (rat)	US EPA IRIS, 1991c	SF	NA	-	-
Arsenic	RfD	0.3	Hyperpigmentation, keratosis, and possible vascular complications (human)	US EPA IRIS, 1993b; ATSDR, 2007b	SF	$1.5x10^{-3}$	Prevalence of skin cancer (human)	US EPA IRIS, 1998c
Barium	RfD	200	Nephropathy (mouse)	US EPA IRIS, 2005b	SF	NA	-	-
Beryllium	RfD	2	Small intestinal lesions (dog)	MOE, 2011a; US EPA IRIS, 1998; WHO, 2001; ATSDR, 2002a; Cal EPA, 2001	SF	NA	-	-
Boron	RfD	200	Decreases in mean fetal weight (rat)	US EPA IRIS, 2004b	SF	NA	-	-
Cadmium	MRL	0.1	Renal damage (human)	ATSDR, 2012a	SF	NA	-	-
Chromium (total)	RfD	1,500	None observed (rat)	US EPA IRIS, 1998e	SF	NA	-	-
Chromium (VI)	MRL	0.9	Diffuse epithelial hyperplasia of the duodenum (mouse)	ATSDR, 2012b	SF	$4.2x10^{-4}$	Benign and malignant stomach tumour (mouse)	Cal EPA, 2011
Cobalt	TDI	1.4	Cardiomyopathy (human)	RIVM, 2001	SF	NA	-	-
Copper	UL	91	Hepatoxicity and gastrointestinal effects (human)	Health Canada, 2010	SF	NA	-	-
Lead	RfD	0.17	One IQ point decrement in children	Adapted from OEHHA, 2009	SF	NA	-	-
Manganese	RfD	140	CNS effects and impairment of neurobehavioral function (human)	US EPA IRIS, 1996a	SF	NA	-	-
Mercury (inorganic)	RfD	0.3	Nephrotoxicity (rat)	US EPA IRIS, 1995b; adopted by Health Canada, 2010	SF	NA	-	-
Molybdenum	RfD	5	Increased uric acid levels (human)	US EPA IRIS, 1993c	SF	NA	-	-
Nickel	REL	11	Perinatal mortality (rat)	Cal EPA, 2012	SF	NA	-	-
Selenium	RfD	5	Selenosis (human)	US EPA IRIS, 1991d	SF	NA	-	-
Strontium	RfD	600	Rachitic bone (rat)	US EPA IRIS, 1996b	SF	NA	-	-

Table 5-4 Summary of Oral TRVs and Benchmarks Selected for Use in the HHRA

Chemical of Potential Concern	Non-Carcinogenic Oral/Dermal TRVs (µg/kg bw/d)				Carcinogenic Oral/Dermal Slope Factors ((µg/kg bw/d)$^{-1}$)			
	Exposure Limit		Critical Effect	Source	Exposure Limit		Critical Effect	Source
	Type	*Value*			*Type*	*Value*		
Zinc	RfD	300	Decrease in erythrocyte Cu, Zn-super oxide dismutase (ESOD) activity (human)	US EPA IRIS, 2005c	SF	NA	-	-
Volatile Organic Compounds (VOCs)								
Benzene	RfD	4	Decreased lymphocyte cell count (human)	US EPA IRIS, 2003b	SF	$5.5x10^{-5}$	Leukemia (human)	US EPA IRIS, 2000
Polychlorinated biphenyls (PCBs)								
Polychlorinated biphenyls (PCBs, total)	RfD	0.02	Immunological effects (monkey)	US EPA IRIS, 1996c	SF	$2.0x10^{-3}$	Liver hepatocellular adenomas, carcinomas, *etc.* (rat)	US EPA IRIS, 1997
Polychlorinated dibenzo-p-dioxins and Polychlorinated dibenzofurans (PCDD/PCDF)								
Polychlorinated dibenzo-p-dioxins and -furans as Toxic Equivalents (TEQ)	RfD	$7.0x10^{-7}$	Developmental effects; decreased sperm count and motility (human)	US EPA IRIS, 2012b	SF	NA	-	-
Carcinogenic Polycyclic Aromatic Hydrocarbons (PAHs)								
Polycyclic aromatic hydrocarbons (PAHs) as benzo(a)pyrene Toxic Equivalents (TEQ)	RfD	NA	-	-	SF	$7.3x10^{-3}$	Forestomach, squamous cell papillomas and carcinomas (mouse, rat)	US EPA IRIS, 1994

Abbreviations: MRL, minimal risk level; REL, reference exposure level; RfD, reference dose; SF, slope factor; TDI, tolerable daily intake; UL, upper limit intake
NA Not available. No TRV or benchmark is available for this endpoint.

5.3 Chemical Mixtures and Additive Risks

Because chemical exposures rarely occur in isolation, the potential health effects associated with mixtures of COC were considered. The interaction between chemicals can take many forms and as such, Health Canada (2012) recommends that additive interactions be assumed when chemicals (within a given mixture) are structurally similar, act toxicologically through similar mechanisms or affect the same target tissue in the body (*i.e.*, share a common effect).

There are currently no Ontario or Canada reference benchmarks (beyond those chemical groups that have established toxic equivalent factors such as dioxins, furans and polycyclic aromatic hydrocarbons) by which one can evaluate whether exposure to a given mixture from, or in isolation from, multiple sources could pose a health concern. Health effects from mixtures are typically assessed by assuming additive effects of chemicals with similar exposure characteristics (*e.g.*, acute exposure; chronic exposure) and similar toxic effects (*e.g.*, respiratory irritants, nasal irritants, reproductive effects) (Health Canada, 2012). In other words, risk estimates for each chemical in a mixture were summed for illustrative, rather than regulatory compliance purposes.

For the purposes of the current assessment, and consistent with the approach used by Toronto Public Health in the City LAQ reports, mixture risks were calculated by summing the predicted Project Alone risks for the carcinogenic and non-carcinogens COCs, separately. In order to get a sense of the impact of the various scenarios over-and-above the existing risks from the background air quality, the predicted worst case scenario mixture risks for each scenario were also added to the average background mixture risks. The receptor points for this analysis were selected from the worst case scenario close to the facility or along the transportation route.

5.3.1 Toxicity Equivalence Factors for Carcinogenic PAHs

As indicated in Health Canada (2012), as well as most other regulatory guidance, the assessment of risks related to exposures to carcinogenic PAHs is primarily conducted through the use of potency or toxicity equivalence factors (PEF or TEF). TEFs allow large groups of compounds with a common mechanism of action such as PAHs to be assessed when limited data is available for all but one of the compounds (*i.e.*, benzo(a)pyrene). Through this approach, exposures to each of the carcinogenic PAHs are adjusted by their carcinogenic potency relative to benzo(a)pyrene. These potency-adjusted exposures can then be summed to provide an overall exposure to the group of carcinogenic PAHs, based on benzo(a)pyrene as the primary surrogate (*i.e.*, B[a]P-TEQ equivalent).

The primary source of PAHs within the Study Area is from diesel engine emissions. Air dispersion modelling was conducted by the Study Team using the US EPA MOVES model which predicts emissions of both vapour and particulate bound aspects of each specific PAH to produce an estimate of Total PAHs present at a given receptor location. However, as it would greatly over-estimate predicted carcinogenic risk estimates to assume all emitted PAHs were equivalent to benzo(a)pyrene, it is important to adjust the total PAH concentration to account for the relative potency of each of the individual PAHs included in the Total PAH estimate.

The MOVES model uses a preset PAH emission profile to calculate the contribution of each of the individual PAHs emitted for both vapour and particulate phase aspects. By adjusting the relative percentage of each of the individual PAHs by its benzo(a)pyrene-TEF, one can calculate a specific TEQ adjustment factor for that specific PAH. By summing all of the individual TEQ adjustment factors, one can calculate a TEQ adjustment factor for the overall Total PAH group based on the diesel engine PAH emission fingerprint. If one then multiplies the

Total PAH group estimated air concentration for a given receptor by this TEQ group adjustment factor, this will result in an overall estimate PAH concentration that has been adjusted for benzo(a)pyrene potency.

Table 5-5 provides the approach used to calculate the overall TEQ adjustment factor based on the PAH emission profile used by the US EPA MOVES model. TEF Potency values recommended by Health Canada (2012) were selected when available. TEFs recommended by RIVM (2001), Cal EPA (2005), and WHO (2003) were considered in the absence of equivalence factors from Health Canada. In the case of fluoranthene, the selected TEF has been adopted from Kalberlah *et al*. (1995) based on a previous recommendation of the MOECC, as it is more conservative than that presented by Health Canada.

Table 5-5 Approach used to Calculate PAH Potency Adjustment Factor for Predicted Ground-Level Air Concentrations

Carcinogenic PAH COC	PAH-specific Emission Profile (g/VKT) [a]	Percent of Total PAH Emissions	B[a]P-TEF	Potency Adjustment Factor
Acenaphthene	1.1E-04	7.2%	0.001	7.2E-05
Acenaphthylene	1.9E-04	12.1%	0.01	1.2E-03
Anthracene	1.1E-04	7.2%	0.01	7.2E-04
Benzo(a)anthracene	6.4E-05	4.0%	0.1	4.0E-03
Benzo(a)pyrene	1.8E-05	1.1%	1	1.1E-02
Benzo(b)fluoranthene	5.3E-06	0.33%	0.1	3.3E-04
Benzo(ghi)perylene	1.2E-06	0.07%	0.01	7.4E-06
Benzo(k)fluoranthene	7.3E-07	0.05%	0.1	4.6E-05
Chrysene	3.6E-05	2.3%	0.01	2.3E-04
Dibenzo(a,h)anthracene	7.3E-07	0.05%	1	4.5E-04
Fluoranthene	2.3E-04	14.3%	0.001	1.4E-04
Fluorene	2.3E-04	14.5%	0.001	1.4E-04
Indeno(1,2,3 – cd)pyrene	1.3E-06	0.08%	0.1	8.4E-05
Phenanthrene	2.9E-04	18.3%	0.001	1.8E-04
Pyrene	3.0E-04	18.5%	0.001	1.9E-04
Overall Potency Adjustment Factor for B[a]P equivalence				**0.019**

[a] The PAH-specific emission profile, in g/VKT, is a sum of both the vapour and particulate-bound emission rates provided by the US EPA MOVES model.

[b] The individual PAH potency adjustment factor is calculated by multiplying the percent of total PAH emissions represented by the given PAH by its B[a]P-TEF. The overall potency adjustment factor is then calculated by summing these individual potency adjustment factors to given an overall factor that represents 100% of the carcinogenic PAHs present within the Total PAH group.

Based on the individual carcinogenic PAH emissions fingerprint from the US EPA MOVES model, the overall potency adjustment factor for benzo(a)pyrene equivalence (*i.e.*, B[a]P-TEQ) is 0.019. Therefore, each predicted Total PAH air concentration were multiplied by 0.019 for the Transportation alternatives (*i.e.*, Alternative 2a, 2b, 3a, and 3b) to estimate the corresponding B[a]P-TEQ air concentration. As it is expected that the diesel PAH speciation would provide a reasonable fingerprint for background conditions given the dominant source of PAHs in background air concentrations within the Study Area is vehicle traffic (*i.e.*, gasoline and diesel), this approach was also used to correct the local background B[a]P-TEQ concentrations.

Unfortunately, no PAH fingerprint was readily available for Incineration options (*i.e.*, Base Case and Scenario 1). In the absence of this, to avoid grossly overestimating the potential toxicity of the Total PAH group for these scenarios (as was identified in the City's LAQ studies) and in consultation with Toronto Public Health, the aforementioned approach was also used for these two incineration options, with the inherent uncertainty acknowledged.

6.0 RISK CHARACTERIZATION

The final step of a risk assessment is risk characterization which involves the estimation, description, and evaluation of risk associated with exposure to COCs by comparing the estimated exposure to the appropriate reference benchmark or TRV for a specific chemical or group of compounds. Risk characterization involves the comparison of estimated exposures (identified in the exposure assessment) with reference benchmarks or TRVs (identified during the hazard/toxicity assessment) to identify potential human health risks. This comparison is typically expressed as a CR or HQ for non-carcinogenic chemicals and is calculated by dividing the predicted exposure by the reference benchmark/TRV. In the case of direct acting non-threshold carcinogenic chemicals, potential risks are expressed as ILCRs, and represents the incremental risk of an individual within a given population developing cancer over his or her lifetime due to exposures from a specific carcinogenic chemical of concern.

The following sections provide the worst-case short- and long-term human health risk estimates for both Project Alone and Cumulative conditions for each of the five biosolids treatment alternatives and the existing Base Case scenario. Short- and long-term inhalation risk estimates (expressed as CR values) are presented in Sections 6.1 and 6.2, while long-term health risks associated with exposures *via* multiple pathways and environmental media (*i.e.*, soil, dust, home garden produce, *etc.*) are presented in Section 6.3.

As presented in Section 2.1.4.1, CR values were used to evaluate short- and long-term health risks resulting from exposures to COC *via* inhalation. CR values were calculated by dividing the predicted ground-level air concentration (Section 4.1) by the appropriate health-based reference benchmark (Sections 5.1 and 5.2).

In general, a CR value less than or equal to one (CR value ≤1) represents a situation where the predicted ground-level air concentration is less than a corresponding health-based reference benchmark. Considering the various assumptions used that attempt to over predict rather than under predict ground-level air concentrations and the typical uncertainty factors applied during the development of a health-based TRV, a CR value less than or equal to one (CR value ≤ 1) is a strong indicator of negligible health risks resulting from exposure to a particular COC.

A CR value greater than one (CR value > 1) is indicative of a scenario whereby the predicted ground level air concentration is greater than the corresponding health-based reference benchmark, suggesting that the potential for an adverse health effect may be present. The significance of the exceedance must be balanced against the degree of conservatism incorporated in the derivation of the TRVs as well as the predicted ground-level concentrations.

6.1 Short-Term Inhalation Assessment

6.1.1 Project Alone Scenarios

Table 6-1 presents worst-case short-term (*i.e.*, 24-hour) inhalation risk estimates (expressed as CR values) for each of the Project Alone assessment scenarios. The results of the short-term exposure assessment indicate that none of the predicted worst-case ground-level air concentrations emitted from the various biosolids treatment alternatives for the Project Alone scenarios indicated any potential health risk. In fact, most predicted concentrations were many orders of magnitude below their corresponding regulatory health-based benchmark.

Table 6-1 Summary of Predicted Worst-Case Short-Term Health Risks – Project Alone Scenarios

Chemical of Concern	Worst-Case Short-Term Concentration Ratios (CR) – Project Alone Scenarios					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
8-Hour Concentrations						
Criteria Air Contaminants						
Carbon Monoxide (CO)	3.0E-03	5.8E-05	5.9E-05	6.1E-05	6.6E-05	6.7E-05
24-Hour Concentrations						
Criteria Air Contaminants						
Nitrogen Dioxide (NO_2)	3.3E-02	1.0E-03	1.0E-03	1.0E-03	1.4E-03	1.5E-03
Fine Particulate ($PM_{2.5}$)	1.4E-02	1.4E-03	7.2E-04	6.1E-04	8.1E-04	8.3E-04
Respirable Particulate (PM_{10})	9.3E-03	7.4E-04	4.2E-04	3.3E-04	4.4E-04	4.5E-04
Sulphur Dioxide (SO_2)	3.7E-03	1.4E-03	4.4E-06	4.6E-06	6.3E-06	6.4E-06
Inorganics						
Antimony	1.9E-06	1.7E-07	NA	NA	NA	NA
Arsenic	1.3E-03	1.1E-04	1.9E-06	1.3E-06	1.3E-06	1.9E-06
Beryllium	9.9E-05	8.9E-06	NA	NA	NA	NA
Boron	3.5E-07	3.2E-08	NA	NA	NA	NA
Cadmium	6.1E-03	5.6E-04	7.3E-05	7.3E-05	1.1E-04	1.1E-04
Chromium (total)	3.0E-04	2.8E-05	5.6E-06	5.6E-06	8.1E-06	8.1E-06
Chromium (VI)	4.0E-02	3.7E-03	1.4E-03	1.4E-03	2.0E-03	2.0E-03
Cobalt	2.2E-05	2.3E-06	2.4E-06	1.7E-06	1.7E-06	2.4E-06
Manganese	6.6E-05	6.3E-06	1.9E-06	1.9E-06	2.7E-06	2.8E-06
Selenium	3.5E-05	3.2E-06	6.9E-09	4.8E-09	4.8E-09	6.9E-09
Strontium	8.5E-08	7.6E-09	NA	NA	NA	NA
Volatile Organic Chemicals (VOCs)						
Acetaldehyde	6.2E-08	6.2E-08	2.3E-06	1.8E-06	8.1E-07	6.3E-07
Acrolein	9.2E-05	9.2E-05	5.3E-04	4.2E-04	1.8E-04	1.5E-04
Benzene	4.3E-05	1.4E-07	8.7E-06	6.8E-06	3.0E-06	2.4E-06
Dichlorobenzene, 1,4-	4.0E-07	2.5E-08	2.5E-08	2.5E-08	3.7E-08	3.7E-08
Dichloromethane	5.2E-06	NA	NA	NA	NA	NA
Formaldehyde	2.3E-06	2.3E-06	2.3E-06	2.3E-06	3.3E-06	3.3E-06
Tetrachloroethylene	NA	NA	NA	NA	NA	NA
Toluene	1.3E-07	1.7E-09	4.8E-08	3.8E-08	1.7E-08	1.3E-08
Trichloroethylene	NA	NA	NA	NA	NA	NA
Vinyl chloride	7.7E-05	8.0E-08	NA	NA	NA	NA

Note: NA indicates that that either that particular COC is not emitted under the given scenario, or emission factors were unavailable.

As discussed previously, in typical transportation risk assessments, the assessment of 1-hour acute exposures is generally evaluated to ensure potential short-term impacts on local air quality around a given corridor are considered. However, given the nature of the emission sources under consideration in the current assessment (*i.e.*, a biosolids incinerator/pelletizer or a minimal number of trucks travelling on nearby routes), it was agreed in consultation with TPH that it is unlikely that 1-hour exposures would be significant. In transportation air quality assessments, NO_2 is typically the COC of primary concern for acute 1-hour exposure conditions. Therefore, to confirm the assumption of minimal risk, potential inhalation risks were estimated for worst-case 1-hour exposures to NO_2 along the two proposed haul routes.

Table 6-2 provides a summary of predicted worst-case acute 1-hour inhalation health risks arising from exposure to NO_2 emitted from HCTP trucks using the two evaluated haul routes. For the purpose of this confirmation assessment, CR values were predicted based on a comparison of predicted worst-case ground-level air concentrations of NO_2 along the two haul routes to the WHO 1-hour health-based benchmark for NO_2 of 200 µg/m^3 (WHO, 2006a).

Table 6-2 Summary of Predicted Worst-Case Acute 1-hour Project Alone Health Risks from HCTP Truck Haulage Route Use

Chemical of Concern	Worst-Case Acute 1-hour Concentration Ratios (CR)	
	Haulage along HR1	Haulage along HR4
1-Hour Concentrations		
Nitrogen Dioxide (NO_2)	0.010	0.010

Based on the results of this worst-case assessment, the predicted worst-case incremental contribution to short-term 1-hour NO_2 air concentrations emitted from HCTP trucks using either of the two proposed haulage routes was 1% of the health-based acute reference benchmark, and represented a negligible health risk to individuals living, working or playing along the two proposed haul routes.

6.1.2 Cumulative Scenarios

Table 6-3 presents worst-case short-term (*i.e.*, 24-hour) inhalation risk estimates (expressed as CR values) for each of the cumulative (*i.e.*, local background plus the incremental contribution from the given biosolids treatment alternative) assessment scenarios.

In this study, the maximum 24-hour average concentrations represent a “worst-case scenario” because the value at each point (*e.g.*, the most polluted day of the entire year at each location) was combined into a single dataset to represent the neighbourhood (receptor grid locations). Using maximum concentrations in this way is a health-protective approach, since it is unlikely that the maximum concentration for any substance would occur at all locations at the same time. It is also unlikely that the maximum concentration for all substances would occur simultaneously at any of the locations. Instead, the spatial profile of individual and total concentrations would be in constant flux. Therefore, when characterizing the risk associated with 24-hour maximum concentrations for individual substances, the risk should be viewed as the “worst-case scenario” for the community.

It should be noted that the cumulative assessment only evaluated those COCs that are on the City of Toronto’s PAC list, as these were the only COCs for which modelled local background concentrations are available.

Cumulative concentrations are dominated almost entirely by existing local background conditions, with the various proposed biosolids management alternatives providing negligible contributions to the overall worst-case air quality conditions within the Study Area.

The results of the short-term assessment of cumulative exposures indicated that with a few exceptions, none of the predicted worst-case ground-level air concentrations for the cumulative scenarios indicated any potential health risk. Exceedances of the reference benchmarks were noted for 24-hour exposures to particulate matter (both PM_{10} and $PM_{2.5}$) and hexavalent chromium (*i.e.*, chromium VI). As noted previously, air quality studies in Ontario have indicated that approximately 20-25% of the routinely monitored ambient airborne total chromium was in the hexavalent form and the PM_{10} size fractionation study suggested that the majority of the chromium VI was in the inhalable fraction (MOE, 2011b). CEPA states that 3 - 8 % of air concentrations of total chromium could be chromium VI (CEPA, 1994). Estimates from Marshall, Macklin and Monaghan show that 13% of the total chromium air emissions are chromium VI (TPH, 2007). Studies conducted in 1991-1993 concluded that 20% of the routinely monitored chromium in Southwestern Ontario was in the hexavalent form (Bell and Hipfner, 1997). In the current study, chromium VI was conservatively assumed to represent 25% of the total chromium estimated to be present within the airshed.

Under the cumulative scenarios evaluated for both the Base Case and all the proposed Alternatives, background conditions contribute more than 99.99% of all the cumulative emissions within the overall Study Area. In most cases, any contribution from the HCTP would be undetectable compared to existing conditions across the Study Area, regardless of Alternative considered.

Table 6-3 Summary of Predicted Short-Term Health Risks for Exposures at Maximum 24-hour Concentrations at Worst-Case Receptor Grid Location – Cumulative Scenarios

Chemical of Concern	Worst-Case Background	Worst-Case Short-Term Concentration Ratios (CR) – Cumulative Scenarios					
		Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
8-Hour Concentrations							
Criteria Air Contaminants							
Carbon Monoxide (CO)	0.32	0.32	0.32	0.32	0.32	0.32	0.32
24-Hour Concentrations							
Criteria Air Contaminants							
Nitrogen Dioxide (NO_2)	0.18	0.18	0.18	0.18	0.18	0.18	0.18
Fine Particulate ($PM_{2.5}$)	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**	**1.9**
Respirable Particulate (PM_{10})	**2.5**	**2.5**	**2.5**	**2.5**	**2.5**	**2.5**	**2.5**
Sulphur Dioxide (SO_2)	0.47	0.47	0.47	0.47	0.47	0.47	0.47
Inorganics							
Cadmium	0.032	0.032	0.032	0.032	0.032	0.032	0.032
Chromium (total)	0.018	0.018	0.018	0.018	0.018	0.018	0.018
Chromium (VI)	**3.2**	**3.2**	**3.2**	**3.2**	**3.2**	**3.2**	**3.2**
Manganese	0.073	0.073	0.073	0.073	0.073	0.073	0.073
Volatile Organic Chemicals (VOCs)							
Acetaldehyde	0.0026	0.0026	0.0026	0.0026	0.0026	0.0026	0.0026
Acrolein	0.35	0.35	0.35	0.35	0.35	0.35	0.35
Benzene	0.18	0.18	0.18	0.18	0.18	0.18	0.18
Dichlorobenzene, 1,4-	0.0080	0.0080	0.0080	0.0080	0.0080	0.0080	0.0080
Dichloromethane	0.0050	0.0050	0.0050	0.0050	0.0050	0.0050	0.0050
Formaldehyde	0.069	0.069	0.069	0.069	0.069	0.069	0.069
Tetrachloroethylene	0.0054	0.0054	0.0054	0.0054	0.0054	0.0054	0.0054
Toluene	0.0072	0.0072	0.0072	0.0072	0.0072	0.0072	0.0072
Trichloroethylene	0.018	0.018	0.018	0.018	0.018	0.018	0.018
Vinyl chloride	0.0057	0.0057	0.0057	0.0057	0.0057	0.0057	0.0057

Bolded values highlighted in grey are in excess of the acceptable CR of 1.0 (*i.e.*, the predicted cumulative air concentration exceeds the respective reference benchmark for that particular COC.

6.2 Long-Term Inhalation Assessment

The potential for chronic adverse health effects resulting from long-term exposures (*via* inhalation) were evaluated at each of the receptor grid locations throughout the Study Area.

6.2.1 Project Alone Scenarios

Tables 6-4 and 6-5 provide the worst-case long-term CR values and incremental lifetime cancer risks (ILCR) for each of the five biosolids treatment alternatives. The Base Case scenario is presented to help aid in the interpretation of the Project Alone cases for each of the Alternatives.

The results of the chronic assessment indicate that none of the predicted worst-case ground-level air concentrations emitted from the various biosolids treatment alternatives for the Project Alone case indicated any potential health risk from either a carcinogenic or non-carcinogenic point-of-view. Again, most predicted concentrations were many orders of magnitude below their corresponding regulatory health-based benchmark.

As noted in Figures 6-1 and 6-2, various metals such as cadmium, arsenic, and nickel drive predicted non-cancer risks for both the Base Case scenario and the proposed fluidized bed incinerator alternative. While acrolein is the primary non-cancer risk driver for all of the other Alternatives involving off-site haulage (see Figures 6-3 through 6-6). This is not surprising given it is a significant emission of diesel trucks. Figures 6-7 through 6-12 provide the relative incremental lifetime cancer risks for the carcinogenic COCs under the Base Case and various proposed Alternatives. As noted in these figures, cumulative cancer risks are primarily driven by hexavalent chromium, benzene, and cadmium predicted exposures. However, as noted above, none of these predicted concentrations exceeded their health-based benchmarks for any of the proposed Alternatives.

Table 6-4 Summary of Predicted Long-Term Non-Cancer Health Risks for Exposures at Annual Average Concentrations at the Worst-Case Receptor Grid Location[a] – Project Alone Scenarios

Chemical of Concern	Worst-Case Long-Term Non-Cancer Concentration Ratios (CR) – Project Alone					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Criteria Air Contaminants						
Nitrogen Dioxide (NO_2)	6.6E-03	4.7E-04	4.8E-04	5.6E-04	5.3E-04	5.5E-04
Fine Particulate ($PM_{2.5}$)	1.7E-03	1.9E-04	6.6E-04	4.7E-04	2.4E-04	2.4E-04
Respirable Particulate (PM_{10})	9.4E-04	8.2E-05	3.1E-04	2.2E-04	1.1E-04	1.1E-04
Sulphur Dioxide (SO_2)	1.4E-03	5.1E-04	8.4E-06	6.1E-06	4.5E-06	5.0E-06
Inorganics						
Antimony	9.2E-06	8.3E-07	NA	NA	NA	NA
Arsenic	1.0E-03	9.0E-05	2.6E-06	2.0E-06	1.8E-06	2.6E-06
Barium	8.6E-07	8.2E-07	9.2E-07	8.2E-07	8.2E-07	9.2E-07
Beryllium	5.7E-06	5.1E-07	NA	NA	NA	NA
Boron	8.5E-07	7.6E-08	NA	NA	NA	NA
Cadmium	1.5E-03	1.4E-04	4.1E-05	4.1E-05	4.6E-05	4.6E-05
Chromium (total)	4.2E-05	4.1E-06	1.9E-06	1.9E-06	2.1E-06	2.1E-06
Chromium (VI)	1.1E-05	1.1E-06	8.8E-07	8.8E-07	9.9E-07	9.9E-07
Cobalt	9.1E-07	1.6E-07	1.8E-07	1.6E-07	1.6E-07	1.8E-07
Copper	5.3E-05	4.8E-06	1.8E-07	1.6E-07	1.6E-07	1.8E-07
Lead	3.1E-04	2.8E-05	4.4E-07	4.4E-07	6.3E-07	6.3E-07
Manganese	2.1E-05	2.3E-06	1.5E-06	1.7E-06	1.6E-06	1.7E-06
Mercury (inorganic)	8.5E-04	3.9E-04	1.6E-06	1.6E-06	1.8E-06	1.8E-06
Molybdenum	2.8E-06	2.7E-07	7.7E-08	6.8E-08	6.8E-08	7.7E-08
Nickel	1.8E-04	3.3E-05	2.8E-05	3.0E-05	3.1E-05	3.2E-05
Selenium	7.0E-05	6.3E-06	2.5E-08	2.2E-08	2.2E-08	2.5E-08
Strontium	2.0E-07	1.8E-08	NA	NA	NA	NA
Zinc	2.8E-04	2.6E-05	3.0E-06	2.7E-06	2.7E-06	3.0E-06
Volatile Organic Chemicals (VOCs)						
Acetaldehyde	2.1E-08	2.1E-08	2.5E-06	1.7E-06	8.5E-07	6.0E-07
Acrolein	1.7E-04	1.7E-04	3.1E-03	2.2E-03	1.1E-03	7.8E-04
Benzene	1.7E-05	1.3E-07	2.5E-05	1.8E-05	8.6E-06	6.1E-06
Butadiene, 1,3-	4.1E-08	4.1E-08	1.3E-05	9.0E-06	4.4E-06	3.1E-06
Carbon tetrachloride	NA	1.6E-11	NA	NA	NA	NA
Chloroform	NA	1.6E-11	NA	NA	NA	NA
Dichlorobenzene, 1,4-	2.6E-08	3.7E-09	3.7E-09	3.7E-09	4.2E-09	4.2E-09
Dichloroethane, 1,2-	4.0E-09	4.0E-12	NA	NA	NA	NA

Table 6-4 Summary of Predicted Long-Term Non-Cancer Health Risks for Exposures at Annual Average Concentrations at the Worst-Case Receptor Grid Location[a] – Project Alone Scenarios

Chemical of Concern	Worst-Case Long-Term Non-Cancer Concentration Ratios (CR) – Project Alone					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Dichloromethane	1.1E-07	NA	NA	NA	NA	NA
Ethylene dibromide	3.9E-06	4.0E-09	NA	NA	NA	NA
Formaldehyde	1.1E-06	1.1E-06	1.1E-06	1.1E-06	1.6E-06	1.6E-06
Tetrachloroethylene	NA	NA	NA	NA	NA	NA
Toluene	4.1E-09	9.1E-11	1.1E-08	7.6E-09	3.8E-09	2.6E-09
Trichloroethylene	NA	NA	NA	NA	NA	NA
Vinyl chloride	5.1E-08	5.3E-11	NA	NA	NA	NA
Polychlorinated biphenyls (PCBs)						
PCBs (total)	2.8E-06	8.0E-11	NA	NA	NA	NA
Polychlorinated dibenzo-p-dioxins and Polychlorinated dibenzofurans (PCDD/PCDF)						
PCDD/F as Toxic Equivalents (TEQ)	9.7E-07	7.0E-08	NA	NA	NA	NA

Note: NA indicates that that either that particular COC is not emitted under the given scenario, emission factors were unavailable to estimate ground-level air concentrations, or a regulatory TRV was not available for that COC during that averaging period.

[a] The worst-case receptor grid location may vary from COC to COC, depending on scenario and emission profiles.

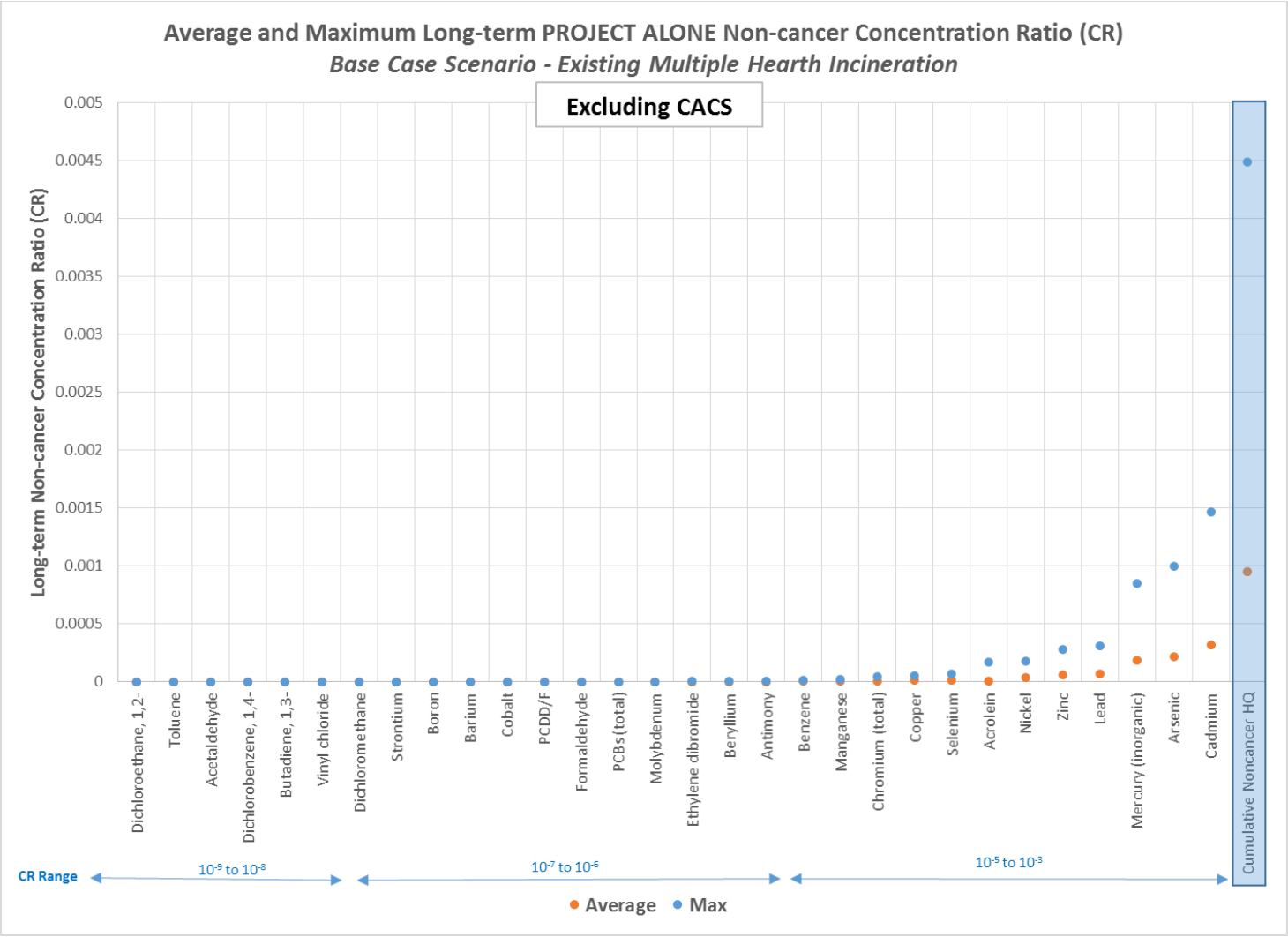


Figure 6-1 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Base Case

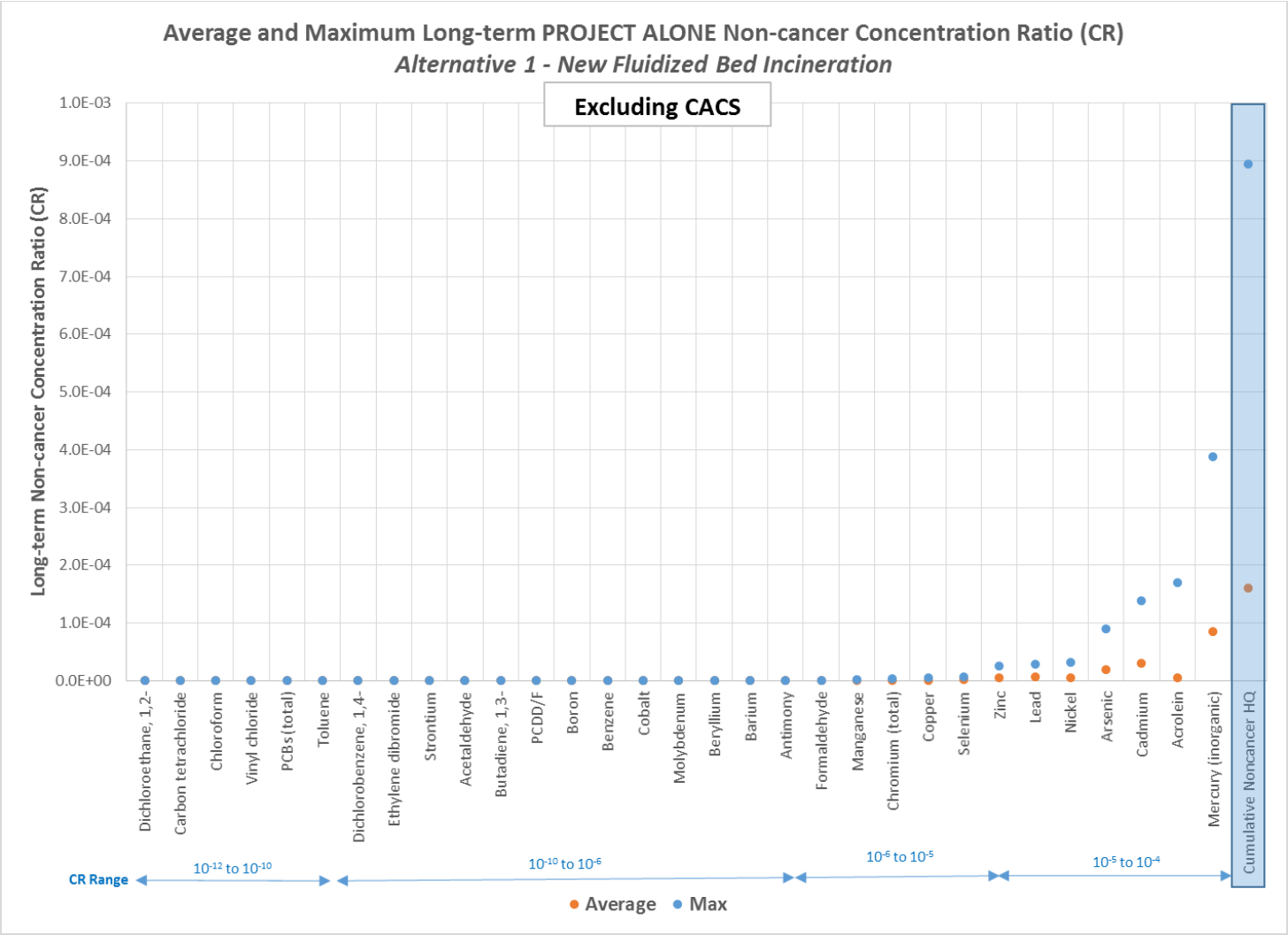


Figure 6-2 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Alternative 1

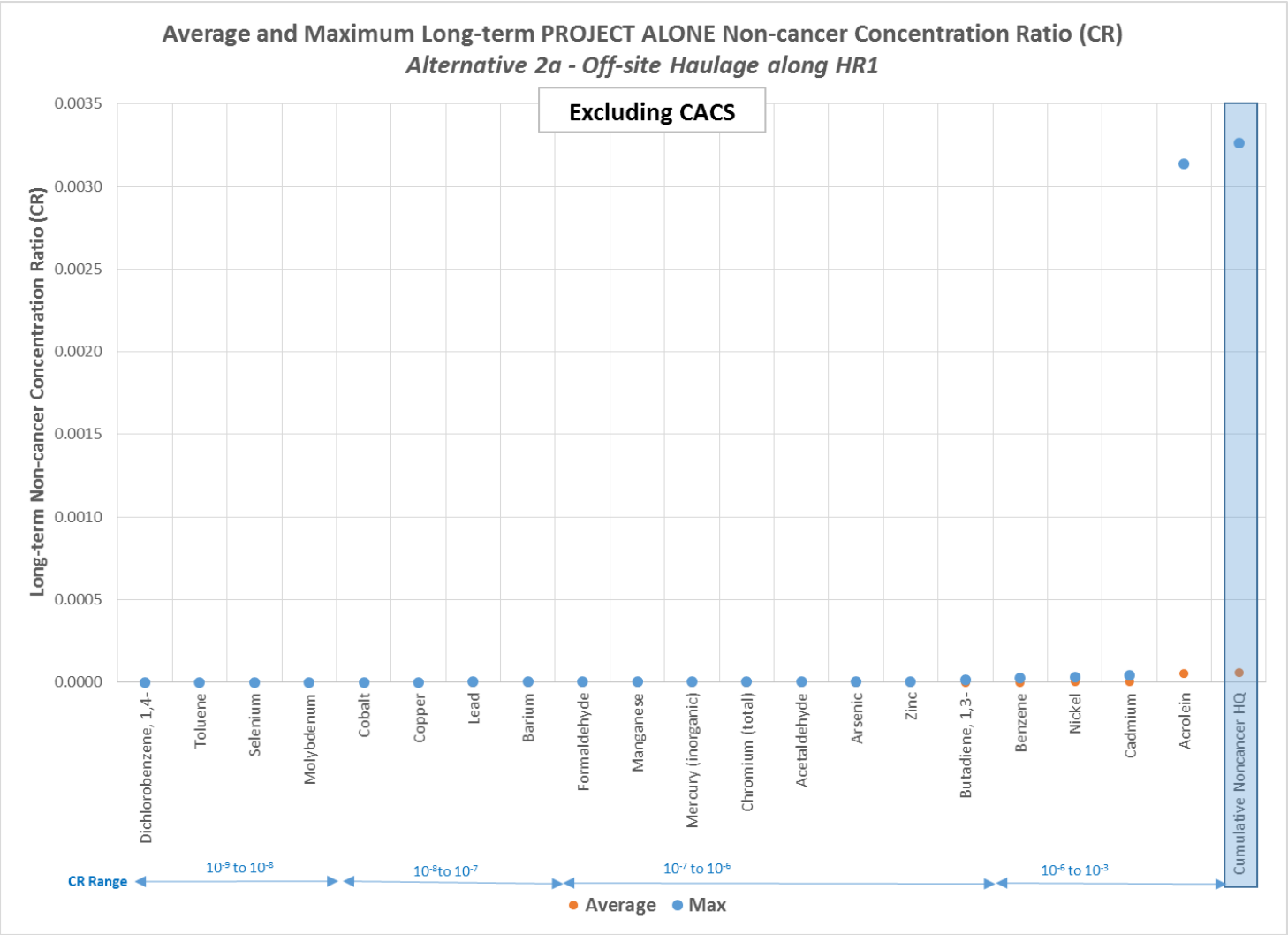


Figure 6-3 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Alternative 2a

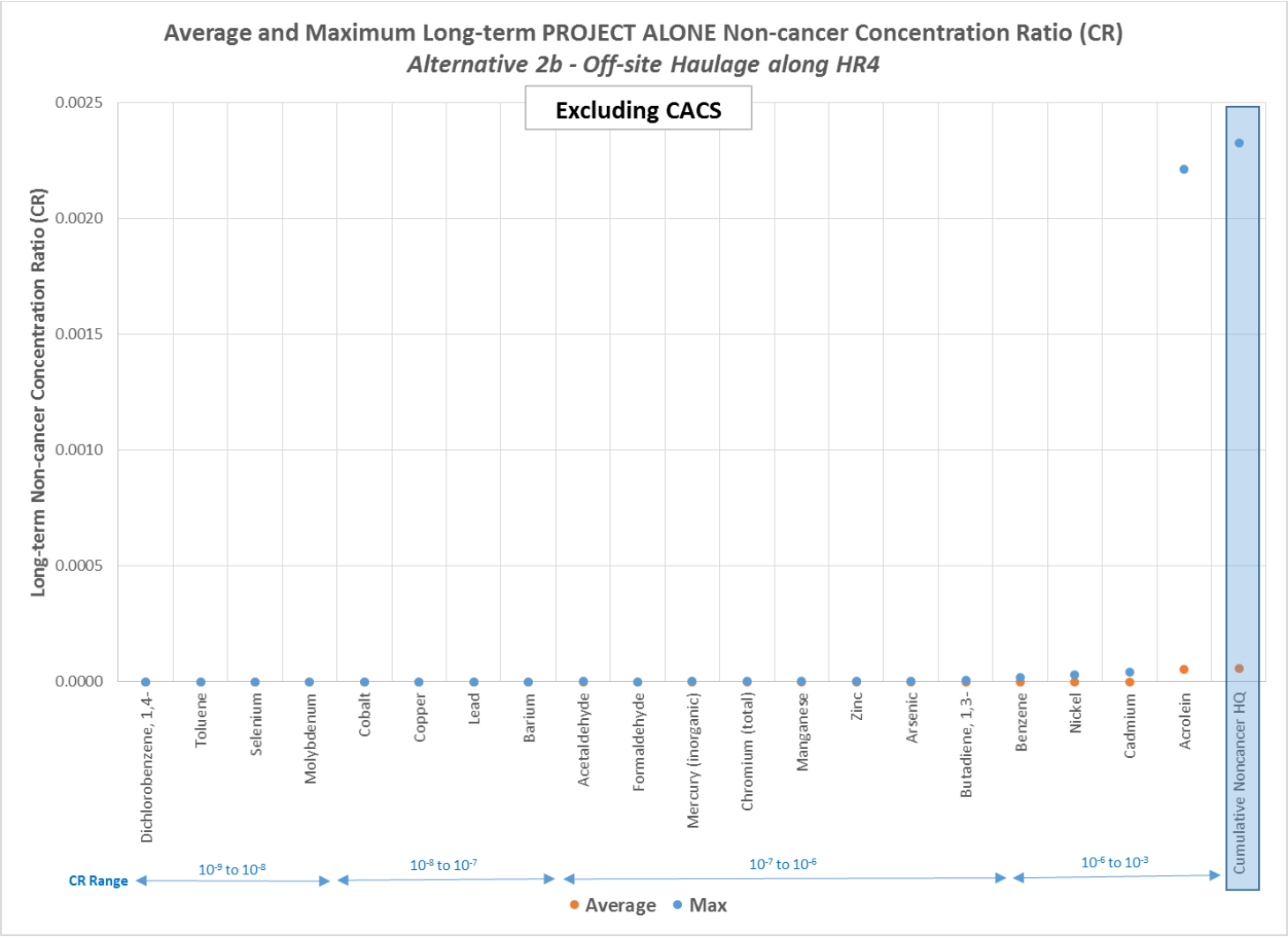


Figure 6-4 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Alternative 2b

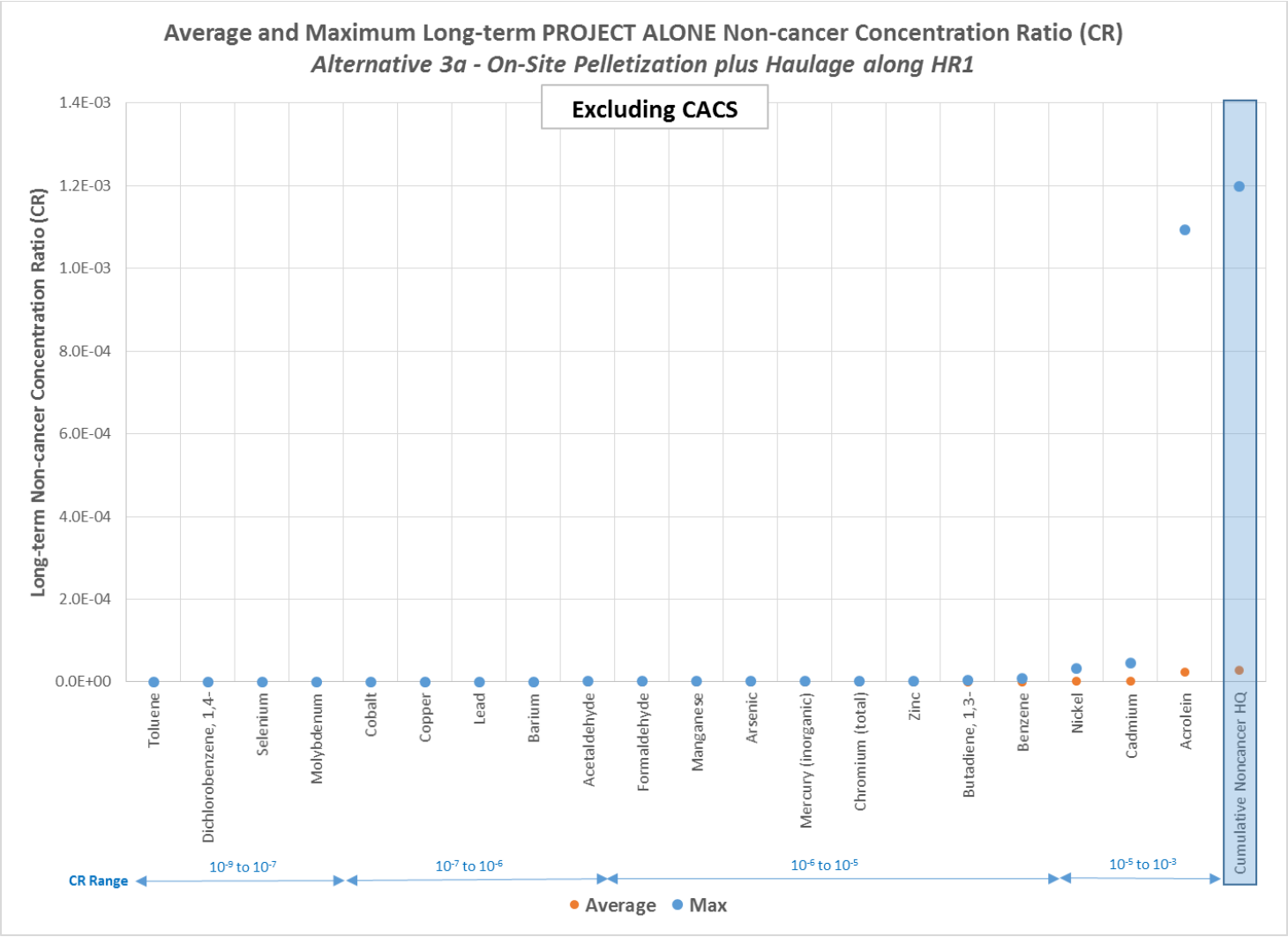


Figure 6-5 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Alternative 3a

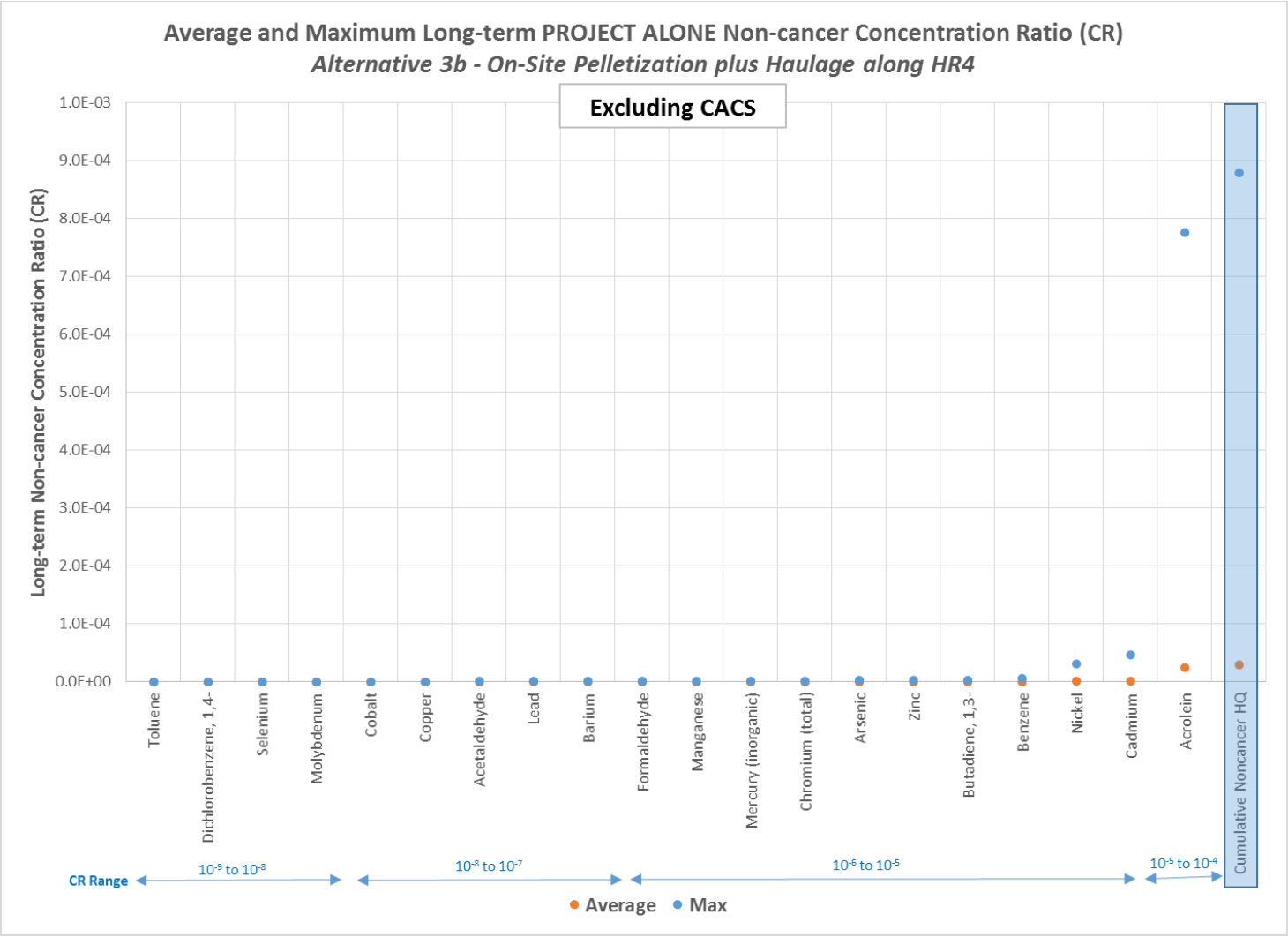


Figure 6-6 COC-Specific Non-Cancer Risks due to Project Alone Emissions from Alternative 3b

Table 6-5 Summary of Predicted Incremental Lifetime Cancer Risks for Exposures at Annual Average Concentrations at the Worst-Case Receptor Grid Location[a] – Project Alone Scenarios

Chemical of Concern	Worst-Case Predicted Incremental Lifetime Cancer Risks (ILCR) – Project Alone Scenarios					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Inorganics						
Arsenic	9.6E-8	8.7E-9	2.5E-10	1.9E-10	1.7E-10	2.5E-10
Beryllium	9.5E-11	8.6E-12	NA	NA	NA	NA
Cadmium	7.2E-8	6.8E-9	2.0E-9	2.0E-9	2.3E-9	2.3E-9
Chromium (total)	6.5E-8	6.4E-9	2.9E-9	2.9E-9	3.2E-9	3.2E-9
Chromium (VI)	8.6E-8	8.4E-9	6.7E-9	6.7E-9	7.6E-9	7.6E-9
Lead	5.6E-10	5.1E-11	7.9E-13	7.9E-13	1.1E-12	1.1E-12
Nickel	6.6E-10	1.2E-10	1.0E-10	1.1E-10	1.1E-10	1.2E-10
Volatile Organic Chemicals (VOCs)						
Acetaldehyde	7.8E-12	7.8E-12	9.3E-10	6.6E-10	3.2E-10	2.3E-10
Benzene	1.5E-9	1.1E-11	2.2E-9	1.5E-9	7.5E-10	5.3E-10
Butadiene, 1,3-	4.1E-14	4.1E-14	1.3E-11	9.0E-12	4.4E-12	3.1E-12
Carbon tetrachloride	NA	9.6E-15	NA	NA	NA	NA
Chloroform	NA	8.5E-15	NA	NA	NA	NA
Dichlorobenzene, 1,4-	1.7E-11	2.4E-12	2.4E-12	2.4E-12	2.8E-12	2.8E-12
Dichloroethane, 1,2-	4.1E-11	4.2E-14	NA	NA	NA	NA
Dichloromethane	4.6E-11	NA	NA	NA	NA	NA
Ethylene dibromide	1.8E-9	1.9E-12	NA	NA	NA	NA
Formaldehyde	6.0E-11	6.0E-11	6.0E-11	6.0E-11	8.5E-11	8.5E-11
Tetrachloroethylene	NA	NA	NA	NA	NA	NA
Trichloroethylene	NA	NA	NA	NA	NA	NA
Vinyl chloride	2.4E-11	2.5E-14	NA	NA	NA	NA
Polychlorinated biphenyls (PCBs)						
PCBs (total)	9.7E-12	2.8E-16	NA	NA	NA	NA
Polycyclic Aromatic Hydrocarbons (PAHs)						
Total PAHs	4.1E-14	8.7E-12	8.4E-12	5.9E-12	2.9E-12	2.0E-12

Note: NA refers to scenarios where the COC in question is either not an emission product or no emission factor could be identified to estimate ground-level air concentrations within the Study Area.

[a] The worst-case receptor grid location may vary from COC to COC, depending on scenario and emission profiles.

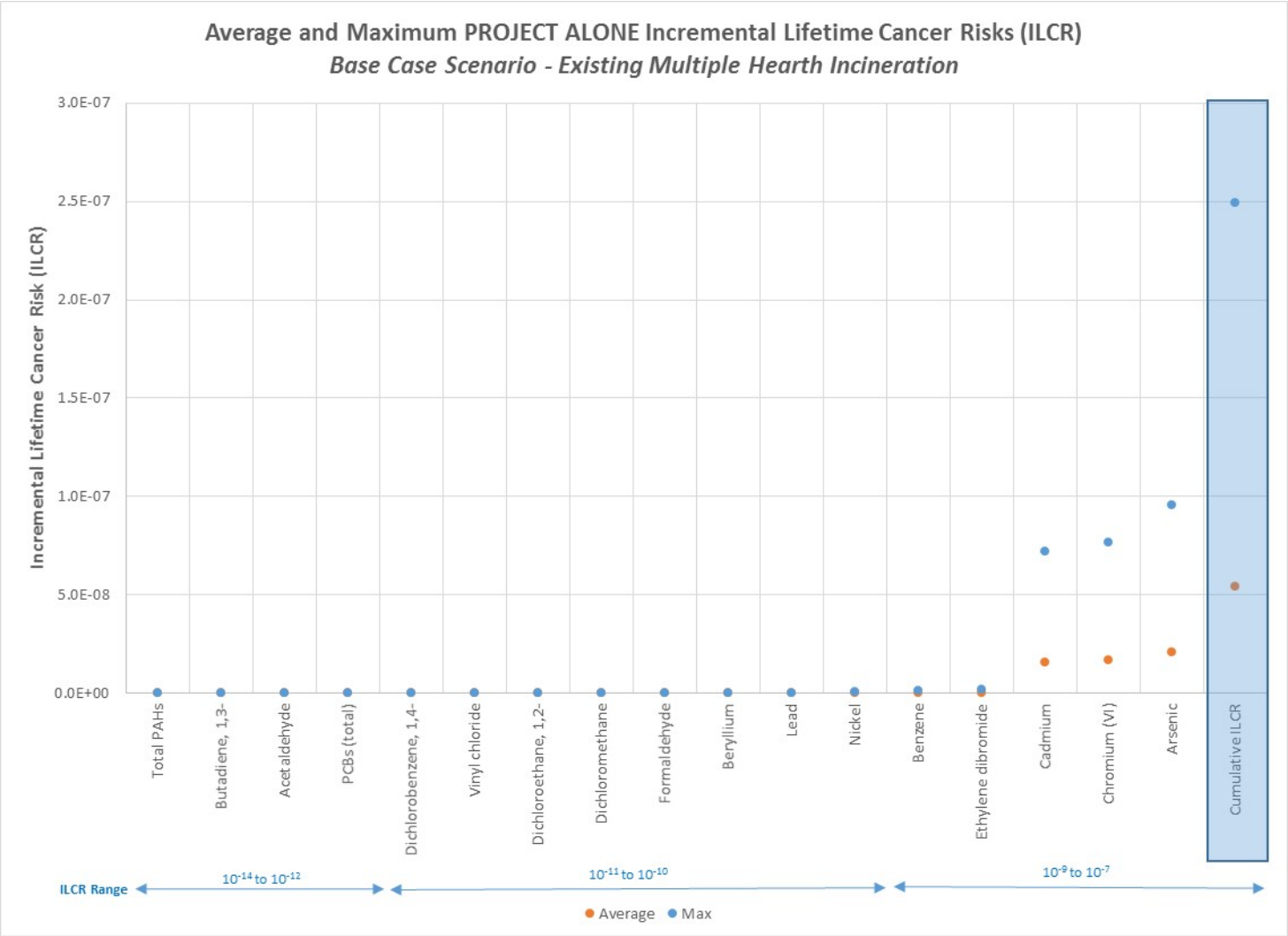


Figure 6-7 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Base Case

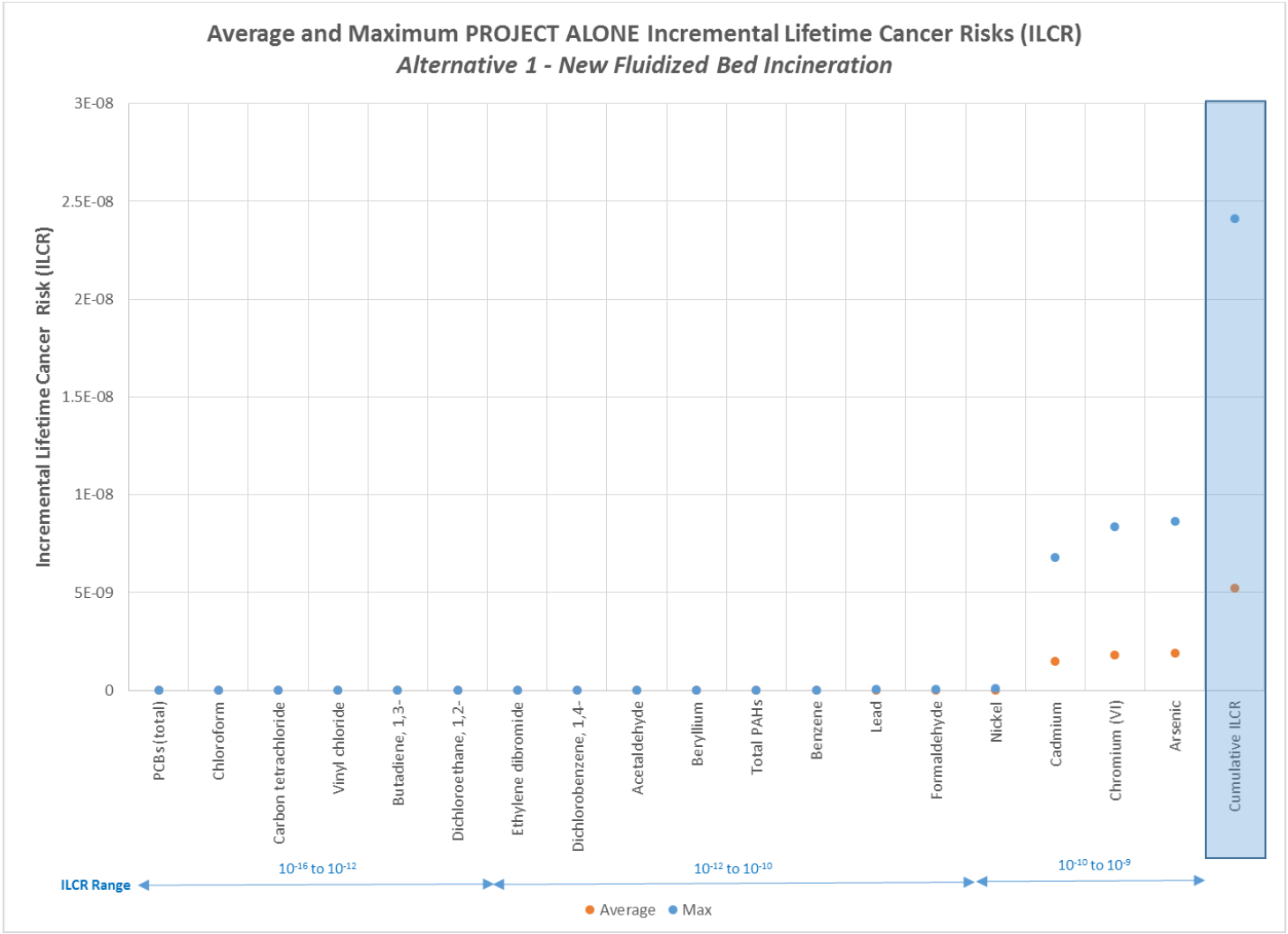


Figure 6-8 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Alternative 1

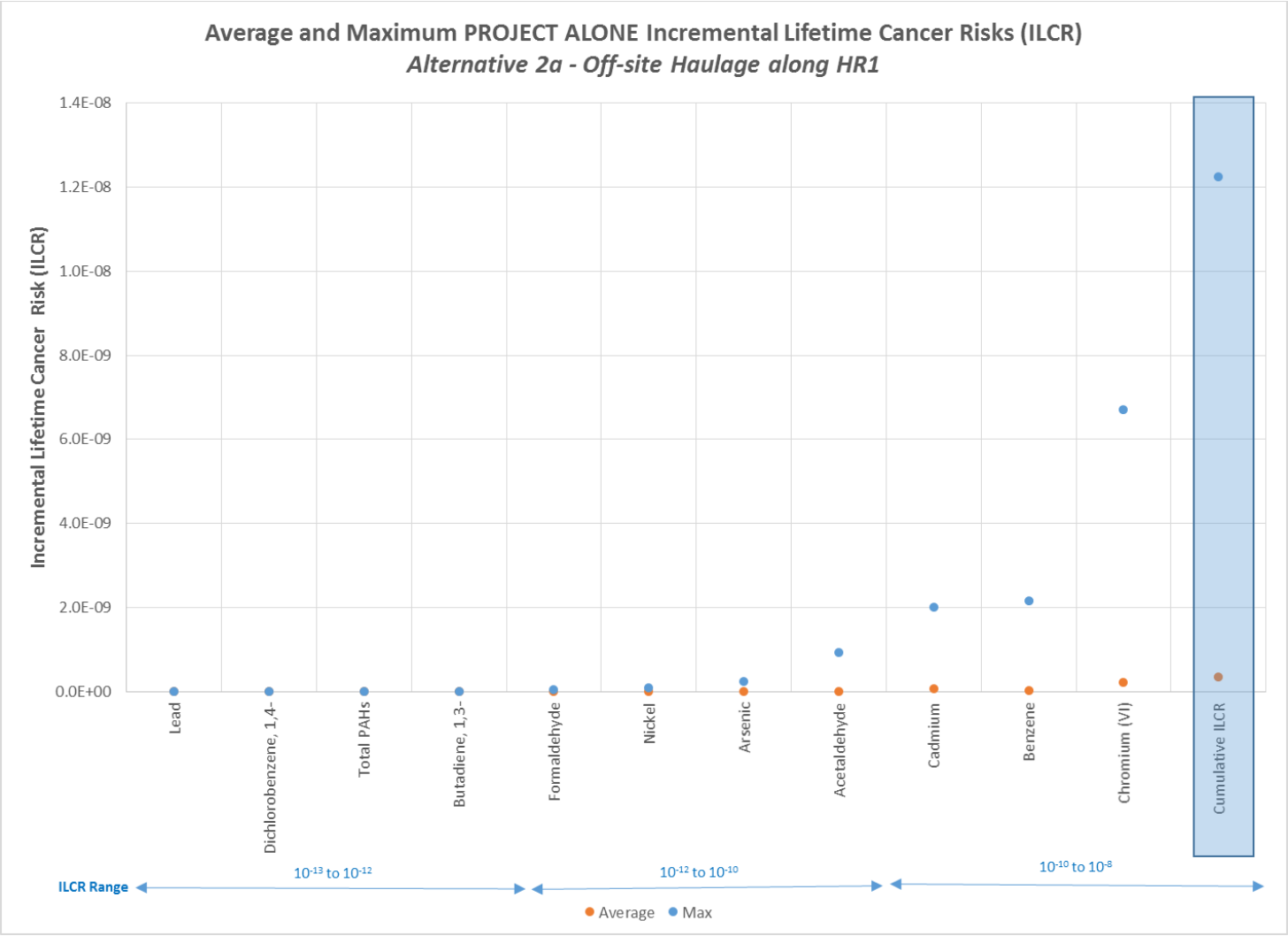


Figure 6-9 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Alternative 2a

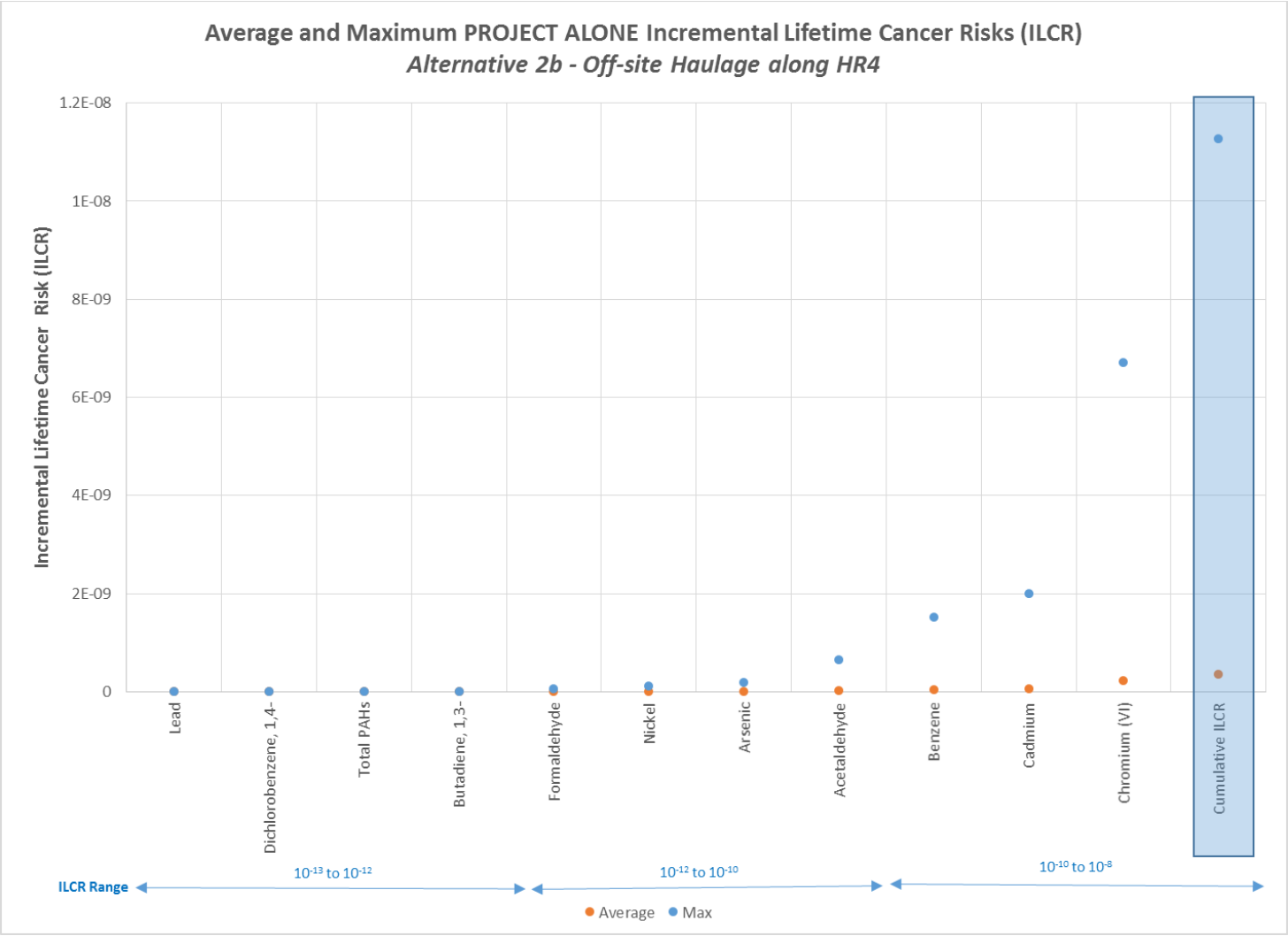


Figure 6-10 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Alternative 2b

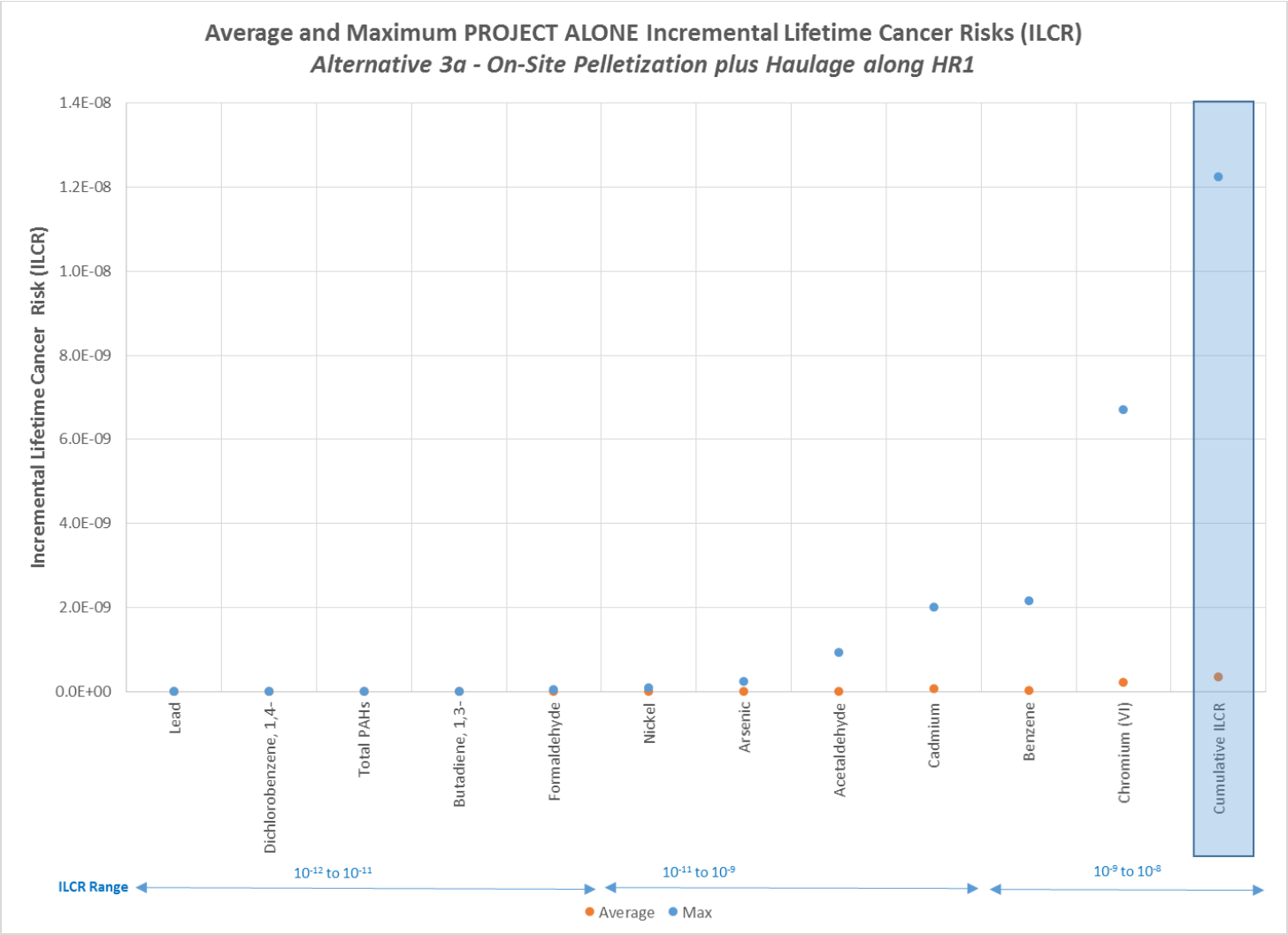


Figure 6-11 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Alternative 3a

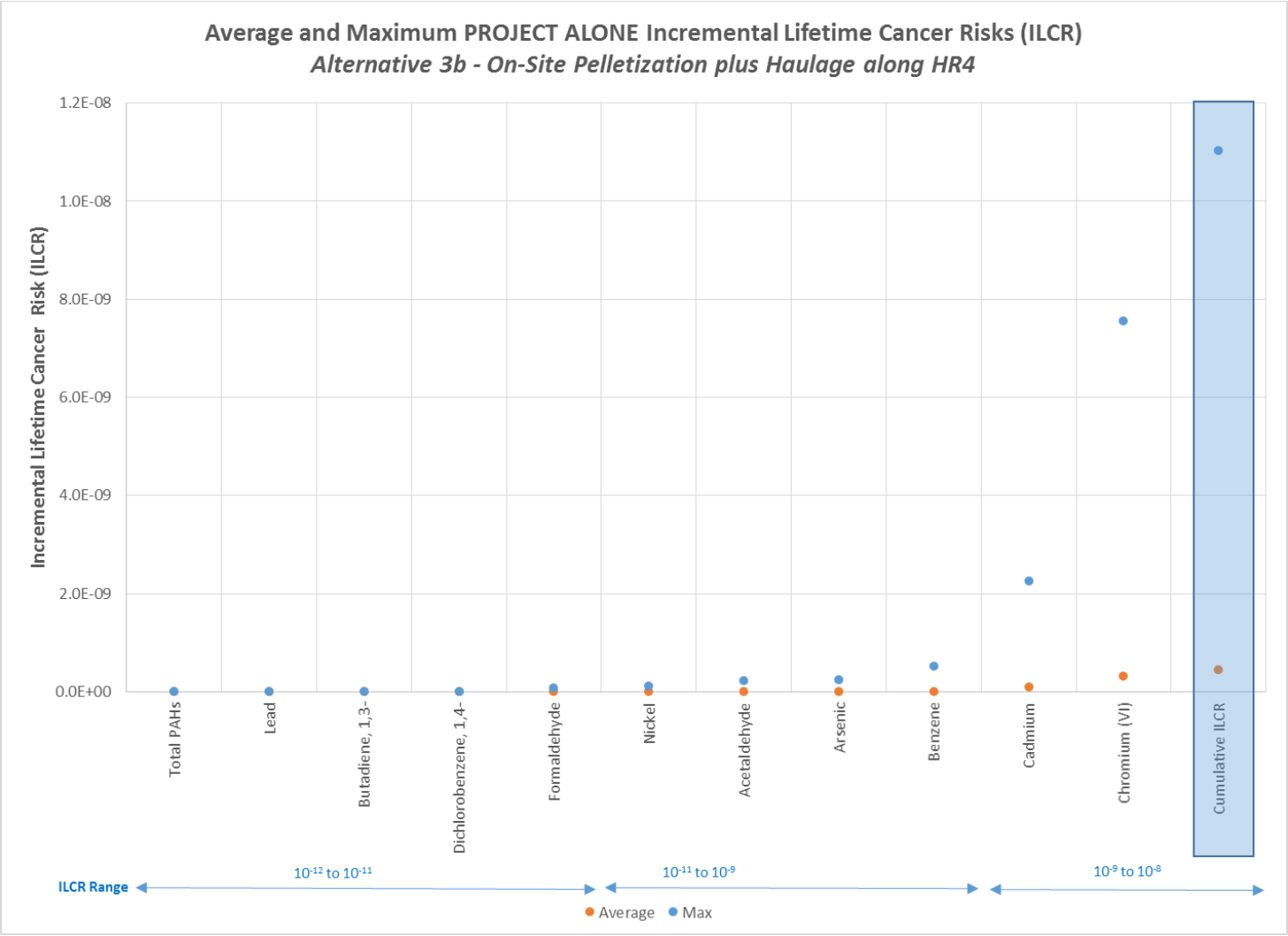


Figure 6-12 COC-Specific Incremental Lifetime Cancer Risks due to Project Alone Emissions from Alternative 3b

6.2.2 Morbidity and Premature Mortality Risks

To properly permit an evaluation of the potential morbidity and mortality outcomes related to an incremental change in air quality using Health Canada's epidemiologically-based CRF values, it is important that the area under consideration has a sufficient population base so as to permit the generalizability of the epidemiological assumptions within the CRF values. In the case of the current assessment, the Study Area is composed of two City of Toronto Wards, which is a sufficient population base to permit the evaluation of morbidity and mortality impacts using the Health Canada methodology. Therefore, it is important to evaluate the impacts of each of the biosolids management alternatives across the entire Study Area, not just the location of the maximum predicted concentrations. To do this, incremental changes in air quality were calculated based on the average contribution to ambient concentrations for each CAC across the entire Study Area.

Table 6-6 provides an overview of the average incremental increase in ambient concentrations of the criteria air contaminants across the entire Study Area for the Base Case and each of the proposed biosolids treatment alternatives.

Table 6-6 Summary of Average Incremental Changes in CAC Concentrations in Study Area above Existing Local Background Air Quality

Morbidity/Mortality Risk Factor	Average Incremental Change in CAC Concentration (µg/m^3) *above Background* across Entire Study Area					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Carbon monoxide	0.060	0.00054	0.0012	0.0011	0.0010	0.0010
Nitrogen dioxide	0.062	0.0012	0.00090	0.00086	0.0010	0.0010
Respirable particulate matter ($PM_{2.5}$)	0.0035	0.00037	0.00015	0.00013	0.00010	0.00010
Sulphur dioxide	0.0094	0.0035	0.0000080	0.0000074	0.0000069	0.0000067

Results of this evaluation indicates that all of the potential biosolids treatment alternatives would result in a small reduction the average CAC concentration across the Study Area compared to the existing base case scenario. As impacts are evaluated over the entire Study Area, none of the alternatives have a significantly different impact fingerprint than each other, or even the existing base case scenario. Note that the average incremental change in ambient ozone concentrations across the entire study area could not be calculate, and as such were not included in the current evaluation. However, given the low concentrations of ozone precursors being emitted by each of the proposed Alternatives, it is unlikely that the proposed Project would result in a significant contribution to ozone formation.

Table 6-7 provides a summary of the predicted incremental percent change above background conditions in various morbidity and premature mortality rates across the Study Area for both the Base Case and various biosolids treatment alternatives. Results of the assessment indicate that each of the proposed biosolids treatment alternatives would result in a negligible improvement in air quality compared to the existing multiple hearth incineration (*i.e.*, a small reduction in the overall risk factor), when evaluated across the entire Study Area.

Table 6-7 Summary of Predicted Average Incremental Percent Change in Morbidity and Mortality Rates across Study Area Compared to Base Case

Morbidity/Mortality Risk Factor	Predicted Average Incremental % Change in Morbidity and Mortality Rates					
	Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Premature mortality risks	0.0050%	0.00036%	0.00014%	0.00013%	0.00011%	0.00011%
Carbon monoxide	0.0000096%	0.000000086%	0.00000020%	0.00000018%	0.00000016%	0.00000016%
Nitrogen dioxide	0.0025%	0.000047%	0.000036%	0.000034%	0.000040%	0.000039%
Respirable particulate matter ($PM_{2.5}$)	0.0024%	0.00025%	0.00010%	0.000091%	0.000070%	0.000067%
Sulphur dioxide	0.00016%	0.000061%	0.00000014%	0.00000013%	0.00000012%	0.00000012%
Cardiovascular Hospital Admissions	0.00025%	0.000026%	0.000010%	0.0000096%	0.0000073%	0.0000070%
Respirable particulate matter ($PM_{2.5}$)	0.00025%	0.000026%	0.000010%	0.0000096%	0.0000073%	0.0000070%
Respiratory Hospital Admissions	0.00027%	0.000028%	0.000011%	0.000010%	0.0000078%	0.0000074%
Respirable particulate matter ($PM_{2.5}$)	0.00027%	0.000028%	0.000011%	0.000010%	0.0000078%	0.0000074%
TOTAL RISK FACTOR	**0.0056%**	**0.00041%**	**0.00016%**	**0.00015%**	**0.00012%**	**0.00012%**

6.2.3 Cumulative Scenarios

Table 6-8 presents worst-case long-term inhalation risk estimates (expressed as CR values) for each of the cumulative (*i.e.*, local background plus the incremental contribution from the given biosolids treatment alternatives) assessment scenarios. It should be noted that the cumulative assessment only evaluated those COCs that are on the City of Toronto's PAC list, as these were the only COCs for which modelled local background concentrations are available.

The results of the assessment of cumulative long-term exposures indicated that again with a few exceptions, none of the predicted worst-case ground-level air concentrations for the cumulative scenarios indicated any potential health risk. Exceedances of the TRVs were noted for annual average exposures to particulate matter (specifically PM_{10}) and acrolein at the worst-case receptor locations. These two COCs are primarily emissions from combustion engines, and this fact is confirmed with the highest concentrations predicted in the receptor grid locations closest to Highway 401 within the Study Area.

Cumulative concentrations are dominated almost entirely by existing local background conditions, with the worst-case exposure scenarios of each proposed biosolids management alternatives providing negligible contributions to the local air quality conditions within the Study Area. Similar to the short-term ambient concentrations, the Base Case contributes more than 99.99% of all the cumulative long-term emissions within the overall Study Area. In most cases, any contribution from the HCTP would be undetectable compared to existing conditions across the Study Area, regardless of Alternative considered.

Furthermore, when one evaluates predicted maximum cumulative ground-level air concentrations at "near field" locations (see Section 4.1) compared to those across the entire Study Area, the maximum concentrations in the area surrounding the HCTP facility are only slightly less than observed throughout the remainder of the Study Area. Given that, even in close proximity the proposed Alternatives still represent a very small to negligible contribution to the cumulative exposure, this likely further demonstrates the far-reaching influence of vehicle emissions from Highway 401 and other major roadways on local air quality within the Study Area.

Figure 6-13 and 6-14 provide a chemical-by-chemical overview of non-cancer and incremental lifetime cancer risks arising based on existing local background air quality, respectively. Non-cancer risks are compared to a regulatory benchmark of 1 (*i.e.*, a CR = 1), while incremental lifetime cancer risks are compared to a regulatory benchmark of 1-in-1,000,000 (*i.e.*, 1×10^{-6}). It should be noted that comparison of background conditions to the 1×10^{-6} ILCR benchmark is highly conservative, as this benchmark is typically used for the evaluation of one Project source to an existing airshed, and not for the evaluation of risks arising from the existing airshed itself (with multitudes of separate contributing sources).

Table 6-8 Summary of Predicted Long-Term Non-Cancer Health Risks for Exposure at Annual Average Concentrations at the Worst-Case Scenario Receptor Location – Cumulative Scenarios

Chemical of Concern	Worst-Case Background	Worst-Case Long-Term Non-Cancer Concentration Ratios (CR) – Cumulative Scenarios					
		Base Case Existing Multiple Hearth Incineration	**Alternative 1** New Fluidized Bed Incineration	**Alternative 2a** Off-site Haulage along HR1	**Alternative 2b** Off-site Haulage along HR4	**Alternative 3a** On-Site Pelletization plus Haulage along HR1	**Alternative 3b** On-Site Pelletization plus Haulage along HR4
Criteria Air Contaminants							
Nitrogen Dioxide (NO_2)	0.20	0.20	0.20	0.20	0.20	0.20	0.20
Fine Particulate ($PM_{2.5}$)	0.90	0.90	0.90	0.90	0.90	0.90	0.90
Respirable Particulate (PM_{10})	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**
Sulphur Dioxide (SO_2)	0.56	0.56	0.56	0.56	0.56	0.56	0.56
Inorganics							
Cadmium	0.031	0.031	0.031	0.031	0.031	0.031	0.031
Chromium (total)	0.0087	0.0087	0.0087	0.0087	0.0087	0.0087	0.0087
Chromium (VI)	0.0030	0.0030	0.0030	0.0030	0.0030	0.0030	0.0030
Lead	0.023	0.023	0.023	0.023	0.023	0.023	0.023
Manganese	0.067	0.067	0.067	0.067	0.067	0.067	0.067
Mercury (inorganic)	0.0049	0.0056	0.0049	0.0049	0.0049	0.0049	0.0049
Nickel	0.10	0.10	0.10	0.10	0.10	0.10	0.10
Volatile Organic Chemicals (VOCs)							
Acetaldehyde	0.0017	0.0017	0.0017	0.0017	0.0017	0.0017	0.0017
Acrolein	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**	**1.3**
Benzene	0.36	0.36	0.36	0.36	0.36	0.36	0.36
Butadiene, 1,3-	0.063	0.063	0.063	0.063	0.063	0.063	0.063
Carbon tetrachloride	9.8E-07	9.8E-07	9.8E-07	9.8E-07	9.8E-07	9.8E-07	9.8E-07
Chloroform	7.8E-05	7.8E-05	7.8E-05	7.8E-05	7.8E-05	7.8E-05	7.8E-05
Dichlorobenzene, 1,4-	0.0027	0.0027	0.0027	0.0027	0.0027	0.0027	0.0027
Dichloromethane	0.00054	0.00054	0.00054	0.00054	0.00054	0.00054	0.00054
Ethylene dibromide	1.9E-05	2.2E-05	1.9E-05	1.9E-05	1.9E-05	1.9E-05	1.9E-05
Formaldehyde	0.074	0.074	0.074	0.074	0.074	0.074	0.074
Tetrachloroethylene	0.010	0.0097	0.0097	0.0097	0.0097	0.0097	0.0097
Toluene	0.0011	0.0011	0.0011	0.0011	0.0011	0.0011	0.0011
Trichloroethylene	0.016	0.016	0.016	0.016	0.016	0.016	0.016
Vinyl chloride	1.4E-05	1.4E-05	1.4E-05	1.4E-05	1.4E-05	1.4E-05	1.4E-05

Bolded values highlighted in grey are in excess of the acceptable CR of 1.0 (*i.e.*, the predicted cumulative air concentration exceeds the respective reference benchmark for that particular COC.

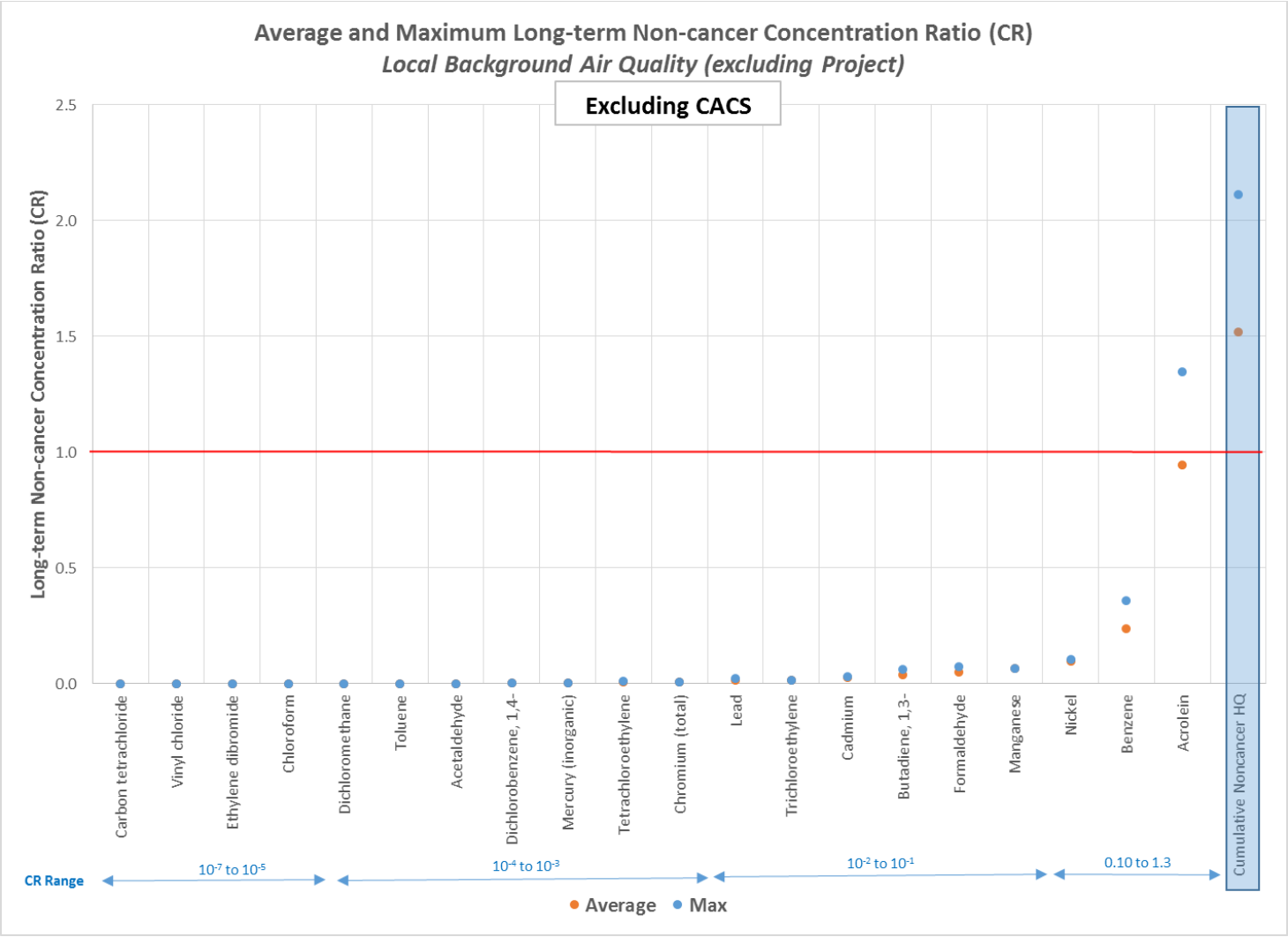


Figure 6-13 COC-Specific Non-Cancer Risks due to Local Air Quality in Study Area

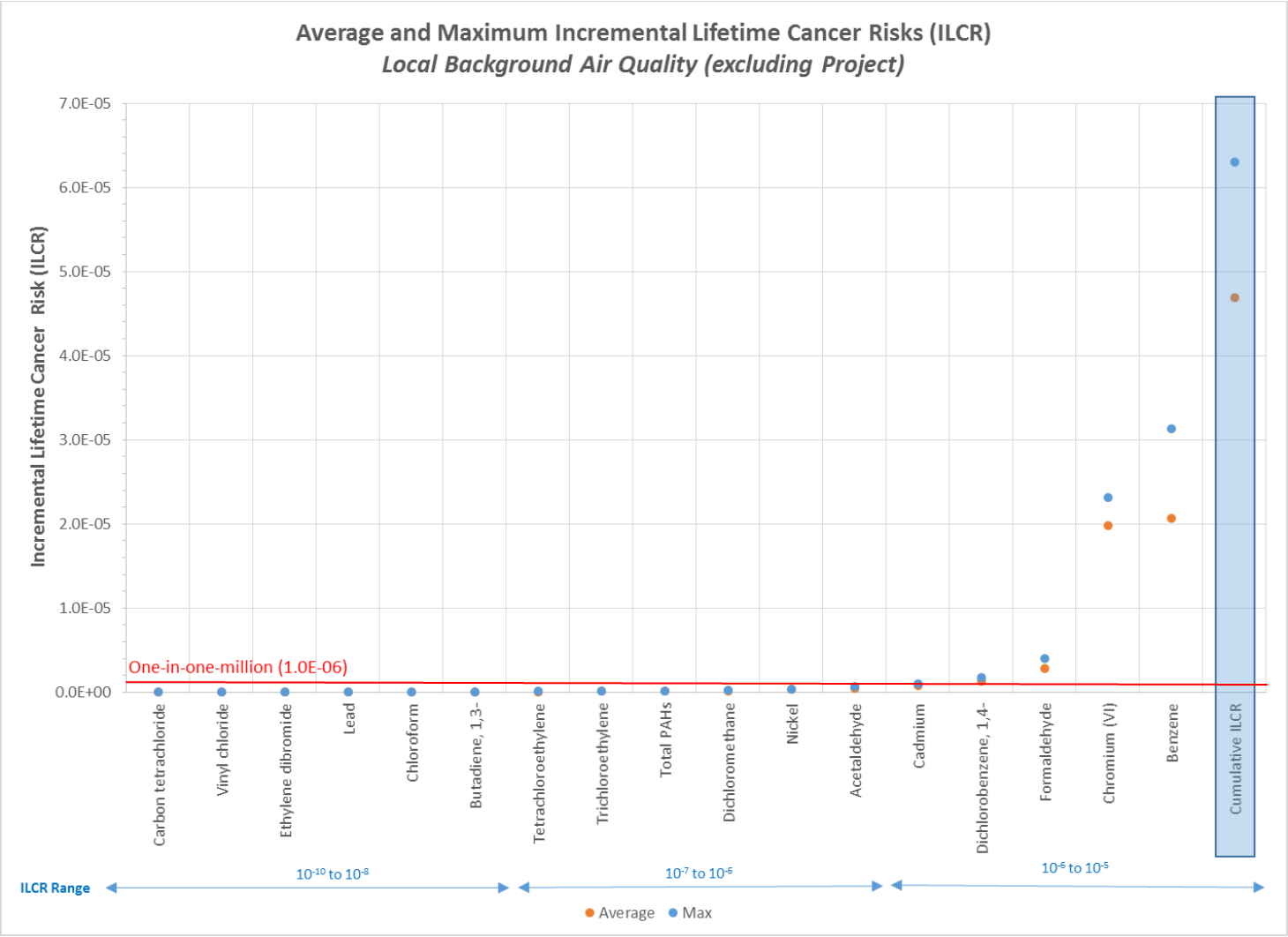


Figure 6-14 COC-Specific Incremental Lifetime Cancer Risks due to Local Air Quality in Study Area

6.3 Multimedia Pathway Assessment

As demonstrated by the multimedia screening approach (Section 3.3.1.2), not all COC identified for evaluation *via* inhalation will persist and/or accumulate in the environment. The multimedia screening approach identified those COC that have the potential to persist and/or accumulate in the environment, therefore, triggering a quantitative multimedia exposure assessment. The multimedia assessment was conducted for the Project Alone scenarios to determine what additional incremental contribution deposition from the various biosolids management alternatives may have on existing soil and home garden quality.

The objective of the multimedia assessment was to predict human health risks resulting from long-term exposures to COC *via* multiple exposure pathways and environmental media. Table 6-9 provides a comparison of non-carcinogenic multimedia risk estimates, as hazard quotients (HQs) for each relevant COC, while Table 6-10 provides a comparison of the multimedia ILCR estimates. To provide an indication in the variability across the entire Study Area, both risk estimates based on both maximum and average deposition rates are provided. Average values are a more appropriate predictor of actual exposures. HQ values were compared to a 0.2 benchmark to account for potential other background sources of each of the COCs, while ILCRs were compared to a one-in-one-million *de minimus* incremental lifetime cancer risk level.

As with the Inhalation Assessment, none of the Alternatives showed predicted risk based on multimedia exposures that exceeded the relevant reference benchmark for either non-carcinogenic or carcinogenic risks. In fact, predicted risks were both orders of magnitude below the corresponding regulatory reference value, as well as significantly less than those predicted for the existing Base Case scenario.

For the Base Case scenario (*i.e.*, the existing multiple hearth incinerators), worst-case maximum deposition of arsenic and lead resulted in predicted risks slightly above the corresponding selected benchmark. In the case of arsenic, worst-case deposition conditions resulted in a predicted incremental lifetime cancer risk of seven times the acceptable one-in-one-million incremental lifetime cancer risk benchmark (*i.e.*, 6.7-in-1,000,000). For lead, worst-case deposition conditions resulted in a non-cancer risk slightly above the 0.2 benchmark (*i.e.*, 20% of the recommended regulatory limit) – 0.46 *versus* 0.20.

However, there is considerable conservatism built into the assumptions underlying the worst-case deposition condition. For example, this scenario assumes that the worst-case emission conditions occurred at the same place, every year for the entire modelled deposition period (*e.g.*, 48 years), and an individual was exposed to that theoretical maximum conditions every day for their entire life. Given the current multiple hearth incinerators have only been in operation for 38 years (not yet 48 years), predicted worst-case soil concentrations would be overestimated for current and historical conditions. Furthermore, the current assessment assumes the worst-case deposition and resulting soil concentration would occur at the same location every day – which is not the case given varying wind directions and other environmental fluctuations on an hourly and daily basis. Based on these and other conservative assumptions (such as those inherent in the development of the toxicological benchmarks used in the assessment, as well as the use of a 20% allocation factor), the scenarios evaluating the average levels of lead and arsenic deposition across the Study Area are a more appropriate predictor of a reasonable worst-case multimedia risk. The results of the current modelling indicate that potential exposures arising from average deposition conditions are many orders of magnitude below the corresponding regulatory benchmark for both arsenic and lead under the Base Case scenario.

Finally, the model predicted soil levels of arsenic and lead are well below rural background levels of arsenic and lead in Ontario. Therefore, given all the inherent conservatism built into the multimedia assessment, it is not anticipated that emissions from the past and current operations of the existing multiple hearth incinerators would result in adverse health impacts to the surrounding community.

Table 6-9 Comparison of Non-Carcinogenic Multimedia Risk Estimates – Project Alone Scenarios

Chemical of Concern	Hazard Quotient[a] (unitless)											
	Base Case Existing Multiple Hearth Incineration		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	Max	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**
Inorganic Parameters												
Antimony	9.2E-03	2.0E-05	8.3E-04	1.8E-06	-	-	-	-	-	-	-	-
Arsenic	8.3E-02	1.8E-04	7.4E-03	1.6E-05	4.4E-06	2.0E-08	4.4E-06	1.9E-08	6.4E-06	2.9E-08	6.4E-06	2.8E-08
Barium	3.9E-06	9.3E-09	3.6E-07	1.5E-09	1.7E-07	7.5E-10	1.7E-07	7.5E-10	2.4E-07	1.1E-09	2.4E-07	1.1E-09
Beryllium	3.3E-05	7.3E-08	3.0E-06	6.6E-09	-	-	-	-	-	-	-	-
Boron	5.5E-04	1.2E-06	5.0E-05	1.1E-07	-	-	-	-	-	-	-	-
Cadmium	1.9E-01	4.2E-04	1.7E-02	3.9E-05	1.1E-04	5.1E-07	1.1E-04	5.1E-07	1.7E-04	7.5E-07	1.7E-04	7.5E-07
Chromium	6.7E-06	1.5E-08	6.1E-07	1.4E-09	6.3E-09	2.8E-11	6.3E-09	2.8E-11	9.2E-09	4.1E-11	9.2E-09	4.1E-11
Cobalt	1.0E-04	2.3E-07	9.4E-06	2.2E-08	3.9E-07	1.8E-09	3.9E-07	1.8E-09	5.7E-07	2.6E-09	5.7E-07	2.6E-09
Copper	2.6E-03	5.8E-06	2.4E-04	5.2E-07	1.7E-07	7.5E-10	1.7E-07	7.5E-10	2.4E-07	1.1E-09	2.4E-07	1.1E-09
Lead	**4.6E-01**	1.0E-03	4.2E-02	9.1E-05	2.0E-05	8.7E-08	2.0E-05	8.8E-08	2.9E-05	1.3E-07	2.9E-05	1.3E-07
Manganese	1.8E-05	3.9E-08	1.6E-06	3.7E-09	2.6E-08	1.2E-10	2.5E-08	1.2E-10	3.7E-08	1.7E-10	3.7E-08	1.7E-10
Mercury	1.1E-01	2.7E-04	5.2E-02	1.2E-04	4.7E-06	2.7E-08	4.7E-06	2.7E-08	6.8E-06	3.9E-08	6.8E-06	3.9E-08
Molybdenum	4.2E-03	9.2E-06	3.8E-04	8.3E-07	2.1E-06	9.6E-09	2.1E-06	9.6E-09	3.1E-06	1.4E-08	3.1E-06	1.4E-08
Nickel	3.9E-04	8.6E-07	3.5E-05	8.3E-08	1.3E-06	6.0E-09	1.3E-06	5.9E-09	1.9E-06	8.6E-09	1.9E-06	8.6E-09
Selenium	5.0E-03	1.1E-05	4.5E-04	9.9E-07	3.4E-08	1.5E-10	3.4E-08	1.5E-10	4.9E-08	2.2E-10	4.9E-08	2.2E-10
Strontium	5.0E-06	1.1E-08	4.5E-07	9.8E-10	-	-	-	-	-	-	-	-
Zinc	1.1E-02	2.5E-05	1.0E-03	2.3E-06	2.4E-06	1.1E-08	2.4E-06	1.1E-08	3.4E-06	1.5E-08	3.4E-06	1.5E-08
Organic Parameters												
Polychlorinated biphenyls (PCBs)	1.5E-02	3.3E-05	4.2E-07	9.5E-10	-	-	-	-	-	-	-	-
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	1.7E-01	3.8E-04	1.2E-02	2.8E-05	-	-	-	-	-	-	-	-

Note: "-" values indicate COCs for which emissions data was unavailable for that particular Scenario.

Bolded values highlighted in grey are in excess of the acceptable HQ of 0.2.

[a] Hazard quotient estimates were based on predicted exposures of the toddler residential receptor.

Table 6-10 Comparison of Carcinogenic Multimedia Risk Estimates – Project Alone Scenarios

Chemical of Concern	Incremental Lifetime Cancer Risk[a] (unitless)											
	Base Case Existing Multiple Hearth Incineration		**Alternative 1** New Fluidized Bed Incineration		**Alternative 2a** Off-site Haulage along HR1		**Alternative 2b** Off-site Haulage along HR4		**Alternative 3a** On-Site Pelletization plus Haulage along HR1		**Alternative 3b** On-Site Pelletization plus Haulage along HR4	
	Max	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**	**Max**	**Average**
Inorganic Parameters												
Arsenic	**6.7E-06**	1.5E-08	6.1E-07	1.3E-09	3.5E-10	1.7E-12	3.5E-10	1.6E-12	5.2E-10	2.3E-12	5.2E-10	2.3E-12
Organic Parameters												
Polychlorinated biphenyls (PCBs)	2.4E-07	5.4E-10	7.0E-12	1.6E-14	-	-	-	-	-	-	-	-
Polycyclic aromatic hydrocarbons (PAHs)	1.5E-09	3.7E-12	3.3E-07	7.8E-10	2.1E-10	3.4E-12	1.5E-10	4.9E-12	7.6E-11	1.2E-12	5.8E-11	1.7E-12

Note: "-" values indicate COCs for which emissions data was unavailable for that particular Scenario.

Bolded values highlighted in grey are in excess of the acceptable ILCR of one-in-one million ($1x10^{-6}$).

[a] Incremental lifetime cancer risk estimates were based on predicted exposures of the composite (or lifetime) residential receptor.

For information purposes, Table 6-11 provides an overview of the percent decrease in non-carcinogenic multimedia risk estimates (such as provided in Table 6-9) when one compares Alternative 1 (*i.e.*, the proposed fluidized bed incineration option) to the Base Case scenario (*i.e.*, the existing multiple hearth incinerators).

Table 6-11 Comparison of Alternative 1 to Base Case Scenario Expressed as a Percentage Decrease in Non-Carcinogenic Multimedia Risk Estimates – Project Alone Scenarios

Chemical of Concern	Percent Decrease in Hazard Quotient between Alternative 1 and the Existing Base Case [a]	
	Alternative 1 New Fluidized Bed Incineration	
	Max	Average
Inorganic Parameters		
Antimony	91.0 %	91.0 %
Arsenic	91.1 %	91.1 %
Barium	90.8 %	83.9 %
Beryllium	90.9 %	91.0 %
Boron	90.9 %	90.8 %
Cadmium	91.1 %	90.7 %
Chromium	90.9 %	90.7 %
Cobalt	90.6 %	90.4 %
Copper	90.8 %	91.0 %
Lead	90.9 %	90.9 %
Manganese	91.1 %	90.5 %
Mercury	52.7 %	55.6 %
Molybdenum	91.0 %	91.0 %
Nickel	91.0 %	90.3 %
Selenium	91.0 %	91.0 %
Strontium	91.0 %	91.1 %
Zinc	90.9 %	90.8 %
Organic Parameters		
Polychlorinated biphenyls (PCBs)	100.0 %	100.0 %
Polychlorinated dibenzo-p-dioxins and furans (PCDD/F)	92.9 %	92.6 %

[a] Percent reduction in risk estimates was calculated using the formula [1 - (Alternative / Base Case)] * 100.

As can be observed in the above table, the results of the multimedia assessment show a significant reduction in risk for all COCs should the Base Case incinerators be replaced by new fluidized bed incinerators. Furthermore, the reduction in potential risk for Alternatives 2 and 3 are even greater, as can be observed in Tables 6-9 and 6-10.

As such, the current assessment concludes that replacement of the existing Base Case incinerators with any of the proposed Alternatives would result in significant reductions in potential health risks arising from deposition of emitted COCs onto soils and home gardens within the Study Area.

6.4 Additive Risks for Mixtures

As discussed in Section 5.3, health effects from mixtures are typically assessed by assuming additive effects of chemicals with similar exposure characteristics (*e.g.*, acute exposure; chronic exposure) and similar toxic effects (*e.g.*, respiratory irritants, nasal irritants, reproductive effects, cancer) (Health Canada, 2012). However, there are currently no Ontario or Canadian reference benchmarks by which one could evaluate whether exposure to a given mixture from, or in isolation from, multiple sources could pose a health concern. Therefore, in the current assessment, risk estimates for each chemical in the theoretical mixture were summed to produce a cumulative risk prediction for illustrative purposes.

Table 6-12 provides a summary comparison of worst-case short- and long-term mixture risks, incremental lifetime cancer risks, and predicted increases in morbidity and mortality rates for existing local background conditions, base case, and the various treatment alternative scenarios.

Table 6-12 Comparison of Worst-case Mixture Risks from Annual Average Air Emissions arising from Proposed Biosolids Alternatives

Type of Health Outcome [a]	Existing Local Background Air Quality	PROJECT ALONE INCREMENTAL RISKS					
		Base Case Existing Incinerator	Alternative 1 New Fluidized Bed Incinerator	Alternative 2 Off-site Haulage		Alternative 3 On-Site Pelletization plus Haulage	
				HR1	HR4	HR1	HR4
Short term non-cancer risk	5.1	0.048	0.0046	0.0020	0.0019	0.0023	0.0023
Long term non-cancer risk	2.1	0.0042	0.00090	0.0033	0.0023	0.0012	0.00088
Cancer risk	76 in one million	0.25 in one million	0.024 in one million	0.012 in one million	0.011 in one million	0.011 in one million	0.011 in one million
Respiratory and cardiovascular induced hospitalizations and mortality [b]	NA [c]	0.0056% contribution	0.00041% contribution	0.00016% contribution	0.00015% contribution	0.00012% contribution	0.00012% contribution

[a] To be consistent with the approach used in the City of Toronto's LAQ studies, the contributions of CACs were only included in the morbidity and mortality estimations, and not the short- and long-term non-cancer risk predictions.

[b] The contribution of ozone to premature mortality risks were not included in the current increase calculation as it is difficult to specify the contribution from the specific Alternatives based on the existing data. Given the low concentrations of ozone precursors being emitted by each of the proposed Alternatives, it is unlikely that the proposed Project would result in a significant contribution to ozone formation, and relatedly premature mortality risks.

[c] This is not applicable as the values provided for respiratory and cardiovascular induced hospitalizations and mortality are presented as an increase above existing background conditions. However, if one used the Health Canada Concentration Response Functions to estimate these outcomes based on existing average background concentrations of the CACs, the predicted increase would be approximately 7%.

Figures 6-15 and 6-16 provide an overview of the short-term non-cancer mixture risks, with average local background from the Study Area and without for each evaluated scenario, respectively. Figures 6-17 and 6-18 provide an overview of the long-term non-cancer mixture risks, with average local background from the Study Area and without for each evaluated scenario, respectively. Finally, Figure 6-19 provides an overview of the incremental lifetime cancer mixture risks for each of the evaluated scenarios.

As has been discussed previously, even the worst-case Project mixture risks provide a negligible contribution to the overall average local background conditions within the airshed of the Study Area.

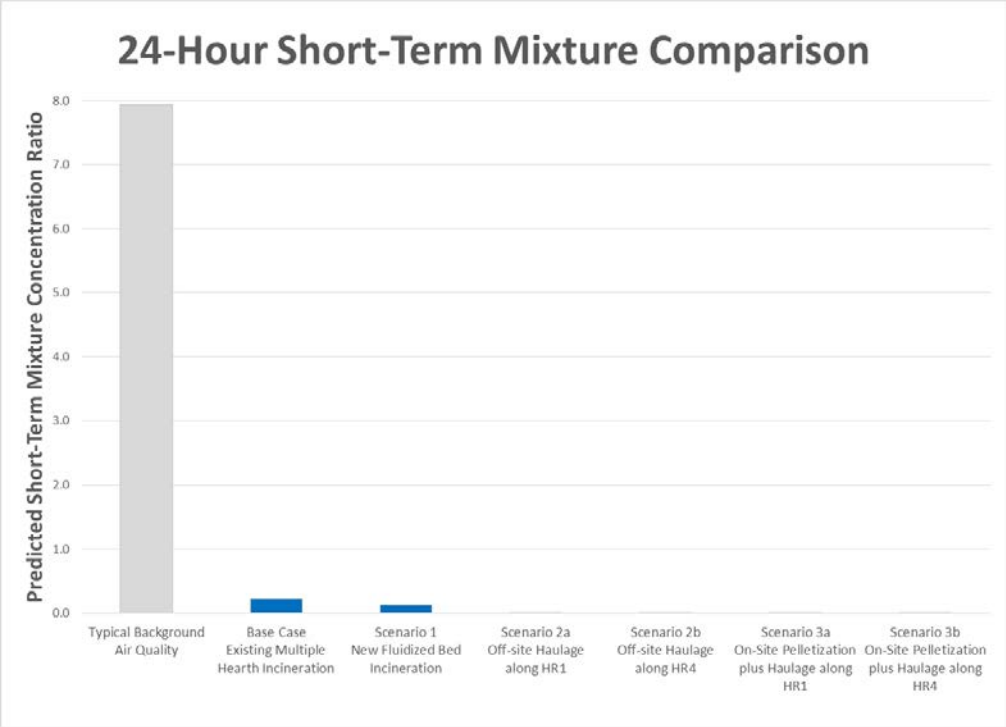


Figure 6-15 Short-Term Non-Cancer Mixture Risks within Study Area including Background

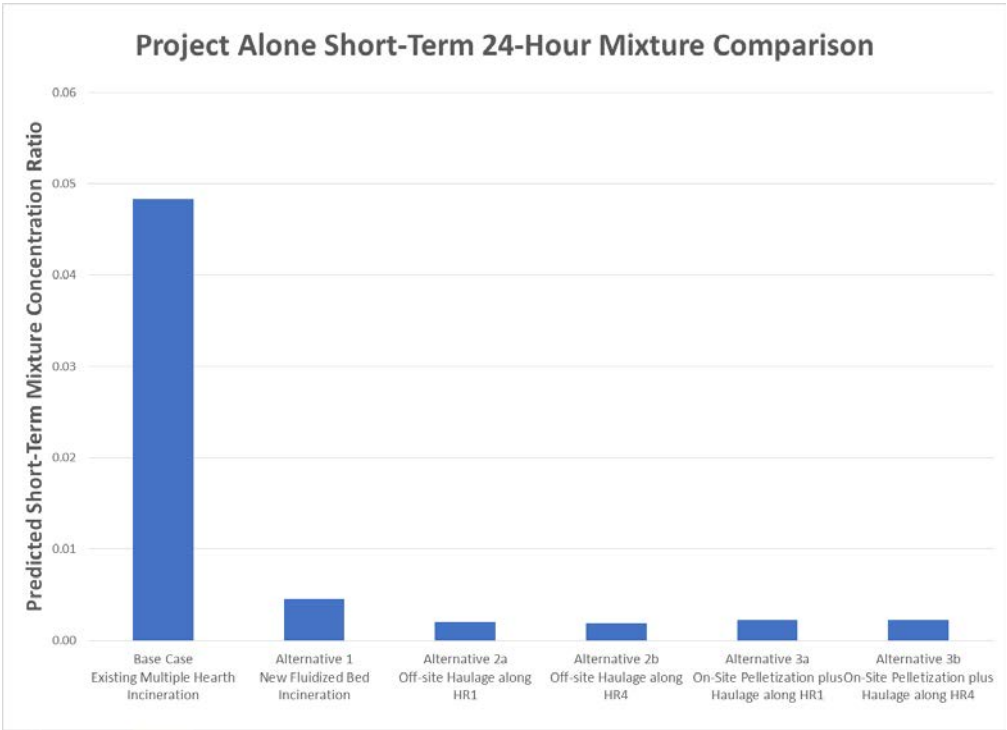


Figure 6-16 Short-Term Non-Cancer Mixture Risks within Study Area excluding Background

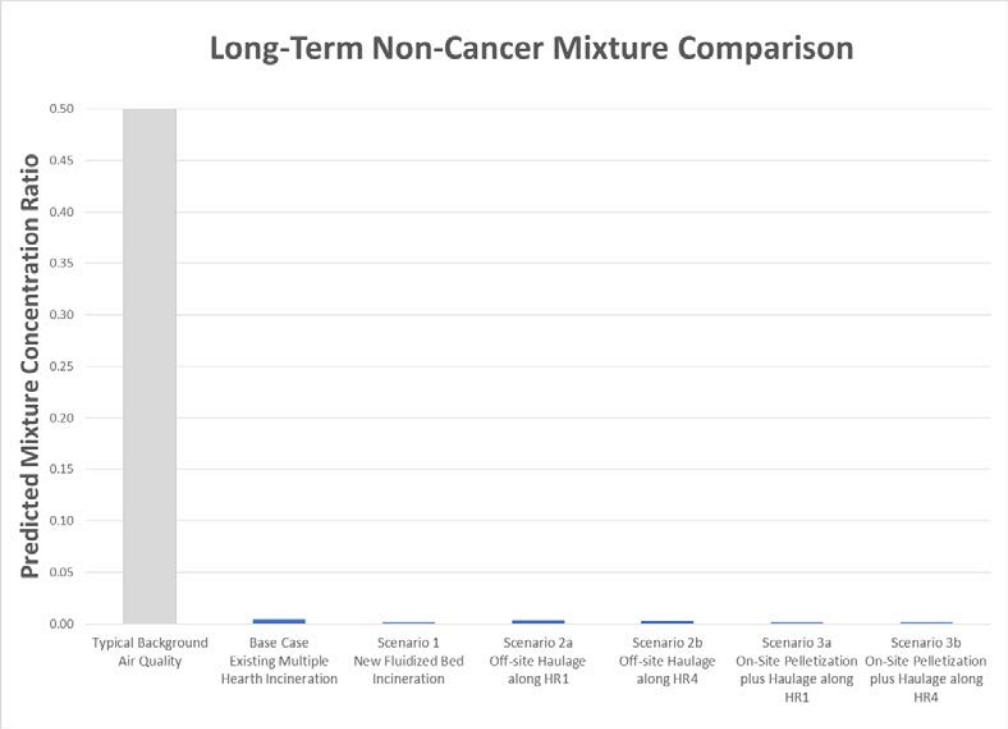


Figure 6-17 Long-Term Non-Cancer Mixture Risks within Study Area including Background

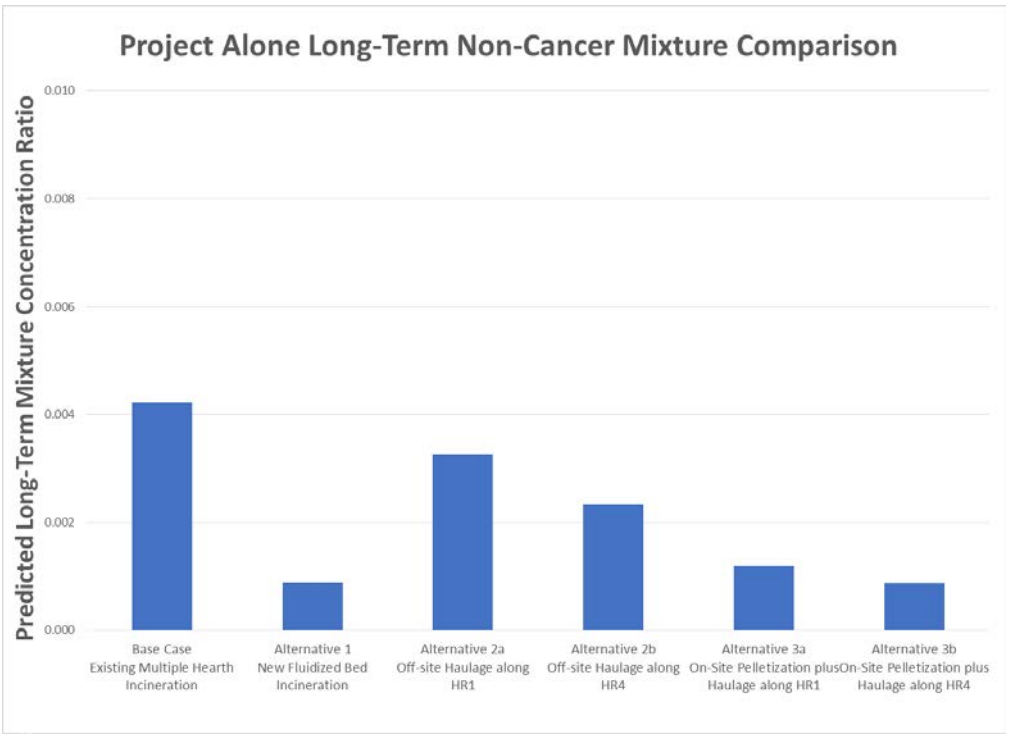


Figure 6-18 Long-Term Non-Cancer Mixture Risks within Study Area excluding Background

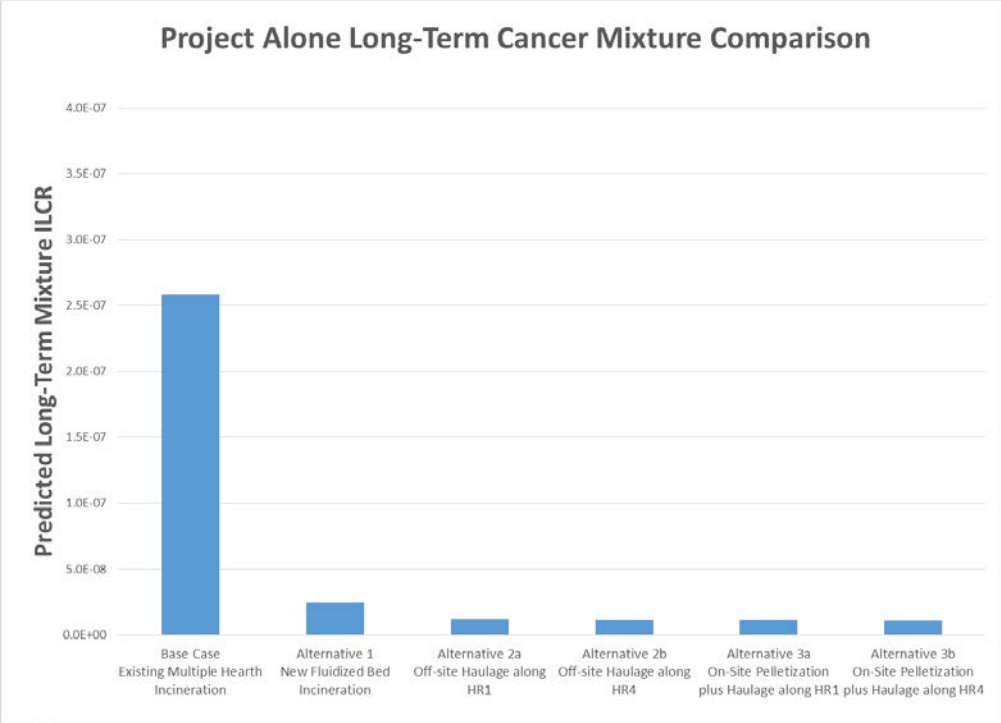


Figure 6-19 Long-Term Incremental Lifetime Cancer Mixture Risks within Study Area

If one drills down into the COC-specific risk from both local background and worst-case Project contribution for each scenario (see Table 6-13), the results confirm that even the worst-case Project Alone contributions for each scenario are negligible compared to existing average background conditions across the Study Area.

The key element to remember is that Table 6-13 presents the relative percentages of the overall (background + project) non-cancer risk predictions, so while the average local background concentrations for each COC is the same for all scenarios, the relative contribution from the Project Alone source changes depending on the scenario under assessment. Furthermore, the overall cumulative risk prediction for each scenario are not the same (*i.e.*, the Base Case scenario shows the highest cumulative mixture risk of all the scenarios).

The results of this analysis indicate that the relative contribution of Project Alone sources is being driven almost entirely by predicted concentrations of acrolein given its inherent toxicity. As such, even a minor variation in Project Alone acrolein concentration between the scenarios can result in a significant impact on the overall percentage represented by that particular project source (*i.e.*, Scenario 2a had the highest predicted acrolein emissions from all scenarios, resulting in the highest predicted project contribution percentage).

Table 6-13 Summary of Relative Percent Contribution to Predicted Non-Cancer Risk from Local Background and Project Sources for each COC by Evaluated Scenario

Chemical of Concern	Base Case Existing Multiple Hearth Incineration		Alternative 1 New Fluidized Bed Incineration		Alternative 2a Off-site Haulage along HR1		Alternative 2b Off-site Haulage along HR4		Alternative 3a On-Site Pelletization plus Haulage along HR1		Alternative 3b On-Site Pelletization plus Haulage along HR4	
	Average Background	Maximum Project Contribution	Average Background	Maximum Project Contribution	Average Background	Maximum Project Contribution	Average Background	Maximum Project Contribution	Average Background	Maximum Project Contribution	Average Background	Maximum Project Contribution
Acrolein	62.1%	0.011%	62.2%	0.011%	62.1%	0.21%	62.1%	0.15%	62.2%	0.072%	62.1%	0.051%
Benzene	15.6%	0.0011%	15.6%	0.0000085%	15.6%	0.0016%	15.6%	0.0012%	15.6%	0.00057%	15.6%	0.0004%
Nickel	6.4%	0.012%	6.4%	0.040%	6.4%	0.0018%	6.4%	0.0020%	6.4%	0.0021%	6.4%	0.002%
Manganese	4.3%	0.0014%	4.3%	0.0025%	4.3%	0.00010%	4.3%	0.00011%	4.3%	0.00011%	4.3%	0.0001%
Formaldehyde	3.4%	0.000072%	3.4%	0.000072%	3.4%	0.000072%	3.4%	0.000072%	3.4%	0.00010%	3.4%	0.000103%
Butadiene, 1,3-	2.6%	0.0000027%	2.6%	0.0000027%	2.6%	0.00084%	2.6%	0.00059%	2.6%	0.00029%	2.6%	0.0002047%
Cadmium	1.7%	0.10%	1.7%	0.0041%	1.7%	0.0027%	1.7%	0.0027%	1.7%	0.0030%	1.7%	0.00%
Trichloroethylene	1.0%	-	1.0%	-	1.0%	-	1.0%	-	1.0%	-	1.0%	-
Lead	1.0%	0.020%	1.0%	0.00019%	1.0%	0.000029%	1.0%	0.000029%	1.0%	0.000041%	1.0%	0.000%
Chromium (total)	0.49%	0.0028%	0.49%	0.0013%	0.49%	0.00012%	0.49%	0.00012%	0.49%	0.00014%	0.49%	0.0001%
Tetrachloroethylene	0.42%	-	0.42%	-	0.42%	-	0.42%	-	0.42%	-	0.42%	-
Mercury (inorganic)	0.32%	0.056%	0.32%	0.025%	0.32%	0.00011%	0.32%	0.00011%	0.32%	0.00012%	0.32%	0.000%
Chromium (VI)	0.17%	0.00066%	0.17%	0.00030%	0.17%	0.000058%	0.17%	0.000058%	0.17%	0.000065%	0.17%	0.00007%
Dichlorobenzene, 1,4-	0.13%	0.0000017%	0.13%	0.00000024%	0.13%	0.00000024%	0.13%	0.00000024%	0.13%	0.00000027%	0.13%	0.0000003%
Acetaldehyde	0.086%	0.0000013%	0.086%	0.0000013%	0.086%	0.00016%	0.086%	0.00011%	0.086%	0.000056%	0.086%	0.0000395%
Toluene	0.053%	0.00000027%	0.053%	0.0000000060%	0.053%	0.00000071%	0.053%	0.00000050%	0.053%	0.00000025%	0.053%	0.00000017%
Dichloromethane	0.028%	0.0000075%	0.028%	-	0.028%	-	0.028%	-	0.028%	-	0.028%	-
Chloroform	0.0044%	-	0.0044%	-	0.0044%	-	0.0044%	-	0.0044%	-	0.0044%	-
Ethylene dibromide	0.0013%	0.00025%	0.0013%	0.00000026%	0.0013%	-	0.0013%	-	0.0013%	-	0.0013%	-
Vinyl chloride	0.00092%	0.0000034%	0.00092%	0.0000000035%	0.00092%	-	0.00092%	-	0.00092%	-	0.00092%	-
Carbon tetrachloride	0.000064%	-	0.000064%	-	0.000064%	-	0.000064%	-	0.000064%	-	0.000064%	-
Total Contribution	**99.8%**	**0.20%**	**99.9%**	**0.085%**	**99.8%**	**0.21%**	**99.8%**	**0.15%**	**99.9%**	**0.078%**	**99.8%**	**0.057%**

Note: "-" entries indicate that the given COC was not emitted by that particular biosolids treatment alternative.

7.0 UNCERTAINTY ANALYSIS

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

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

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

The following sections describe uncertainty within the HHRA, and discuss the potential impacts of these limitations on the conclusions drawn from the assessment. Given the tendency for the assumptions described below to overestimate both exposure and toxicity, it is likely that the risk characterization errs on the side of caution and over predicts risk. A summary of the conservative assumptions that were incorporated into the HHRA can be found in Table 7-1, arranged according to the steps of the risk assessment paradigm. Examination of the table shows that conservatism was introduced at virtually every step of the assessment, and extended to the problem formulation, exposure assessment, and toxicity assessment of the HHRA.

Table 7-1 Major Assumptions Used in the HHRA

Risk Assessment Paradigm	*Assumption*	*Discussion of Impact on Risk Characterization*	*Degree of Impact*
Problem Formulation	Selection of chemicals of potential concern is adequate to characterize potential facility emissions	Chemical selection and identification was based on the City of Toronto's PAC and augmented by those additional COCs evaluated in the HHRAs conducted for the Ashbridges Bay and City of Hamilton biosolids incinerator assessments. Both of these HHRAs employed two discretely different detailed chemical screening processes to select appropriate COCs from their existing stack emission monitoring databases. As such, it is unlikely that potential chemicals of concern emitted from the various proposed biosolids treatment alternatives have not been considered. Consultation on COC selection was also conducted with Toronto Public Health throughout the process.	Neutral
	Emissions of all COCs, except inorganics, for the proposed fluidized bed incineration alternative (*i.e.*, Alternative 1) was based on stack monitoring from the GE Booth Lakeview Waste Water Treatment Facility in Mississauga, Ontario.	As the incineration process does not create inorganics, there is the potential for discrepancies in inorganic emissions predicted between the Base Case and Alternative 1 scenarios due to differences in inorganic content within the waste streams treated at the respective facilities. In other words, the Sewer Use Bylaws in either of the City of Toronto or the Region of Peel/City of Mississauga, and their respective enforcement activities, can have a significant impact on the contaminant loads present within biosolids treated in their municipal biosolids management facilities. The type and effectiveness of their respective air pollution control systems, in particular any baghouse units, can also impact on the relative emissions of inorganics from a given facility. To address the potential inaccuracies in inorganic content due to use of Region of Peel data to represent City of Toronto data, inorganic emissions data from the existing multiple hearth incinerators was used and adjusted by the fractional difference in particulate emissions between the HCTP multiple hearth incinerators and the fluidized bed incinerators at the G.E. Booth facility. This ensures that the increased efficiency of the air pollution control systems used in a modern fluidized bed incinerators are accounted for, while more accurately representing the typical inorganic load present in City of Toronto biosolids. Furthermore, emissions from the proposed fluidized bed incineration option assumes the addition of a mercury scrubber (similar to that present at the G.E. Booth facility) as part of the HCTP air pollution control systems.	Mixed
	Air quality assessment scenarios reflect realistic operating conditions of the proposed biosolids treatment options	Careful consideration was given to the assessment scenarios evaluated in the HHRA, with reasonable worst-case operating conditions assumed for both the air quality assessment and ultimately the HHRA.	Over Predict
	A cumulative assessment could only be conducted on those COCs present on the City's PAC list	Local background concentrations have only been calculated for the COCs on the City's PAC list. Given most of the remaining COCs are not monitored by the MOECC or Environment Canada NAPS stations, there is insufficient information to calculate background concentrations for the non-PAC COCs.	Under Predict
	Potential exposures were evaluated throughout the Study Area.	Care was taken to select locations in the surrounding area that would likely demonstrate the highest potential impacts from the proposed biosolids management alternatives. By employing a grid approach throughout the Study Area, residential receptor locations representing actual nearby geographical locations that currently have, or have the potential to have, occupied by residential dwellings were evaluated in the HHRA.	Neutral

Table 7-1 Major Assumptions Used in the HHRA

Risk Assessment Paradigm	*Assumption*	*Discussion of Impact on Risk Characterization*	*Degree of Impact*
	Biosolids and pelletizers end-of-use were not considered	The negative and positive impacts of beneficial use on agricultural land have been studied and broadly consulted on by the Ministry of the Environment and Climate Change, Ontario Ministry of Agriculture, Food and Rural Affairs, and other regulating agencies outside of Ontario. The findings have been used as a basis to develop provincial health, safety and environmental regulations. Thus, the decision was made to scope end of use out of the current HHRA.	Mixed
Exposure Assessment	Potential 1-hour acute exposures were not evaluated in the current assessment.	Given the nature of the emission sources under consideration (*i.e.*, a biosolids incinerator/pelletizer or a minimal number of trucks travelling on nearby routes), it was agreed in consultation with TPH that it is unlikely that 1-hour exposures would be significant. However, potential worst-case health risks related to 1-hour exposures to NO_2 were assessed to confirm this assumption.	Under Predict
	Maximum 24-hour air concentrations predicted at each of the grid receptor locations were used to evaluate short-term inhalation risks for a subset of COCs.	This assumption is highly improbable and represents a worst-case scenario. The frequency with which the maximum would occur at any one receptor location varies with respect to the COC and the receptor location. Individual exposure to 24-hour maximum ground-level air concentrations requires that a receptor (person) be present at the same time and duration of the maximum predicted air concentration at that particular receptor location each day that the modelled predicted concentration occurs.	Over Predict
	Maximum predicted annual average ground-level air concentrations and chemical-specific deposition rates were used to predict various environmental media concentrations (*e.g.*, soil and garden vegetables) at each receptor grid location assuming that deposition had already occurred for 48 years.	As an added protective measure, the multi-media assessment assumed that maximum chemical-specific annual deposition rates would occur for 48 years prior to exposure, resulting in receptors being exposed to maximum predicted environmental media concentrations.	Over Predict
	Ground-level air concentrations of COCs related to emissions from the various biosolids treatment alternatives were estimated based on mathematical air dispersion models.	The HHRA relied on the results of air dispersion modelling to evaluate the health risks from direct inhalation exposure as well as to predict inhalation health risks. The MOECC has discussed matters of confidence and uncertainty in the predictions of dispersion models with regard to ground level concentrations and deposition rates. This remains the best mechanism to forecast future distributions of emissions in built environments. The air dispersion models used to provide data for the current assessment are approved by the MOECC and the US EPA for use on these types of emission studies. Refer to the Air Quality study for further discussion of the uncertainty inherent in the use of these models.	Mixed
	Background ground-level air concentrations of vinyl chloride may be underestimated	Based on information provided by the Air Quality Study (Technical Memo 5C), background concentrations of vinyl chloride are very likely underestimated due to a lack of city-wide release information. This will likely underestimate the contribution of background sources to the cumulative assessment. However, given the minimal contribution of vinyl chloride from emissions of any of the proposed Alternatives, this is unlikely to have any impact on the conclusions of the HHRA.	Under Predict

Table 7-1 Major Assumptions Used in the HHRA			
Risk Assessment Paradigm	***Assumption***	***Discussion of Impact on Risk Characterization***	***Degree of Impact***
	No site-specific emission factors or measurement data was available to speciate the amount of hexavalent chromium is present within the modelled total chromium concentration.	For incineration scenarios, chromium VI was assumed to represent 19% of total chromium emitted based on the US EPA (2005) National Emissions Inventory Data and Documentation for a sewage sludge incineration facility, while for the remaining scenarios chromium VI was conservatively assumed to represent 34% of chromium emitted from diesel engines based on data presented in the US EPA MOVES model.	Neutral
	Air pollution control systems used to reduce/modify emissions from the incineration option evaluated in Alternative 1 were assumed to be unchanged from those currently used by the comparable G.E. Booth facility.	Air pollution control systems are expected to improve over the lifetime of the facility as technologies improve and the existing facility undergoes retrofit updates.	Over Predict
	Diesel emissions evaluated in the Transportation scenarios were assumed to reflect today's emission standards into the future.	Diesel emissions from trucks used in the Transportation scenarios are likely to improve over the planned lifetime of the facility with improvement in engine and fleet technologies over time.	Over Predict
	Residential receptors were assumed to be present at a given receptor grid location for 24 hours/day, 7 days/week, 52 weeks/year for an entire lifetime.	The multi-media assessment assumed all receptors would never leave the assessed receptor location and, in the case of developing ILCR estimates, live an entire lifetime at this location while being exposed to maximum predicted environmental media concentrations. This assumption likely results in an overprediction of risk.	Over Predict
	Potential exposures *via* breastmilk were not evaluated in the current assessment.	While a small number of potentially bioaccumulative COCs were evaluated (*e.g.*, PCBs, PCDD/Fs), due to the nature of the waste stream, predicted air concentrations of these COCs were very small and considered negligible when compared to the primary source of exposure (*i.e.*, market basket food and seafood). As such, in consultation with TPH, this exposure pathway was screened off from further consideration.	Neutral
	Multi-media assessment conducted for Project Alone Scenarios only	Multimedia assessment could only be carried out for project alone scenario only due to the lack of background data on COCs. The project alone scenario is likely an underpredict risk. However, regulatory bodies account for this underprediction by using an acceptable HR of 0.2. This assumption allows the project only scenario to account for 20% of the overall exposure to the COCs. This is considered to likely overpredict risk.	Mixed
	Particulate deposition has historically occurred with the existing HCTP, and would continue if new fluidized bed incinerators (Alternative 1) were selected as the preferred option going forward.	Assuming there is not significant fluctuations, contributions of COC concentrations arising from particulate deposition has been shown to reach a steady state with surrounding soil concentrations over time. The current multimedia modelling assumes minimal environmental degradation, which likely overestimates potential accumulation over the lifetime of the facility. It should be noted that the MOECC air quality standards (*i.e.*, O. Reg. 419) assume that this continued accumulation of chemicals does not occur.	Over Predict

Table 7-1 Major Assumptions Used in the HHRA			
Risk Assessment Paradigm	***Assumption***	***Discussion of Impact on Risk Characterization***	***Degree of Impact***
	All COC evaluated in the multi-media assessment were assumed to be 100% bioavailable via the oral route.	The magnitude of the toxicological impact of a chemical on a receptor is dependent on the fraction of the ingested quantity of the chemical that is absorbed and subsequently transported to target tissues or organs. Complete absorption of a chemical almost never occurs; some fraction is not absorbed, but is excreted from the body, and is thus not available to exert a toxic effect. For this assessment it was assumed that 100% of all COC concentrations in various environmental media (*e.g.*, soil, food) were 100% available *via* the oral route.	Over Predict
	The HHRA did not evaluate an operation upset scenario where the facility may malfunction or not work as intended.	In the case of the fluidized bed incinerator alternative, start-up and shut-down operations would use natural gas to control the fluidized bed temperature, so there would be no increased risk to emissions. Furthermore, these start-up and shut-down conditions would be infrequent, during scheduled maintenance periods. Because there is continuous monitoring, there would be minimal risk due to air quality parameters as a result of incomplete combustion if there were operational temperature issues, because temperature would be maintained with natural gas supplement. Particulate emissions will also be continuously monitored after the air pollution control system, so any problem detected would result in incinerator shut-down for maintenance. Given there will be two completely independent incinerator trains, each with full capacity, if one is down for maintenance, the other train can operate to continue biosolids treatment. With respect to all of the remaining alternatives, risk of failure (*i.e.*, spills, process performance, etc.) is addressed in the Class EA evaluation but not the HHRA and HIA as a whole. This is because any such event would be very infrequent and due to monitoring, procedures and controls, short-lived so that they would not present a significant health risk.	Neutral
Toxicity Assessment	Toxicity reference values (TRVs) have been developed by regulatory agencies with sufficient conservatism to assure protection of the most sensitive and/or susceptible individuals within the general population (*e.g.*, infants and young children, the elderly, individuals with compromised health). Uncertainty and data gaps are addressed in the derivation of the TRVs through the use of uncertainty factors.	A considerable amount of conservatism is incorporated in the TRVs developed by regulatory agencies. TRVs are deliberately set by regulatory agencies with the protection of the most sensitive individuals in mind. Typically, the TRVs used in the current assessment were derived from the most sensitive health-related endpoints, and then adjusted to account for differences in sensitivity to chemicals among individuals. The use of uncertainty factors (of 10 to 1,000 fold) are directed, in part, toward the protection of sensitive individuals. In most cases, the most conservative TRV was used, unless there was compelling and recent evidence to indicate that a more robust TRV was more appropriate.	Over Predict
	For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions.	The existence of enzymes and biological pathways that routinely repair damage to genetic material (DNA) is well documented in the scientific literature. The potential adverse health outcomes arising from damage to DNA are usually observed only when the ability of these repair enzymes to "fix" the damage is blocked or exceeded. This is a conservative assumption.	Over Predict

Table 7-1 Major Assumptions Used in the HHRA			
Risk Assessment Paradigm	***Assumption***	***Discussion of Impact on Risk Characterization***	***Degree of Impact***
	Humans were assumed to be the most sensitive species with respect to toxic effects of COC.	For obvious reasons, toxicity assays are not generally conducted on humans, so toxicological data from the most sensitive laboratory species were used in the estimation of toxicological criteria for humans, as appropriate. In some cases, however, human-specific data was available and was used in the Toxicity Assessment. Uncertainty and data gaps are addressed in the derivation of the TRVs through the use of uncertainty factors. This is a conservative approach.	Over Predict
	Predicted concentrations of PAHs were adjusted based on a diesel emission PAH fingerprint to estimate a B[a]P-TEQ concentration for the assessment.	This fingerprint was applied for all evaluated scenarios to be consistent. While this is likely reasonable for the transportation based scenarios (*i.e.*, Alternatives 2a, 2b, 3a, and 3b) as well as the local background scenario (as it is dominated by the impacts of vehicle emissions), it is uncertain as to whether the diesel fingerprint is appropriate to characterize the breakdown of PAHs emitted by incinerators (*i.e.*, the Base Case and Alternative 1 scenarios). However, to avoid grossly overestimating potential risks (*i.e.*, by assuming all PAHs are toxicologically equivalent to benzo[a]pyrene), the selected PAH fingerprint as also applied to these scenarios.	Unknown

8.0 OVERALL FINDINGS AND CONCLUSIONS

The primary purpose of this project is to meet the requirements of the Municipal Engineers Association (MEA) Class Environmental Assessment (Class EA) process to identify a preferred approach for managing the biosolids generated at the HCTP. To address concerns with respect to potential human health impacts related to the management of biosolids, each of the potential biosolids management alternatives were evaluated through the use of an HIA framework. A key element of the HIA was a quantitative evaluation of health risks related to potential exposures to chemicals released during the treatment or transportation of biosolids. The quantification of potential chemical health risks was conducted through the use of a human health risk assessment approach.

The primary objective of the HHRA was to determine the potential short- and long-term human health risks to individuals in the surrounding community who may be impacted by emissions from any of the proposed biosolids management alternatives. The HHRA involved an evaluation of the potential health impacts related to inhalation of emissions from each of the proposed alternatives, both project-specific and in the broader cumulative context of the overall airshed (*i.e.*, existing background conditions ***plus*** project-specific contributions). Finally, the assessment also considered the potential impacts emissions may have on soil concentrations throughout the Study Area through long-term deposition, and potential health outcomes that may arise from exposures to impacted soils, dusts, and home garden produce.

The results of the assessment indicate that none of the assessed biosolids management alternatives would result in any unacceptable short- or long-term health risks, either from an inhalation, soil or vegetation exposure routes. Most predicted air concentrations were many orders of magnitude below their corresponding health-based reference benchmark (*i.e.*, typically between 3- and 12-orders of magnitude below). When one focuses in on the criteria air contaminants (*i.e.*, carbon monoxide, nitrogen dioxide, ozone, particulate matter, and sulphur dioxide), all of the proposed biosolids management alternatives resulted in a similar very small improvement in air quality across the Study Area compared to the existing base case scenario. These incremental changes in CAC concentrations were also evaluated for potential impacts on various morbidity and premature mortality rates across the Study Area using the methodology employed by the City in their LAQ reports. Results of this assessment indicate that each of the proposed biosolids treatment alternatives would result in a very small improvement in overall morbidity and mortality rates related to local air quality compared to the existing multiple hearth incinerator.

While the health impacts were negligible for all the proposed alternatives, there were differences in the potential levels of risk attributable to the various alternatives. While the proposed fluidized bed incineration alternative had slightly higher short-term risks than the off-site haulage alternatives, the longer term risks were mixed among the alternatives. Alternative 2 (*i.e.*, off-site haulage alternative) had slightly higher long-term risks, and the fluidized bed incinerator alternative had slightly higher risks from exposures to carcinogenic chemicals, exposure to criteria air contaminants and from multi-media exposures. On balance, all of these risks were orders of magnitude below levels that could potentially result in a health risk to the surrounding community.

When comparing the potential contribution of the various proposed biosolids management alternatives to the overall existing air quality within the Study Area, the assessment showed that the cumulative concentrations were dominated almost entirely by existing local background conditions. The various proposed biosolids management alternatives provided negligible contributions to the overall worst-case air quality conditions which was primarily dominated by

vehicle emissions from Highway 401 and other major roadways within the Study Area. These findings are similar to the conclusions provided in the LAQ assessments conducted by the City in Wards near major transportation routes.

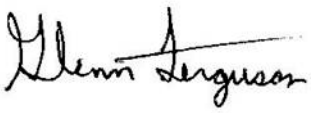
Even when the assessment focused on the local area closely surrounding the HCTP facility (*i.e.*, "near field"), the various alternatives still represented a very small to negligible contribution to the cumulative exposure, despite the further distance to Highway 401 as the dominant air quality impact within the Study Area.

In conclusion, the results of the HHRA indicate that none of the proposed biosolids management alternatives would result in any unacceptable health risks to the surrounding community. Furthermore, none of the project alternatives provide a significant contribution to short- or long-term cumulative concentrations in the Study Area. While each of the proposed options result in a marginal improvement in air quality compared to the existing multiple hearth incinerators, differences between the various proposed options are largely negligible from a health outcome point-of-view.

9.0 DOCUMENT SIGN-OFF

The RA has been performed in accordance with accepted practice and usual standards of thoroughness and competence for the profession of toxicology and environmental RA. The information, opinions and recommendations provided within the aforementioned report have been developed using reasonable and responsible practices, and the report was completed to the best of our knowledge and ability.

Intrinsik Environmental Sciences Inc.



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SCIENCE INTEGRITY KNOWLEDGE

HIGHLAND CREEK TREATMENT PLANT (HCTP) CLASS ENVIRONMENTAL ASSESSMENT

HUMAN HEALTH RISK ASSESSMENT (HHRA) REPORT

APPENDICES

October, 2015

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APPENDIX A

TOXICITY REFERENCE VALUE IDENTIFICATION AND SELECTION

APPENDIX A: TOXICITY REFERENCE VALUE IDENTIFICATION AND SELECTION

A-1.0 INTRODUCTION

All chemicals have the potential to cause toxicological effects; however, it is the chemical concentration, the route of exposure, the duration of exposure, and the inherent toxicity of the chemical that determines the level of effect and hence the potential for unacceptable health risks. The methods and approaches used to determine Toxicity Reference Values (TRVs) for use in the HHRA are outlined in this appendix. Toxicity Reference Values were obtained for each chemical of concern (COC), where available. For the purpose of this assessment, TRVs were defined as values used to describe acceptable doses of chemicals that will not result in the development of unacceptable adverse health effects (e.g., RfD, RfC) or are benchmarks that are policy derived and health based (e.g., AAQC).

When TRVs for a particular COC were available from multiple regulatory agencies, values were reviewed and the professional judgment of an experienced toxicologist and/or risk assessor was used to select the most appropriate TRV. A number of different considerations went into selecting a TRV for use in the HHRA, including:

- Is the TRV derived by a reputable regulatory agency?
- Is there sufficient documentation available concerning the derivation of the TRV (*e.g.,* study, endpoint, point of departure, uncertainty factors applied, *etc.*)?
- How current is the derivation and most recent validation of the TRV?
- How relevant is the TRV in terms of route of exposure and durations of interest?

The TRVs and inhalation benchmarks employed in the current HHRA were obtained from reputable regulatory agencies including, but not limited to:

- Ontario Ministry of the Environment (MOE);
- Health Canada;
- US EPA Integrated Risk Information System (US EPA IRIS);
- Agency for Toxic Substances and Disease Registry (ATSDR);
- Canadian Council of the Ministers of the Environment (CCME);
- World Health Organization (WHO);
- California Environmental Protection Agency (Cal EPA); and,
- Texas Commission on Environmental Quality (TCEQ).

A-2.0 TOXICITY REFERENCE VALUES

Inhalation and oral TRVs were evaluated and selected for all COCs outlined in Appendix C. In addition to providing a tabulated summary of TRVs for each COC, the following sections also provide a brief rationale as to why each TRV was selected for use in the assessment.

A-2.1.1 Acetaldehyde

The 24-hour acute inhalation exposure limit of 500 µg/m^3 proposed by the MOE (2012) and the chronic inhalation exposure limit of 140 µg/m^3 proposed by the Cal EPA (2008) were used for the assessment of acetaldehyde (Table A-1). These exposure limits were chosen as the most conservative values, where available.

The UR of 2.7x10^{-6} (µg/m^3)$^{-1}$ proposed by the Cal EPA (2011) was used for the assessment of acetaldehyde. The UR was selected based on its level of conservatism and considering the date of its most recent validation.

Table A-1 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	500	Tissue damage	Appelman *et al.*, 1986	NOAEL (ADJ): 4.9 x 10^4 µg/m^3	100	MOE, 2012	NA
TC; 24-hour	Acute	2,000	Irritancy in humans	Silverman *et al.*, 1946	NOAEL: 45 mg/m^3 (4.5 x 104 µg/m^3)	20	WHO, 1995	1995
RfC	Chronic	9	Degeneration of olfactory epithelium	Appelman *et al.*, 1982; 1986	NOAEL (HEC): 8.7 mg/m^3 (8.7 x 10^3 µg/m^3)	1,000	US EPA IRIS, 1991a	1991
REL	Chronic	140	Degeneration of olfactory epithelium	Appelman *et al.*, 1982; 1986	NOAEL (ADJ): 24 ppm (4.32 x 10^4 µg/m^3)	300	Cal EPA, 2008	NA
ESL; Annual Average	Chronic	45	Health based	NA	NA	NA	TCEQ, 2014	2012
UR	Chronic	5.8x10^{-7} (µg/m^3)$^{-1}$	Increased incidence of nasal adenocarcinomas and squamous cell carcinomas (combined)	Woutersen *et al.*, 1986	NA	NA	Environment Canada and Health Canada, 2000	2000
UR	Chronic	2.2x10^{-6} (µg/m^3)$^{-1}$	Nasal squamous	Woutersen and	NA	NA	US EPA IRIS, 1991b	1991

Table A-1 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
			cell carcinoma or adenocarcinoma	Appelman, 1984				
UR	Chronic	$2.7x10^{-6}$ $(\mu g/m^3)^{-1}$	Nasal tumour incidence data	Woutersen *et al.*, 1986	NA	NA	Cal EPA, 2011	2002

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.2 Acrolein

The 24-hour acute inhalation exposure limit of 0.4 µg/m^3 proposed by the MOE (2012), and the chronic inhalation exposure limit of 0.02 µg/m^3 proposed by the US EPA IRIS (2003) were used for the assessment of acrolein (Table A-2). These exposure limits were chosen based on its conservatism relative to other values.

Table A-2 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.4	Health based	NA	NA	NA	MOE, 2012	NA
ReV	Chronic	2.7	Mild hyperplasia and lack of recovery of the respiratory epithelium	Dorman *et al*. 2008	$NOAEL_{HEC}$: 0.007 ppm (~15 µg/m^3)	30	TCEQ, 2014	2010
TC	Chronic	0.4	5% increase in non-neoplastic lesions in the nasal respiratory epithelium of rats	Cassee *et al*., 1996	35 µg/m^3	100	Health Canada, 2004	2004
REL	Chronic	0.35	Nasal lesions	Dorman *et al*. 2008	$NOAEL_{HEC}$: 70 µg/m^3	200	Cal EPA, 2008	NA
RfC	Chronic	0.02	Nasal lesions	Feron *et al*. 1978	$LOAEL_{HEC}$: 20 µg/m^3	1,000	US EPA IRIS, 2003	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.3 Antimony

The 24-hour acute inhalation exposure limit of 25 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 0.2 µg/m^3 proposed by the US EPA IRIS (1995) were used for the assessment of antimony (Table A-3). The acute exposure limits were chosen as the only available values. The chronic inhalation exposure limit was chosen based on its level of conservatism and the robustness of the supporting study data.

The chronic oral exposure limit of 0.4 µg/kg/d proposed by US EPA IRIS (1991) was used for the assessment of antimony (Table A-4). The MOE (2001) proposed the same oral exposure limit of 0.4 µg/kg/d. The chronic oral exposure limits were chosen based on its level of conservatism.

Table A-3 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	25	Skin and eye irritation	NA	NA	NA	MOE, 2012	2012
RfC	Chronic	0.2	Pulmonary toxicity, chronic interstitial inflammation	Newton *et al*., 1994	BMC_{10} (HEC): 0.074 mg/m^3 (74 µg/m^3)	300	US EPA IRIS, 1995	1995
ESL; Annual average	Chronic	0.5	NA	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-4 Oral Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	0.4	Longevity and blood and glucose levels	Schroeder *et al*., 1970	LOAEL: 0.36 mg/kg/day	1,000	US EPA IRIS, 1991	1991
RfD	Chronic	0.4	NA	NA	NA	NA	MOE, 2011	2011
TDI	Chronic	6.0	Reduced body weight gain and water intake	Poon *et al*., 1998; Lynch *et al.,* 1999	NOAEL: 6 mg/kg/day	1,000	WHO, 2003	2003
TDI	Chronic	6.0	Reduced body weight gain and water intake	Poon *et al*., 1998; Lynch *et al.,* 1999	NOAEL: 6 mg/kg/day	1,000	RIVM, 2009	2009

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.4 Arsenic

The 24-hour acute inhalation exposure limit of 0.3 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 0.015 µg/m^3 proposed by the Cal EPA (2008) were used for the assessment of arsenic (Table A-5). The acute exposure limits was chosen as it was the only available 24-hour values. The chronic exposure limit was chosen based on its level of conservatism and because it is protective of sensitive individuals (children).

The UR of 6.4x10^{-3} (µg/m^3)$^{-1}$ proposed by the Health Canada (2010) was selected for the current assessment, however, it is noted that a more recent derivation provided by TCEQ (2012) places the inhalation UR for arsenic at 1.5x10^{-4} (µg/m^3)$^{-1}$.

The chronic oral exposure limit of 0.3 µg/kg/d proposed by US EPA IRIS (1993), ATSDR (2007), and MOE (2011) was used for the assessment of arsenic (Table A-6). The oral SF of 1.5x10^{-3} (µg/kg/d)$^{-1}$ proposed by US EPA IRIS (1998) was selected for the current assessment.

Table A-5 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.3	Respiratory tract irritation, gastrointestinal effects, and central nervous system depression	NA	NA	NA	MOE, 2012	NA
REL	Chronic	0.015 (children); 0.044 (adults)	Decreased intellectual function, adverse effects on neurobehavioural development	Wasserman *et al*., 2004; Tsai *et al*., 2003 (children); von Ehrenstein *et al*., 2006 (adults)	LOAEL: 0.46 µg/m^3	30 (children); NA (adults)	Cal EPA, 2008	NA
RfC	Chronic	0.03	NA	NA	NA	NA	MOE, 2011	NA
TCA	Chronic	1	Incidence of lung tumours	NA	LOAEC: 10 µg/m^3	10	RIVM, 2001	1999/2000
UR	Chronic	6.4x10^{-3} (µg/m^3)$^{-1}$	Lung cancer	Higgins *et al*., 1982	NA	NA	Health Canada, 2010	1993
UR	Chronic	3.3x10^{-3} (µg/m^3)$^{-1}$	Lung tumour incidence	Enterline *et al*., 1987	NA	NA	Cal EPA, 2011	2002
UR	Chronic	4.3x10^{-3} (µg/m^3)$^{-1}$	Lung cancer	Enterline and Marsh, 1982; Higgins *et al*., 1982; Brown and Chu 1983a,b,c; Lee-Feldstein, 1983	NA	NA	US EPA IRIS, 1998	1995
UR	Chronic	1.5x10^{-3} (µg/m^3)$^{-1}$	NA	NA	NA	NA	WHO, 2000	2000

Table A-5 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UR	Chronic	$1.5x10^{-3}$ $(\mu g/m^3)^{-1}$	NA	WHO, 2000	NA	NA	MOE, 2011	NA
UR	Chronic	$1.5x10^{-4}$ $(\mu g/m^3)^{-1}$	Lung cancer rates and survival probabilities from occupational exposures	Jarup *et al*., 1989; Enterline *et al.,* 1995; Lubin *et al*., 2000, 2008	NA	NA	TCEQ, 2012	2012

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

Table A-6 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	0.3	Hyperpigmentation, keratosis, and possible vascular complications	Tseng *et al*., 1968; Tseng, 1977	NOAEL: 0.8 µg/kg/d	3	US EPA IRIS, 1993	1991
MRL	Chronic	0.3	Dermal effects	Tseng *et al*., 1968; Tseng, 1977	NOAEL: 0.8 µg/kg/d	3	ATSDR, 2007	2005
RfD	Chronic	0.3	NA	NA	NA	NA	MOE, 2011	NA
REL	Chronic	$3.5x10^{-3}$	Decreased intellectual function, adverse effects on neurobehavioural development in 10 year old children	Wasserman *et al*., 2004; Tsai *et al*., 2003	LOAEL: 0.105 µg/kg/d	30	Cal EPA, 2008	NA
TDI	Chronic	1.0	Hyperpigmentation	NA	NOAEL: 2.1 mg/kg/d	2	RIVM, 2001	NA
SF	Chronic	$1.8x10^{-3}$ $(\mu g/kg/d)^{-1}$	Bladder, lung and liver cancer	Chen *et al*., 1985; Wu *et al*., 1989; Morales *et al*., 2000	NA	NA	Health Canada, 2010	2006
SF	Chronic	$1.5x10^{-3}$ $(\mu g/kg/d)^{-1}$	Prevalence of skin cancer	Tseng *et al*., 1968; Tseng, 1977	NA	NA	US EPA IRIS, 1998	1995
SF	Chronic	$1.5x10^{-3}$ $(\mu g/kg/d)^{-1}$	Human skin cancer incidence	Tseng *et al*., 1968; Tseng, 1977	NA	NA	Cal EPA, 2008	2002
SF	Chronic	$1.5x10^{-3}$ $(\mu g/kg/d)^{-1}$	NA	Tseng *et al*., 1968; Tseng, 1977	NA	NA	MOE, 2011	2011

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.5 Barium

The 24-hour acute inhalation exposure limit of 10 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 1 µg/m^3 proposed by the RIVM (2001) were used for the assessment of barium (Table A-7). The acute exposure limit was chosen as it was the only available values. The chronic exposure limit was chosen based on the robustness of the supporting study data. Further, this value was adopted and endorsed by the MOE (2011).

The chronic oral exposure limit of 200 µg/kg/d proposed by US EPA IRIS (2005) was used for the assessment of barium (Table A-8). The chronic oral exposure limit was chosen based on robustness of the supporting study. Further, this value was adopted and endorsed by Health Canada (2010) and ATSDR (2007).

Table A-7 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	10	Abdominal cramps, nausea, vomiting, muscle weakness, and paralysis	NA	NA	NA	MOE, 2012	NA
TCA	Chronic	1	Cardiovascular effects	NA	NOAEC: 110 µg/m^3	100	RIVM, 2001	1999/2000
RfC	Chronic	1	NA	RIVM, 2001	NA	NA	MOE, 2011	NA
ESL; Annual average	Chronic	0.5	Health based	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-8 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	200	NA	NA	NA	NA	MOE, 2011	NA
TDI	Chronic	200	Renal Lesions	NTP, 1994	$BMDL_{05}$: 63 mg/kg/day	300	Health Canada, 2010	NA
RfD	Chronic	200	Nephropathy	NTP, 1994	$BMDL_{05}$: 63 mg/kg/day	300	US EPA IRIS, 2005	NA
MRL	Chronic	200	Renal lesions, nephropathy	NTP, 1994	$BMDL_{05}$: 61 mg/kg/day	300	ATSDR, 2007	NA
TDI	Chronic	20	Cardiovascular effects	Vermeire *et al*., 1991	NOAEL: 0.2 mg/kg/day	10	RIVM, 2001	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.6 Benzene

The acute inhalation exposure limit of 29 µg/m^3 proposed by the ATSDR (2007), and the chronic inhalation exposure limit of 3 µg/m^3, also proposed by the Cal EPA (2014) were used for the non-cancer assessment of benzene (Table A-9). These exposure limits were chosen as the most conservative values and considering the robustness of the supporting data.

The UR of 2.9x10^{-5} (µg/m^3)$^{-1}$ proposed by the Cal EPA (2011) was used for the assessment of benzene, based the robustness of the supporting study data.

The chronic oral exposure limit of 4 µg/kg/d proposed by US EPA IRIS (2003) was used for the oral assessment of benzene (Table A-10). The chronic oral exposure limit was selected based on the robustness of the supporting study data.

The SF of 5.5x10^{-5} (µg/kg/d)$^{-1}$ also proposed by US EPA IRIS (2000) was used for the carcinogenic assessment of benzene. This SF was selected based on its level of conservatism and robustness of the supporting study data.

Table A-9 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	2.3	Incidence of cancer	Crump, 1994	NA	NA	MOE, 2011a	2011
MRL[b]	Acute	29 µg/m^3	Reduced lymphocyte proliferation following mitogen stimulation in mice	Rozen *et al*., 1984	LOAEL: 2.55 ppm (8,200 µg/m^3)	300	ATSDR, 2007	2007
RfC	Chronic	30	Decreased lymphocyte count	Rothman *et al*., 1996	BMCL: 8,200 µg/m^3	300	US EPA IRIS, 2003	2003
MRL	Chronic	9.58 µg/m^3	Statistically significant decreased counts of B-lymphocytes	Lan *et al*., 2004	$BMCL_{ADJ}$ (0.25sd): 0.03 ppm (95.8 µg/m^3)	10	ATSDR, 2007	2007
ReV	Chronic	280	Decreased absolute lymphocyte count	Rothman *et al*., 1984	POD (HEC): 2.6 ppm (8,300 µg/m^3)	30 (HQ= 1)	TCEQ, 2007	2007
ESL	Chronic	54	Decreased absolute lymphocyte count	Rothman *et al*., 1984	POD (HEC): 2.6 ppm (8,300 µg/m^3)	30 (HQ= 0.3)	TCEQ, 2007	2007
REL	Chronic	3 µg/m^3	Decreased peripheral blood cells in workers	Lan *et al*., 2004	POD (HEC): 0.204 ppm (0.665 mg/m^3)	200	Cal EPA, 2014	2014
AAQC; Annual Average	Chronic	0.45	Incidence of cancer	Crump, 1994	NA	NA	MOE, 2011a	2011

Table A-9 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UR	Chronic	$2.2x10^{-6}$ $(\mu g/m^3)^{-1}$	Leukemia	Rinsky *et al*., 1987	NA	NA	US EPA IRIS, 2000	2000
UR	Chronic	$2.2x10^{-6}$ $(\mu g/m^3)^{-1}$	NA	US EPA IRIS, 2000	NA	NA	MOE, 2011b	2011
UR	Chronic	$2.9x10^{-5}$ $(\mu g/m^3)^{-1}$	Leukemia	Yin *et al*., 1994; Yin *et al.,* 1996	NA	NA	Cal EPA, 2011	2009
UR	Chronic	$3.3x10^{-6}$ $(\mu g/m^3)^{-1}$	Acute myelogenous leukemia	Rinsky *et al.,* 1987	NA	NA	Health Canada, 2010	2010
UR	Chronic	$6.0x10^{-6}$ $(\mu g/m^3)^{-1}$	Leukaemia	Crump and Allen, 1984; Paustenbach *et al.,* 1992	NA	NA	WHO, 2000	2000
UR	Chronic	$2.2x10^{-6}$ $(\mu g/m^3)^{-1}$	Leukemia	Crump and Allen, 1984	NA	NA	TCEQ, 2007	2007
MPR	Chronic	20 $\mu g/m^3$ (cancer	NA	NA	NA	NA	RIVM, 2001	1999/2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.
[b] Value taken as 24-hour exposure limit.

Table A-10 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	4	Decreased lymphocyte cell count	Rothman *et al*., 1996	BMDL: 1.2 mg/kg/d	300	US EPA IRIS, 2003	2003
MRL	Chronic	0.5	Decreased B cell count	Lan *et al*., 2004	BMCL(adj): 0.014 mg/kg/d	30	ATSDR, 2007	2005
SF	Chronic	$5.5x10^{-5}$ $(\mu g/kg/d)^{-1}$	Leukemia	Rinsky *et al.,* 1981;1987; Paustenbach *et al*., 1993; Crump, 1994; US EPA, 1998; 1999	NA	NA	US EPA IRIS, 2000	2000
SF	Chronic	$1.0x10^{-4}$ $(\mu g/kg/d)^{-1}$	Leukemia incidence	Rinsky *et al*., 1981	NA	NA	Cal EPA, 2011	2009

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.7 Beryllium

The 24-hour acute inhalation exposure limit of 0.01 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 0.007 µg/m^3 proposed by the Cal EPA (2001) were used for the assessment of beryllium (Table A-11). The acute exposure limit was chosen as it was the only available value. The chronic exposure limit was chosen based on its level of conservatism and considering the robustness of the supporting study data. Further, this value was adopted and endorsed by the MOE (2011).

The UR of 2.4x10^{-3} (µg/m^3)$^{-1}$ proposed by the US EPA IRIS (1998b) was used for the carcinogenic assessment of beryllium. Analogous values were also proposed by the WHO (2001) and Cal EPA (2009), and endorsed by the MOE (2011).

The chronic oral exposure limit of 2 µg/kg/d proposed by US EPA IRIS (1998) was used in the assessment of beryllium (Table A-12). Similarly, MOE (2011), WHO (2001), ATSDR, (2002), and Cal EPA (2001) has adopted and endorsed this value. As such, the chronic exposure limit was chosen as it was adopted and endorsed by many agencies and was the only available value.

Table A-11 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.01	Respiratory tract irritation and pulmonary effects	NA	NA	NA	MOE, 2012	NA
ESL; Annual average	Chronic	0.002	Health based	NA	NA	NA	TCEQ, 2014	2003
REL	Chronic	0.007	Beryllium sensitization (chronic beryllium disease)	Kreiss *et al.*, 1996	LOAEL (HEC): 0.2 µg/m^3	30	Cal EPA, 2001	NA
RfC	Chronic	0.007	NA	Cal EPA, 2001	NA	NA	MOE, 2011	NA
RfC	Chronic	0.02	Beryllium sensitization and progression to chronic beryllium disease	Kreiss *et al.*, 1996	LOAEL (HEC): 0.2 µg/m^3	10	US EPA IRIS, 1998a	NA
TC	Chronic	0.02	Beryllium sensitization and chronic beryllium disease	Kreiss *et al.*, 1996	LOAEL (ADJ): 0.2 µg/m^3	10	WHO, 2001	NA
UR	Chronic	2.4x10^{-3} (µg/m^3)$^{-1}$	Lung cancer	Wagoner *et al.*, 1980	NA	NA	US EPA IRIS, 1998b	NA
UR	Chronic	2.4x10^{-3} (µg/m^3)$^{-1}$	Lung cancer	Wagoner *et al.*, 1980	NA	NA	WHO, 2001	NA

Table A-11 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UR	Chronic	$2.4x10^{-3}$ $(\mu g/m^3)^{-1}$	Lung caner	Wagoner *et al.*, 1980	NA	NA	Cal EPA, 2011	NA
UR	Chronic	$2.4x10^{-3}$ $(\mu g/m^3)^{-1}$	NA	US EPA IRIS, 1998b	NA	NA	MOE, 2011	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

Table A-12 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	2	Small intestinal lesions	Morgareidge *et al.*, 1976	BMD_{10}: 0.46 mg/kg/day	300	US EPA IRIS, 1998b	NA
TI	Chronic	2	Gastrointestinal lesions	Morgareidge *et al.*, 1976	BMD_{10}: 0.46 mg/kg/day	300	WHO, 2001	NA
MRL	Chronic	2	Small intestine lesions	Morgareidge *et al.*, 1976	$BMDL_{10}$: 0.56 mg/kg/day	300	ATSDR, 2002	NA
REL	Chronic	2	Small intestinal lesions	Morgareidge *et al.*, 1976	BMD_{05}: 0.244 mg/kg/day	100	Cal EPA, 2001	NA
RfD	Chronic	2	NA	NA	NA	NA	MOE, 2011	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.8 Boron

The acute inhalation exposure limit of 300 µg/m³ proposed by the ATSDR (2010) was chosen for use as the 24-hour value, while the annual inhalation exposure limit of 5 µg/m^3 proposed by the TCEQ (2014) was used for the chronic inhalation assessment of boron (Table A-13). The ATSDR (2010) acute exposure limit was selected based on the robustness of the supporting study data and in lieu of other available values. It is noted that the ATSDR (2010) 14 day MRL is backed by supporting study data and documentation, its application to the 24-hour exposure durations was considered conservative. The chronic exposure limit was also selected due to a lack of other available values.

The chronic oral exposure limit of 200 µg/kg/d proposed by US EPA IRIS (2004) was chosen for use in this assessment (Table A-14). The chronic exposure limit was selected based on the robustness of the supporting study data.

Table A-13 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	120	Particulate based	NA	NA	NA	MOE, 2012	2012
MRL; 14 days or less [b]	Acute	300	Significantly increased volume of nasal secretions	Cain *et al*., 2004; 2008	NOAEL: 0.8 mg/m^3 (800 µg/m^3)	3	ATSDR, 2010	2010
ESL; Annual average	Chronic	5	Respiratory effects	NA	NA	NA	TCEQ, 2014	2007

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-14 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	200	Decreases in mean fetal weight	Heindel *et al*., 1992; Prince *et al.,* 1996	$BMDL_{05}$: 10.3 mg/kg/day	66	US EPA IRIS, 2004	2004
RfD	Chronic	200	NA	NA	NA	NA	MOE, 2011	2011
ADI	Chronic	17.5	Testicular atrophy resulting in infertility and spermatogenic arrest	Weir and Fisher, 1972	NOAEL: 8.75 mg/kg/day	500	Health Canada, 2010	2010
TDI	Chronic	200	Decreased fetal body weight	NA	$BMDL_{05}$: 10.3 mg/kg/day	60	WHO, 2009	2009

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.9 Butadiene, 1,3-

The annual inhalation exposure limit of 2 µg/m^3 proposed by the US EPA IRIS (2002) was used for the assessment of 1,3-butadiene (Table A-15). The chronic exposure limits was chosen based on its level of conservatisms relative to other available values.

The UR of 5.0x10^{-7} (µg/m^3)$^{-1}$ proposed by TCEQ (2008) was used for the carcinogenic assessment of 1,3-butadiene. The UR was selected based on the robustness of the study data of epidemiological data on leukemia risk from occupational exposure.

Table A-15 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfC	Chronic	2	Ovarian atrophy	NTP, 1993	$BMCL_{10}$ (HEC): 1.98 mg/m^3	1,000	US EPA IRIS, 2002	2002
REL	Chronic	20	Ovarian atrophy	NTP, 1993	BMC_{05} (HEC): 0.25 ppm	30	Cal EPA, 2000	2000
UR	Chronic	5.9x10^{-3} (µg/m^3)$^{-1}$	Leukemia incidence data	Delzell *et al*., 1995	NA	NA	Health Canada, 2004; Environment Canada, 2000	2000
UR	Chronic	3.0x10^{-5} (µg/m^3)$^{-1}$	Leukemia incidence data	Delzell *et al.,* 1995	NA	NA	US EPA IRIS, 2002	2002
UR	Chronic	1.7x10^{-4} (µg/m^3)$^{-1}$	Lung alveolar and bronchiolar neoplasms in female mice	Melnick *et al.,* 1990	NA	NA	Cal EPA, 2005	2002
UR	Chronic	5.0x10^{-7} (µg/m^3)$^{-1}$	Leukemia incidence data	NA	NA	NA	TCEQ, 2008	2008

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.10 Cadmium

The acute inhalation exposure limit of 0.03 µg/m³ proposed by the ATSDR (2012) and the annual inhalation exposure limit of 0.005 µg/m^3 proposed by the MOE (2011) were used for the assessment of Cadmium (Table A-16). The exposure limits were chosen based on the absence of other available values and their level of conservatism.

The UR of 0.0098 (µg/m^3)$^{-1}$ proposed by Health Canada (2010) was used for the carcinogenic assessment of cadmium. This value was the most conservative value available and was adopted and endorsed by the MOE (2011).

The chronic oral exposure limit of 0.1 µg/kg/d proposed by ATSDR (2012) was used in the assessment of Cadmium (Table A-17). The selection of the oral exposure limit was based on the level of conservatism and the robustness of the supporting study data.

Table A-16 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.03	Adverse kidney effects	Thun *et al*., 1991	Annual guideline of 0.005 µg/m³	NA	MOE, 2007	2007
MRL	Acute	0.03	Histological changes in the respiratory tract	NTP, 1995	LOAEL(HEC) : 0.01 mg/m³	300	ATSDR, 2012	2012
RfC	Acute	0.03	Adverse kidney effects	MOE, 2007	24-hour AAQC: 25 ng/m^3 (0.025 µg/m^3)	NA	MOE, 2011	2007
AAQC; Annual	Chronic	0.005	Proteinuria associated with proximal tubular dysfunction and lung cancer	Thun *et al.,* 1991	LOAEL(ADJ): 0.270 µg/m^3	50	MOE, 2007	2007
MRL	Chronic	0.01	Renal damage (β_2-microglobulin proteinuria)	Roels *et al*., 1993; Järup and Elinder 1994; Chen *et al*., 2006a, 2006b	95% lower CL of UCD10 (0.5 µg/g creatinine): 0.1 µg/m³	9	ATSDR, 2012	2012
REL	Chronic	0.02	Kidney and respiratory effects	Lauwerys *et al.,* 1974	NOAEL(ADJ) : 0.5 µg/m³	30	Cal EPA, 2012	2000
Guideline	Chronic	0.005	NA	NA	NA	NA	WHO 2000	1999
ESL; Annual average	Chronic	0.01	NA	NA	NA	NA	TCEQ, 2014	2003
UR	Chronic	9.8x10^{-3} (µg/m^3)$^{-1}$	Lung tumors	Takenaka *et al*., 1983; Oldiges *et al*., 1984	NA	NA	Health Canada, 2010	1994
UR	Chronic	9.8x10^{-3} (µg/m^3)$^{-1}$	NA	Health Canada, 2010	NA	NA	MOE, 2011	1994

Table A-16 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UR	Chronic	$1.8x10^{-3}$ $(\mu g/m^3)^{-1}$	Lung and upper respiratory tract cancers	Thun *et al.,* 1985	NA	NA	US EPA, 1994	1994
UR	Chronic	$4.2x10^{-3}$ $(\mu g/m^3)^{-1}$	Lung cancer	Thun *et al.,* 1985	NA	NA	Cal EPA, 2009	1990

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available
[a] Units of $\mu g/m^3$ unless otherwise noted.

Table A-17 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	0.03.2	Urinary cadmium concentrations	Modified from Cal EPA DW (2006)	NA	NA	MOE, 2011	NA
TDI	Chronic	1.0	Renal tubular dysfunction	Health Canada, 1986; Friberg *et al.,* 1971; adopted from the WHO (2004, updated in 2011)	pTWI: 7 µg/kg/week	NA	Health Canada, 2010	NA
RfD	Chronic	0.5 (water) 1.0 (food)	Significant proteinuria	US EPA, 1985	NOAEL: 5.0 µg/kg/d (water) 10 µg/kg/d (food)	10	US EPA IRIS, 1994	1994
REL	Chronic	0.5	Significant proteinuria	US EPA, 1985	NOAEL: 5.0 µg/kg/d	10	Cal EPA, 2012	2000
MRL	Chronic	0.1	Renal damage	Buchet *et al.,* 1990; Järup *et al.,* 2000; Jin *et al.,* 2004; Kobayashi *et al.,* 2006; Shimizu *et al.,* 2006; Suwazono *et al.,* 2006; Wu *et al.,* 2001	$UCDL_{10}$:0.33 µg/kg/d	3	ATSDR, 2012	NA
TDI	Chronic	0.5	Renal tubular dysfunction	Nogawa *et al.,* 1989	Population-based adverse effect level: 1.0 µg/kg/d	2	RIVM, 2001	2001

Table A-17 Oral Toxicity Reference Values								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
TDI	Chronic	1.0	Concentrations in the renal cortex	WHO JECFA, 2011	pTMI: 25 ug/kg/month	NA	WHO, 2011	1988

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.11 Carbon Monoxide

The 8-hour acute inhalation exposure limit of 6,000 µg/m^3 proposed by Health Canada (2006) was used for the assessment of carbon monoxide. This acute inhalation exposure limit was selected for use as it was the most conservative value relative to the available values.

Table A-18 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
Acute; 8-hour	Acute	6,000	Carboxyhemoglobin blood level of less than 1%	NA	NA	NA	Health Canada, 2006	2006
Acute; 8-hour	Acute	9 ppm (11,000 µg/m^3)	NA	NA	NA	NA	US EPA, 2011	2011

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.12 Carbon tetrachloride

The 24-hour acute inhalation exposure limit of 2.4 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 100 µg/m^3 proposed by US EPA (2010) were used for the assessment of carbon tetrachloride (Table A-19). The acute exposure limit was selected for use due to the absence of other viable 24-hour exposure limits. The chronic inhalation exposure limit of 100 µg/m^3 derived by US EPA IRIS (2010) was selected in the assessment as it was more conservative and scientifically defensible than other exposure limits.

The IUR of 6 x 10-6 (µg/m^3)$^{-1}$ derived by US EPA IRIS (2010) was selected as it was the most scientifically defensible value.

Table A-19 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	2.4	Central nervous system depression, gastrointestinal effects, and pulmonary failure	NA	NA	NA	MOE, 2012	2012
RfC	Chronic	100	Fatty change in liver (rat, mouse)	JBRC, 1998; Nagano *et al*., 2007	$BMDL_{10}$ (HEC): 14.3 mg/m^3 (14,300 µg/m^3)	100	US EPA IRIS, 2010	2010
MRL	Chronic	190	Increased liver weight, serum enzymes, liver histopathology	JBRC, 1998; Nagano *et al*., 1998	NOAEL (HEC): 0.9 ppm (5,700 µg/m^3)	30	ATSDR, 2005	2005
REL	Chronic	40	Increased liver weight and hepatic fatty infiltration	Adams *et al*., 1952	LOAEL (HEC): 1.7 ppm (~11,000 µg/m^3)	300	Cal EPA, 2000	2000
TCA	Chronic	60	Hepatic effects	NA	NOAEC (ADJ): 6.4 mg/m^3 (6,400 µg/m^3)	100	RIVM, 2001	2001
ESL; Annual average	Chronic	13	NA	NA	NA	NA	TCEQ, 2014	2003
RfC	Chronic	2.0	NA	NA	NA	NA	MOE, 2011	2011
Unit risk	Chronic	6.0 x 10^{-6} (µg/m^3)$^{-1}$	Pheochromocytoma (mouse)	JBRC, 1998; Nagano *et al*., 2007	NA	NA	US EPA IRIS, 2010	2010
Unit risk	Chronic	4.2 x 10^{-5}	Liver tumour	Edwards *et al*., 1942	NA	NA	Cal EPA, 2011	2009

Table A-19 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value*** [a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
		$(\mu g/m^3)^{-1}$						

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.13 Chloroform

The 24-hour acute inhalation exposure limit of 1 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 100 µg/m^3 proposed by ATSDR (1997) were used for the assessment of chloroform (Table A-20). The acute exposure limit was selected for use due to the absence of other viable 24-hour exposure limits. The chronic inhalation MRL of 100 µg/m^3 derived by the ATSDR (1997) and adopted by the MOE (2011) was selected in the assessment as it was based on an occupational study of workers exposed to 2 to 205 ppm of chloroform over a period of 1 to 4 years.

The IUR of 5.3 x 10-6 (µg/m^3)$^{-1}$ derived by the Cal EPA (2009) was selected in this assessment given that it was the most scientifically defensible exposure limit.

Table A-20 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; 1-hour	Acute	100	NA	NA	NA	NA	TCEQ, 2013	2003
REL; 7-hour	Acute	150	Histological changes in nasal epithelium	Schwetz *et al*., 1974	LOAEL; 30 ppm (30,000 µg/m^3)	1,000	Cal EPA, 2008	NA
AAQC; 24-hour	Acute	1	Respiratory, cardiovascular, hepatic, gastrointestinal, renal, and neurological effects	NA	NA	NA	MOE, 2012	NA
MRL; 14 days or less	Acute	500	Hepatic effects	Larson *et al*., 1994	NOAEL: 3 ppm (14,600 µg/m^3)	30	ATSDR, 1997	1997
ESL; Annual average	Chronic	10	NA	NA	NA	NA	TCEQ, 2013	2003
TCA	Chronic	100	Liver, kidney, and developmental toxicity	NA	NOAEL: 110,000 µg/m^3	1,000	RIVM, 2001	1986
AAQC	Chronic	0.2	Health-based	NA	NA	NA	MOE, 2012	NA
REL	Chronic	300	Liver toxicity (degenerative, foamy vacuolization and necrosis); increased liver weights	Torkelson *et al*., 1976	LOAEL (HEC): 15.9 ppm (78,000 µg/m^3)	300	Cal EPA, 2008	NA
MRL	Chronic	100	Hepatomegaly, toxic hepatitis, and hepatosteatosis (human)	Bomski *et al*., 1967	LOAEL: 2 ppm (9,700 µg/m^3)	100	ATSDR, 1997	NA
RfC	Chronic	98	NA	NA	NA	NA	MOE (2011)	NA

Table A-20 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
Unit risk	Chronic	2.3×10^{-5} $(\mu g/m^3)^{-1}$	Hepatocellular carcinoma	NCI, 1976	NA	NA	US EPA IRIS, 2001	NA
Unit risk	Chronic	5.3×10^{-6} $(\mu g/m^3)^{-1}$	Renal tumors (rat, mouse)	NCI, 1976; Jorgenson *et al*., 1985	NA	NA	Cal EPA, 2011	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.14 Chromium (Total)

The 24-hour acute inhalation exposure limit of 0.5 µg/m^3 also proposed by the MOE (2011), and the annual inhalation exposure limit of 0.14 µg/m^3 proposed by the TCEQ (2013) were used for the assessment of total chromium (Table A-21). The acute exposure limits were chosen based on their level of conservatism and absence of other available values. The chronic exposure limit was chosen based on its level of conservatism relative to other available values.

The UR of 1.1x10^{-2} (µg/m^3)$^{-1}$ proposed by Health Canada (2010) was used for the carcinogenic assessment of total chromium. This was the only available value for this COPC.

The chronic oral exposure limit of 1,500 µg/kg/d proposed by US EPA IRIS (1998) was selected for use in the assessment (Table A-22). The chronic exposure limit was chosen based on its scientific defensibility. Further, the MOE (2011) endorses the use of the value.

Table A-21 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.5	Respiratory effects in rodents	Derelanko *et al.* 1999	NA	NA	MOE, 2011a	2011
Annual Average	Chronic	60	Lack of kidney effects (as measured urinary levels of protein and various enzymes)	RIVM, 2001	NA	NA	MOE, 2011b	NA
TCA (**Cr (III), insoluble & metallic)**	Chronic	60	Lack of kidney effects (as measured urinary levels of protein and various enzymes)	NA	NOAEL: 600 µ/m^3	10	RIVM, 2001	1999/2000
ReV	Chronic	0.14	Increased relative lung and trachea weight in male and female rats	Derelanko *et al.* 1999	POD(HEC): 808.6 µg/m^3	1,000	TCEQ, 2013	2009
UR	Chronic	1.1x10^{-2} (µg/m^3)$^{-1}$	Increased incidence of lung cancer	Mancuso, 1975	NA	NA	Health Canada, 2010	1993

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-22 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	1,500	NA	NA	NA	NA	MOE, 2011b	NA
TDI	Chronic	1.0	Hepatotoxicity, irritation or corrosion of the gastrointestinal mucosa, encephalitis	NA	NA	NA	Health Canada, 2010	1993
RfD (**Cr (III), insoluble salts)**	Chronic	1,500	None observed	Ivankovic and Preussman, 1975	NOAEL (ADJ): 1,468 mg/kg/d	100	US EPA IRIS, 1998	1998
pTDI	Chronic	5.0	Increased tissue levels. No changes in blood or pathological tissues	MacKenzie *et al.*, 1958	NOAEL: 2.4 mg/kg/day	500	RIVM, 2001	1999/2000
TDI	Chronic	5,000	NA	NA	NA	NA	RIVM, 2001	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.15 Chromium (VI)

The 24-hour acute inhalation exposure limit of 0.0007 µg/m^3 also proposed by the MOE (2012), and the annual inhalation exposure limit of 0.1 µg/m^3 proposed by the US EPA IRIS (1998) were used for the assessment of chromium (VI) (Table A-23). The acute exposure limits were chosen based on their level of conservatism and absence of other available values. The chronic exposure limit was chosen based on its level of conservatism relative to other available values and the robustness of the study data.

The UR of 7.6x10^{-2} (µg/m^3)$^{-1}$ proposed by Health Canada (2010) was used for the inhalation carcinogenic assessment of chromium (VI). The UR was selected based on the level of conservatism relative to the other available values.

The chronic oral exposure limit of 0.9 µg/kg/d proposed by ATSDR (2012) was selected for use in the assessment (Table A-24). The chronic exposure limit was chosen based on its level of conservatisms relative to other available values.

The SF of 4.2x10^{-4} (µg/kg/d)$^{-1}$ proposed by Cal EPA (2009) was used for the oral carcinogenic assessment of chromium (VI). The SF was selected as there were no other available value.

Table A-23 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.0007 (in TSP)	Health	NA	NA	NA	MOE, 2012	NA
AAQC; Annual Average	Chronic	0.00014 (in TSP)	Health	NA	NA	NA	MOE, 2012	NA
REL	Chronic	0.1	NA	US EPA IRIS, 1998	NA	NA	MOE, 2011	2011
RfC	Chronic	0.1	Lactate dehydrogenase in bronchioalveolar lavage fluid	Glaser *et al.,* 1990; Malsch *et al.,* 1994	Adjusted BMD: 0.016 mg/m^3	300	US EPA IRIS, 1998	1998
RfC	Chronic	0.008	Nasal septum atrophy	Lindberg and Hedenstierna, 1983	Adjusted LOAEL: 7.14 x 10^{-4} mg/m^3	90	US EPA IRIS, 1998	1998
MRL	Chronic	0.005	Upper respiratory effects	Lindberg and Hedenstierna, 1983	LOAEL: 0.002 mg/m^3	100	ATSDR, 2012	2012
REL	Chronic	0.2	Bronchoalveolar hyperplasia in lungs	Glaser *et al.,* 1990	BMC_{05}: 0.0125 mg/m^3	100	Cal EPA, 2001	2001
ESL	Chronic	0.01	NA	NA	NA	NA	TCEQ, 2014	2013
UR	Chronic	4.0x10^{-2}	NA	WHO, 2000	NA	NA	MOE, 2011	NA

Table A-23 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UR	Chronic	$7.6x10^{-2}$	Lung cancer	Mancuso, 1975	NA	NA	Health Canada, 2010	1993
UR	Chronic	$1.2x10^{-2}$	Lung cancer	Mancuso, 1975	NA	NA	US EPA IRIS, 1998	1998
UR	Chronic	$4.0x10^{-2}$	Lung cancer	Langard and Norseth, 1975; Langard and Vigander, 1983; Braver *et al.*, 1985;	NA	NA	WHO, 2000	1994
UR	Chronic	$1.5x10^{-2}$	Human lung cancer mortality data	Mancuso, 1975	NA	NA	Cal EPA, 2009	Prior to 2002
UR	Chronic	$4.0x10^{-2}$	NA	Slooff, 1990	NA	NA	RIVM, 2001	1990

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-24 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	8.3	NA	NA	NA	NA	MOE, 2011	NA
RfD	Chronic	3.0	None reported	MacKenzie et al., 1958	NOAEL (ADJ): 2.5 mg/kg/day	900	US EPA IRIS, 1998	1998
MRL	Chronic	0.9	Diffuse epithelial hyperplasia of the duodenum in female mice	NTP (2008)	BMD: 90 µg/kg/d	100	ATSDR, 2012	NA
REL	Chronic	20	Red blood cell effects	MacKenzie *et al.*, 1958	NOAEL: 2.4 mg/kg/day	100	Cal EPA, 2000	2000
SF	Chronic	$4.2x10^{-4}$ $(µg/kg/d)^{-1}$	Female mouse benign and malignant stomach tumour	Borneff *et al.,* 1968	NA	NA	Cal EPA, 2011	1991

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.16 Cobalt

The 24-hour acute inhalation exposure limit of 0.1 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 0.1 µg/m^3 proposed by the WHO (2006) were used for the assessment of cobalt (Table A-25). The acute exposure limits were chosen as they were the only available values. The chronic exposure limit was chosen based on its level of conservatism and considering the robustness of the supporting study data.

The chronic oral exposure limit of 1.4 µg/kg/d proposed by RIVM (2001) was used for the assessment of cobalt (Table A-26). The chronic oral exposure limit was chosen based on the level of conservatism and considering the robustness of the supporting study data.

Table A-25 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.1	Respiratory irritation	NA	NA	NA	MOE, 2012	NA
TC	Chronic	0.1	Respiratory symptoms and effects on lung function	Nemery *et al*., 1992	NOAEC (ADJ): 1.3 µg/m^3	10	WHO, 2006	NA
MRL	Chronic	0.1	Reduced lung function	Nemery *et al*., 1992	NOAEC (ADJ): 1.3 µg/m^3	10	ATSDR, 2004	NA
TCA	Chronic	0.5	Interstitial lung disease in humans	NA	LOAEL: 7 µg/m^3	100	RIVM, 2001	1999/2000
RfC	Chronic	0.5	NA	NA	NA	NA	MOE, 2011	NA
ESL; Annual Average	Chronic	0.02	NA	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-26 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
MRL	Intermediate	10	Increased levels of erythrocytes	Davis and Fields, 1958	LOAEL: 1,000 µg/kg/d	100	ATSDR, 2004	NA
RfD	Chronic	1.0	NA	NA	NA	NA	MOE, 2011	NA
TDI	Chronic	1.4	Cardiomyopathy	Vermeire *et al*., 1991	LOAEL: 40 µg/kg/d	30	RIVM, 2001	1991

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.17 Copper

The 24-hour acute inhalation exposure limit of 50 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 1 µg/m^3 proposed by the TCEQ (2014) were used for the assessment of copper (Table A-27). The exposure limits were chosen as they were the only available values, and/or considering their level of conservatism and the robustness of the supporting study data.

The chronic oral exposure limit of 91 µg/kg/d proposed by Health Canada (2009) was used in the assessment of copper (Table A-28). The exposure limit was selected as it was the only available value.

Table A-27 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	50	Health based	NA	NA	NA	MOE, 2012	NA
TCA	Chronic	1	Respiratory and immunological effects	NA	NOAEC: 100 µg/m^3	100	RIVM, 2001	1999/ 2000
ESL; Annual Average	Chronic	1 (in PM10)	Respiratory and immunological effects	NA	NA	NA	TCEQ, 2014	2007

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-28 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
UL	Chronic	91	Hepatotoxicity and gastrointestinal effects	O'Donohue *et al*., 1993	NA	NA	Health Canada, 2009	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.18 Dichlorobenzene, 1,4-

The 24-hour inhalation exposure limit of 95 µg/m^3 proposed by MOE (2012) and the annual inhalation exposure limit of 60 µg/m^3 proposed by ATSDR (2006) were used was used for the assessment of dichlorobenzene, 1,4- (Table A-29). The acute exposure limit was selected for use due to the absence of other viable 24-hour exposure limits. The chronic inhalation exposure limit of 60 µg/m^3 derived by ATSDR (2006) was selected in the assessment as it was more conservative and scientifically defensible than other exposure limits.

The UR of 1.1x10-5 (µg/m^3)$^{-1}$ proposed by the Cal EPA (2011) was used for the carcinogenic assessment of dichlorobenzene, 1,4-.

Table A-29 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC : 24-hour	Acute	95	Eye and respiratory system irritation	NA	NA	NA	MOE, 2012	NA
MRL	Chronic	60	Incidences of nasal lesions (rat)	Aiso *et al*. 2005 and Japan Bioassay Research Center 1995	$NOAEL_{10HEC}$: 27 ppm	30	ATSDR, 2006	NA
RfC	Chronic	800	Increased liver weights (rat)	Chlorobenzene Producers Association. 1986	$NOAEL_{HEC}$: 13 ppm	100	US EPA IRIS, 1996	1996
REL	Chronic	800	Increased liver weights (rat)	Chlorobenzene Producers Association. 1986	$NOAEL_{HEC}$: 13 ppm	100	Cal EPA, 2000	NA
ReV	Chronic	110	Increases in nasal olfactory epithelial lesions (rat)	Aiso *et al*. 2005	$BMCL_{10}$ 14.9 ppm	30	TCEQ, 2009	NA
Unit Risk	Chronic	$1.1x10^{-5}$ $(µg/m^3)^{-1}$	Liver tumours (mouse)	CDHS, 1988	NA	NA	Cal EPA, 2011	NA
Unit Risk	Chronic	$4.0X10^{-6}$ $(µg/m^3)^{-1}$	NA	NA	NA	NA	MOE, 2011	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.19 Dichloroethane, 1,2-

The 24-hour inhalation exposure limit of 2 µg/m^3 proposed by MOE (2012) and the annual inhalation exposure limit of 400 µg/m^3 proposed by Cal EPA (2008) were used was used for the assessment of dichloroethane, 1,2- (Table A-30). The acute exposure limit was selected for use due to the absence of other viable 24-hour exposure limits. The chronic inhalation exposure limit of 400 µg/m^3 derived by Cal EPA (2008) was selected in the assessment as it was more scientifically defensible than other exposure limits. This value was also endorsed by MOE (2011).

The UR of 2.6x10^{-5} (µg/m^3)$^{-1}$ proposed by the US EPA IRIS, 1991 was used for the carcinogenic assessment of dichloroethane, 1,2-. This value was selected for use in this assessment as it was the most scientifically defensible and conservative value.

Table A-30 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC : 24-hour	Acute	2	Respiratory, liver, kidney, and neurological effects				MOE, 2012	
REL	Chronic	400	Hepatotoxicity (rat)	Spreafico *et al.*, 1980.	$NOAEL_{HEC}$: 3.2 ppm	30	Cal EPA, 2008	2000
ESL	Chronic	4	Health based	NA	NA	NA	TCEQ, 2014	2007
MRL	Chronic	2,600	Histopathology (rat)	Spreafico *et al.*, 1980.	NOAEL: 50 ppm	90	ATSDR, 2001	NA
UR	Chronic	2.6x10-5 (µg/m^3)$^{-1}$	Hemangiosarcomas (rat)	Reitz *et al*. 1982	NA	NA	US EPA IRIS, 1991	1991

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.20 Dichloromethane

The 24-hour acute inhalation exposure limit of 220 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 400 µg/m^3 proposed by Cal EPA (2008) were used for the assessment of dichloromethane (Table A-31). The acute exposure limit was chosen as it was the most conservative value. The chronic exposure limit of 400 µg/m^3 derived by Cal EPA (2008) was selected given that it was the most conservative and scientifically defensible value.

The IUR of 1.0 x 10-6 (µg/m^3)$^{-1}$ derived by Cal EPA (2011b) was selected as it was the most conservative value.

Table A-31 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
MRL; 1-hour	Acute	2,000 (0.6 ppm)	Central nervous system effects	Winneke, 1974	$LOAEL_{ADj}$: 60 ppm	100	ATSDR, 2000	2000
REL; 1-hour	Acute	14,000 (4 ppm)	Central nervous system effects	Putz *et al.,* 1979	LOAEL: 680,000 µg/m^3 (195 ppm)	60	Cal EPA, 2008	2008
ReV; 1-hour	Acute	12,000	Central nervous system effects	Putz *et al.,* 1979	LOAEL: 680,000 µg/m^3 (195 ppm)	63	TCEQ, 2011	2011
MRL; 24-hour	Acute	1,000 (0.3 ppm)	Liver histopathology	Haun *et al.*, 1972	LOAEL: 25 ppm	90	ATSDR, 2000	2000
ESL; 24-hour	Acute	3,000	COHb formation	DiVincenzo and Kaplan, 1981	90,000 µg/m^3	NA	WHO, 2000	2000
AAQC; 24-hour	Acute	220	Central nervous system depression	NA	NA	NA	MOE, 2012	NA
MRL	Chronic	1,000 (0.3 ppm)	Liver histopathology	Nitschke *et al.,* 1988	NOAEL: 50 ppm	30	ATSDR, 2000	2000
TCA	Chronic	3,000	COHb formation	DiVincenzo and Kaplan, 1981	90,000 µg/m^3	NA	RIVM, 2001	1999/2000
REL	Chronic	400	COHb formation (human)	DiVincenzo and Kaplan, 1981	LOAEL: 40 ppm	100	Cal EPA, 2008	2008
RfC	Chronic	400	NA	Cal EPA, 2008	NA	NA	MOE, 2011	NA
ESL; annual average	Chronic	390	Liver histopathology	Nitschke *et al.,* 1988	LOAEL; 199 ppm	100	TCEQ, 2011	2011
RfC	Chronic	600	Liver histopathology	Nitschke *et al.,* 1988	1st percentileHEC: 17,200 µg/m^3	30	US EPA, 2011a	2011
AAQC	Chronic	44	Health based	NA	NA	NA	MOE, 2012	NA
UR	Chronic	2.3 x 10^{-8} (µg/m^3)$^{-1}$	Pulmonary/hepatic adenomas and carcinomas	NTP, 1986	NA	NA	Health Canada, 2010	1996
UR	Chronic	1.0 x 10^{-6} (µg/m^3)$^{-1}$	Lung tumors (mouse)	NTP, 1986	NA	NA	Cal EPA, 2011	2009
UR	Chronic	2.3 x 10^{-8} (µg/m^3)$^{-1}$	NA	Health Canada, 2010	NA	NA	MOE, 2011	2011
ReV	Chronic	2.3 x 10^{-8} (350 µg/m^3)	Liver and lung tumors	NTP, 1986	NA	NA	TCEQ, 2011	2011

Table A-31 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value*** [a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
			combined in female mice					
UR	Chronic	1.0×10^{-8} $(\mu g/m^3)^{-1}$	Liver and lung tumors	Mennear *et al.*, 1988; NTP, 1986	NA	NA	US EPA, 2011b	2011

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.21 Dioxins and Furans as Toxic Equivalents (TEQ)

The 24-hour acute inhalation exposure limit of 0.1 pg TEQ/m³ proposed by the MOE (2012) and the annual inhalation exposure limit of 40 pg TEQ/m^3 proposed by the Cal EPA (2008) were used for the assessment of dioxins and furans as toxic equivalents (Table A-32). These values were chosen based on the absence of other available exposure limits (24-hour) and the robustness of the supporting study data (annual). The Cal EPA REL (2008) was adopted and endorsed by the MOE (2011).

The chronic oral exposure limit of 7x10^{-7} µg/kg/d proposed by US EPA IRIS (2012) was used for the oral non-carcinogenic assessment of dioxin and furans as toxic equivalents (Table A-32). The chronic oral exposure limit was selected based on its level of conservatism relative to other available values.

Table A-32 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.1 pg TEQ/m^3	Systemic effects	NA	NA	NA	MOE, 2012	2012
REL	Chronic	0.00004 (40 pg TEQ/m^3)	Increased mortality, decreased weight gain, decreased erythoid values, increased urinary excretion of porphyrins and delta-aminolevulinic acid, and increased serum activities of several enzymes	Kociba *et al.,* 1978	NOAEL: 1 ng/kg/day	100	Cal EPA, 2008	2000
REL	Chronic	0.00004	NA	Cal EPA, 2008	NA	NA	MOE, 2011	NA
ESL; Annual average	Chronic	3x10^{-8}	NA	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-33 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	7x10^{-7}	Developmental effects; Decreased sperm count and motility in men	Baccarelli *et al*., 2008; Mocarelli *et al*., 2008	LOAEL (ADJ): 0.020 ng/kg/day (2x10^{-8} mg/kg/day)	30	US EPA IRIS, 2012	2012
MRL	Chronic	1x10^{-6}	Altered social behavior	Schantz *et al*., 1992	LOAEL: 1.2x10^{-7} mg/kg/day	90	ATSDR, 1998	1998
TDI	Chronic	2.3x10^{-6}	Developmental effects; Immune and reproductive effects in	Ohsako *et al*., 2001	NOAEL: 12.5 ng/kg/day	3.2	Health Canada, 2010	2010

Table A-33 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
			offspring of exposed dams		($1.25x10^{-5}$ mg/kg/day			
TDI	Chronic	$2.3x10^{-6}$	NA	NA	NA	NA	MOE, 2011	2011
pTDI	Chronic	$2x10^{-6}$	Development effects	Ohsako *et al*., 2001	NOAEL: 8 to 10 pg/kg/day ($8x10^{-9}$ to $1x10^{-8}$ mg/kg/day)	3.2	RIVM, 2009	2009
REL	Chronic	$1x10^{-5}$	Increased mortality, decreased weight gain, decreased erythoid values, increased urinary excretion of porphyrins and delta-aminolevulinic acid, and increased serum activities of several enzymes	Kociba *et al.,* 1978	NOAEL: $1x10^{-6}$ mg/kg/day	100	Cal EPA, 2008	2000
TDI	Chronic	$1x10^{-6}$	NA	Kociba *et al.,* 1978	LOAEL: 14 to 37 pg/kg/bw	10	WHO, 2000	2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.22 Ethylene dibromide

The 24-hour acute inhalation exposure limit of 3 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 0.8 µg/m^3 proposed by Cal EPA (2008) were used for the assessment of ethylene dibromide (Table A-34). The acute exposure limit was chosen as it was the most conservative value. The chronic exposure limit was selected for use in the current assessment as it was based on the robustness of the supporting study data. This value was also adopted by MOE (2011).

The IUR of 6.0 x 10^{-4} (µg/m^3)$^{-1}$ derived by US EPA IRIS (2004) was selected in the assessment as it is the most conservative inhalation cancer risk.

Table A-34 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; 1-hour	Acute	4	NA	NA	NA	NA	TCEQ, 2013	2003
AAQC; 24-hour	Acute	3	Hepatic and renal effects	NA	NA	NA	MOE, 2012	2012
RfC	Chronic	9	Nasal inflammation	NTP, 1982	$BMCL_{10}$ (HEC): 2.8 mg/m^3 (2,800 µg/m^3)	300	US EPA IRIS, 2004	2004
REL	Chronic	0.8	Reproductive effects (human)	Ratcliff *et al*., 1987	LOAEL (HEC): 31 ppb (240 µg/m^3)	300	Cal EPA, 2008	2001
RfC	Chronic	0.8	NA	NA	NA	NA	MOE, 2011	2011
ESL	Chronic	0.4	NA	NA	NA	NA	TCEQ, 2013	2003
Unit risk	Chronic	6.0 x 10^{-4} (µg/m^3)$^{-1}$	Nasal cavity tumours, hemangiosarcomas, and mesotheliomas (rat)	NTP, 1982	NA	NA	US EPA IRIS, 2004	2004
Unit risk	Chronic	6.0 x 10^{-4} (µg/m^3)$^{-1}$	NA	NA	NA	NA	MOE, 2011	2011
Unit risk	Chronic	7.1 x 10^{-5} (µg/m^3)$^{-1}$	Nasal tumour incidence	NTP, 1982	NA	NA	Cal EPA, 2011	2009

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.23 Formaldehyde

The 24-hour acute inhalation exposure limit of 65 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 9 µg/m^3 proposed by the Cal EPA (2008) were used for the assessment of formaldehyde (Table A-35). The acute exposure limit was selected in the assessment due to the absence of other viable 24-hour exposure limits. The chronic exposure limit was selected in the assessment as it was the most conservative exposure limit.

The IUR of 6.0 x 10^{-6} (µg/m^3)$^{-1}$ derived by Cal EPA (2011) was selected for use in this assessment as it was the most scientifically defensible.

Table A-35 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ReV; 1-hour	Acute	50 (0.04 ppm)	Eye and nose irritation	Pazdrak *et al.,* 1993; Krakowiak *et al.,* 1998	LOAEL: 0.4 ppm (500 µg/m^3)	30	TCEQ, 2008	2008
ESL; 1-hour	Acute	15 (HQ=0.3)	Eye and nose irritation	Pazdrak *et al.,* 1993; Krakowiak *et al.,* 1998	LOAEL: 0.4 ppm (500 µg/m^3)	30	TCEQ, 2008	2008
MRL; 2-hour	Acute	50	Nasal and eye irritation	Pazdrak *et al*., 1993	LOAEL: 0.4 ppm (500 µg/m^3)	30	ATSDR, 1999	1999
REL; 1-hour	Acute	55	Mild and moderate eye irritation	Kulle *et al*., 1987	$BMCL_{05}$: 0.44 ppm (540 µg/m^3)	10	Cal EPA, 2008	2008
AAQC; 24-hour	Acute	65	Respiratory and eye irritation	NA	NA	NA	MOE, 2012	2012
ReV	Chronic	11	Incidence of eye, nasal, and respiratory irritation	Wilhelmsson and Holmstrom, 1992	NOAEL (HEC): 0.032 mg/m^3 (32 µg/m^3)	3	TCEQ, 2008	2008
REL	Chronic	9	Nasal obstruction and discomfort, lower airway discomfort, eye irritation (human)	Wilhelmsson and Holmstrom, 1992	NOAEL: 0.09 mg/m^3 (90 µg/m^3)	10	Cal EPA, 2008	2008
MRL	Chronic	10 (0.008 ppm)	Clinical symptoms of mild irritation of eyes and upper respiratory tract. Mild damage to nasal epithelium	Holmstrom *et al*., 1989	LOAEL: 0.24 ppm (294 µg/m^3)	30	ATSDR, 1999	1999

Table A-35 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value*** [a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
UR	Chronic	1.3×10^{-5} $(\mu g/m^3)^{-1}$	Incidence of nasal squamous cell carcinoma	Kerns *et al.*, 1983	NA	NA	US EPA IRIS, 1991	1991
UR	Chronic	6.0×10^{-6} $(\mu g/m^3)^{-1}$	Nasal squamous carcinoma incidence (rat)	Kerns *et al.*, 1983	NA	NA	Cal EPA, 2011	2009
UR	Chronic	5.3×10^{-6} $(\mu g/m^3)^{-1}$	Incidence of nasal squamous tumours	Monticello *et al.*, 1996	NA	NA	Environment Canada and Health Canada (2001)	2001
UR	Chronic	5.6×10^{-7} $(\mu g/m^3)^{-1}$	Cell proliferation and cytotoxicity in rats	Schlosser *et al.*, 2003	NA	NA	TCEQ, 2008	2008

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.24 Hexachlorobutadiene

The annual inhalation exposure limit of 0.2 µg/m^3 proposed by TCEQ (2014) was used for the assessment of hexachlorobutadiene (Table A-36). The chronic exposure limit was selected for use in the current assessment due to the absence of other viable chronic exposure limits given it was the only value available.

The UR of 2.2x10^{-5} (µg/m^3)$^{-1}$ proposed by the US EPA IRIS (1991) was used for the carcinogenic assessment of hexachlorobutadiene due to the absence of other viable carcinogenic exposure limits given it was the only value available.

Table A-36 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL	Chronic	0.2	Health based	NA	NA	NA	TCEQ, 2014	2003
UR	Chronic	2.2x10^{-5} (µg/m^3)$^{-1}$	Renal tubular adenomas and adenocarcinomas (rat)	U.S. EPA, 1980	NA	NA	US EPA IRIS, 1991	1991

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.25 Lead

The 24-hour acute inhalation exposure limit of 0.5 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 0.15 µg/m^3 proposed by the US EPA (2008) were used for the assessment of lead (Table A-37). The acute exposure limit was chosen as it was the only available value. The chronic exposure limit was chosen as the most conservative of all other available values.

The UR of 1.2x10^{-5} (µg/m^3)$^{-1}$ proposed by Cal EPA (2011) was used for the assessment of carcinogenicity of lead. The UR was selected as there were no other available values.

For the chronic oral exposure limit, OEHHA (2007) calculated a lower level of concern of 2.86 µg/day, which results in the selected TRV of 0.17 µg/kg bw/day when the OEHHA value was divided by 16.5 kg for a toddler's body weight. This chronic oral exposure limit was selected based on its level of conservatism relative to other available values.

Table A-37 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.5	Blood lead level of 5 µg/dL	NA	NA	NA	MOE, 2012	2007
AAQC; 30-day	Acute	0.2	Primarily based on longer duration exposure limits aimed to reduce blood-lead concentration associated with neurodevelopmental effects in children	NA	NA	NA	MOE, 2012	2007
NAAQS; 3-month	Chronic	0.15	Protective of children and other at-risk populations	US EPA, 2008; US EPA, 2007a,b	NA	NA	US EPA, 2008	2008
Guideline value; Annual Average	Chronic	0.5	Adults: elevated free erythrocyte proporphyrin; Children: Cognitive functioning, such as the psychometric IQ and changes in vitamin D metabolism	Rosen *et al.,* 1980; Mahaffey *et al.,* 1982	100 to 150 µg/l blood lead levels (children)	NA	WHO, 2000	2000
UR	Chronic	1.2x10^{-5} (µg/m^3)$^{-1}$	Kidney tumour incidence	Azar *et al*., 1973	NA	NA	Cal EPA, 2011	1997

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-38 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
PHG	Chronic	0.17 [b]	One IQ point decrement in children	Lanphear et al., 2005; Carlisle and Dowling, 2006	NA	3	OEHHA, 2009; OEHHA, 2007	2007
RfD	Chronic	0.6	One IQ point decrement in children	Lamphear *et al*., 2005	NA	NA	JECFA, 2011	2011
IOC_{pop}	Chronic	1.85	Behavioural effects and learning disabilities in children	NA	NA	2	MOE, 1994	1994
TDI	Chronic	3.6	Increased blood lead concentration	Ziegler *et al.,* 1978; Ryu *et al.,* 1983	NOAEL	NA	RIVM, 2001	1999/2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.

NA Not available.

[a] Units of µg/kg/d unless otherwise noted.

[b] OEHHA, 2007 calculated a lower level of concern of 2.86 µg/day, which is primarily based on the review and slope factor work done by Carlisle and Dowling (2006) and their analysis of Lanphear et al. (2005), using a relative source contribution of 0.2, an uncertainty factor of 3 and a drinking water consumption rate of 1 L/day. The current chronic TRV was calculated by dividing by 16.5 kg for a toddler's body weight.

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A-2.1.26 Manganese

The 24-hour acute inhalation exposure limit of 0.4 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 0.05 µg/m^3 proposed by the US EPA IRIS (1993) were used for the assessment of manganese (Table A-39). The acute exposure limits were chosen as the only available values. The chronic exposure limit was chosen as the most conservative of all other available values.

The chronic oral exposure limit of 140 µg/kg/d proposed by US EPA IRIS (1996) was used for the oral assessment of manganese (Table A-40). The chronic exposure limit was chosen as it was the only available value.

Table A-39 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.4 (in TSP)	Adverse central nervous system effects	NA	NA	NA	MOE, 2012	NA
MRL	Chronic	0.3	Neurological effects	Roels *et al*., 1992	$BMCL_{10}$(ADJ): 33 µg/m^3	100	ATSDR, 2012	2012
ESL; Annual average	Chronic	0.2 (in PM10)	Health based	NA	NA	NA	TCEQ, 2014	2003
RfC	Chronic	0.05	Impairment of neurobehavioral function	Roels *et al*., 1992	LOAEL (HEC): 50 µg/m^3	1,000	US EPA IRIS, 1993	1993
REL	Chronic	0.09	Neurological effects	Roels *et al*., 1992	$BMCL_{0.5}$(ADJ): 72 µg/m^3	300	Cal EPA, 2008	2008
AAQO; Annual	Chronic	0.2	Health based	NA	NA	NA	AENV, 2013	2003
TCA	Chronic	0.15	Neurotoxic effects	Roels *et al*., 1992	$BMDL_5$: 30 µg/m^3	210	WHO, 2000	2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-40 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	140	CNS effects and impairment of neurobehavioral function	NRC, 1989; Freeland-Graves *et al*., 1987; WHO, 1973	NOAEL: 0.14 mg/kg/d	1	US EPA IRIS, 1996	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.27 Mercury (Inorganic)

The 24-hour acute inhalation exposure limit of 2 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 0.03 µg/m^3 also proposed by the Cal EPA (2008) were used for the assessment of inorganic mercury (Table A-41). The acute exposure limits were chosen as the only available values or based on the robustness of the supporting study data. The chronic exposure limit was chosen based on its level of conservatism and considering the robustness of the supporting information.

The chronic oral exposure limit of 0.3 µg/kg/d proposed by US EPA IRIS (1995) was selected for use in the assessment of inorganic mercury (Table A-42). This value was also endorsed by Health Canada (2010) and MOE (2011). The chronic oral exposure limit was selected based on the level of its conservatism and robustness of the study data.

Table A-41 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	2	Behavioural deficits	NA	NA	NA	MOE, 2012	NA
RfC	Chronic	0.3	Hand tremors, cognitive effects	Fawer *et al*., 1983; Piikivi and Tolonen, 1989; Piikivi and Hanninen, 1989; Piikivi, 1989; Ngim *et al*., 1992; Liang *et al*., 1993	LOAEL: 9 µg/m^3	30	US EPA IRIS, 1995a	1995
MRL	Chronic	0.2	Hand tremors, cognitive effects	Fawer *et al*., 1983	LOAEL: 26 µg/m^3	30	ATSDR, 1999	1999
TCA	Chronic	1.0	Tremors, kidney histopathology	Cardenas *et al*., 1993; WHO, 1991	NA	20	WHO, 2000	2000
ESL; annual average	Chronic	0.025	Health based	NA	NA	NA	TCEQ, 2014	2003
REL	Chronic	0.03	Hand tremors, cognitive effects	Fawer *et al*.,1983; Ngim *et al*., 1992; Piikivi, 1989; Piikivi and Hanninen, 1989; Piikivi and Tolonen, 1989	LOAEL: 9 µg/m^3	300	Cal EPA, 2008	2008
RfC	Chronic	0.09	Health based	NA	NA	NA	MOE, 2011	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-42 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	0.3	Nephrotoxicity	Andres, 1984; Bernaudin *et al*., 1981; Druet *et al*., 1978	LOAEL: 0.317 mg/kg/day	1,000	US EPA IRIS, 1995b	1995
RfD	Chronic	2.0	Kidney damage	NTP, 1993	NOAEL: 230 µg/kg/d	100	RIVM, 2001	2001
REL	Chronic	0.16	Kidney damage	NTP, 1993	NOAEL: 160 µg/kg/d	1,000	Cal EPA, 1999; 2008	1999; 2008
TDI	Chronic	0.3	Nephrotoxicity	Andres, 1984; Bernaudin *et al*., 1981; Druet *et al*., 1978	LOAEL: 3,170 µg/kg/d	1,000	Health Canada, 2010	2010
TDI	Chronic	0.57	Kidney damage	NTP, 1993	$BMDL_{10}$: 110 mg/kg/day	100	WHO, 2010	2010
TDI	Chronic	0.3	Kidney damage	Andres, 1984; Bernaudin *et al*., 1981; Druet *et al*., 1978	LOAEL: 317 mg/kg/day	1,000	MOE, 2011	1995

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.28 Molybdenum

The annual inhalation exposure limit of 3 µg/m^3 proposed by the TCEQ (2014) was used for the assessment of molybdenum (Table A-43). The exposure limit was chosen based on its level of conservatism. A health-based 24-hour exposure limit was not available.

The chronic oral exposure limit of 5 µg/kg/d proposed by US EPA IRIS (1994) was used for the oral assessment of molybdenum (Table A-44). The exposure limit was chosen as it was the only available value.

Table A-43 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; annual average	Chronic	3 (in PM10)	Health based	NA	NA	NA	TCEQ, 2014	2010
TCA	Chronic	12	Body weight effects	NA	NOAEC: 12,000 µg/m³	1,000	RIVM, 2001	1999/ 2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-44 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	5	Increased uric acid levels	Koval'skiy *et al*., 1961	LOAEL: 0.14 mg/kg/d	30	US EPA IRIS, 1993	1993

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.29 Nickel

The 24-hour acute inhalation exposure limit of 0.2 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 0.014 µg/m^3 also proposed by the Cal EPA (2012) were used for the assessment of nickel (Table A-45). The acute and chronic exposure limits were chosen as the only available values or based on their level of conservatism.

The UR of 2.6x10^{-4} (µg/m^3)$^{-1}$ proposed by Cal EPA (2011) was used for the carcinogenic assessment of nickel. The UR was selected based on the scientific defensibility of the study data.

The chronic oral exposure limit of 11 µg/kg/d proposed by Cal EPA (2012) was used in the assessment of nickel (Table A-46). The chronic oral exposure limit was selected based on its level of conservatism relative to other available values. Further, Health Canada (2010) endorsed the use of this value.

Table A-45 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.2 (TSP)	Carcinogenic and non-carcinogenic effects	CSTEE, 2001	NA	NA	MOE, 2011a	2011
MRL; 15 to 364 days	Intermed iate	0.2	Chronic active inflammation	NTP, 1996	NOAEL (HEC): 5.2 µg/m^3	30	ATSDR, 2005	2005
TC	Chronic	0.02 (nickel oxide)	Increases in lung granulocytes and multi-nucleated counts	Spiegelberg *et al*., 1984	LOAEL: 25 µg/m^3	1000	Health Canada, 2010	2010
TC	Chronic	0.018 (Nickel subsulph ide)	Respiratory track effects; Alveolar macrophages, hyperplasia	Benson *et al.,* 1990; Dunnick *et al*., 1989	NOAEL (mice), LOAEL (rat): 100 µg/m^3	1000	Health Canada, 2010	2010
TC	Chronic	0.0035 (Nickel sulphate)	Respiratory effects: lesions in lung, nasal epithelium, others	Dunnick *et al*., 1989	LOAEL: 20 µg/m^3	1000	Health Canada, 2010	2010
TC	Chronic	0.018 (metallic nickel)	Respiratory effects, morphological and biological effects	Johansson *et al*., 1983	LOAEL (ADJ): 0.018 mg/m^3	1000	Health Canada, 2010	2010
AAQC	Chronic	0.04 (TSP)	Carcinogenic and non-carcinogenic effects	CSTEE, 2001	NA	NA	MOE, 2011a	2012
RfC	Chronic	0.06	NA	NA	NA	NA	MOE, 2011b	2011
REL	Chronic	0.014 (nickel and compoun ds)	Pathological changes to respiratory system and hematologic system	NTP, 1994	$BMDL_{05}$ (HEC): 1.4 µg/m^3	100	Cal EPA, 2012	2012
REL	Chronic	0.02 (nickel oxide)	Pathological changes in lung: active pulmonary	NTP, 1994	$BMDL_{05}$ (HEC): 2.0 µg/m^3	100	Cal EPA, 2012	2012

Table A-45 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
			inflammation, alveolar proteinosis					
ReV	Chronic	0.23	Chronic active lung inflammation and associated lesions	NTP, 1996	POD (HEC): 7 µg/m^3	30	TCEQ, 2011	2011
MRL	Chronic	0.09	Respiratory effects	NTP, 1996	NOAEL (HEC): 2.7 µg/m^3	30	ATSDR, 2005	2005
TCA	Chronic	0.05	Incidence of alveolar macrophage activity	NA	NOAEC: 30 µg/m^3	NA	RIVM, 2001	1999/ 2000
UR	Chronic	$1.3x10^{-3}$ $(µg/m^3)^{-1}$	Lung cancer	Doll *et al*., 1990	NA	NA	Health Canada, 2010	2010
UR	Chronic	$7.1x10^{-4}$ $(µg/m^3)^{-1}$	Lung cancer	Doll *et al.,* 1990	NA	NA	Health Canada, 2010	2010
UR	Chronic	$2.4x10^{-4}$ $(µg/m^3)^{-1}$	Lung tumour	Enterline and Marsh, 1982; Chovil *et al*., 1981; Peto *et al*., 1984; Magnus *et al*., 1982	NA	NA	US EPA IRIS, 1991a	1991
UR	Chronic	$4.8x10^{-4}$ $(µg/m^3)^{-1}$	Lung tumour	Enterline and Marsh, 1982; Chovil *et al*., 1981; Peto *et al*., 1984; Magnus *et al*., 1982	NA	NA	US EPA IRIS, 1991b	1991
UR	Chronic	$1.7x10^{-4}$ $(µg/m^3)^{-1}$	Lung cancer	Enterline and Marsh, 1982; Grimsrud *et al*., 2003	NA	NA	TCEQ, 2011	2011
UR	Chronic	$2.6x10^{-4}$ $(µg/m^3)^{-1}$	Lung and nasal cancer incidence	Chovil *et al.,* 1981; Roberts *et al*., 1984; Muir *et al*., 1985; ICNCM, 1990	NA	NA	Cal EPA, 2011	2009
UR	Chronic	$3.8x10^{-4}$ $(µg/m^3)^{-1}$	NA	NA	NA	NA	WHO, 2000	2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-46 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
TDI	Chronic	11	Post-implantation perinatal lethality	SLI, 2000	NOAEL: 1.1 mg/kg/day	100	Health Canada, 2010	2010
TDI	Chronic	12	Eczema on hands	NA	NA	NA	WHO, 2005	2005
REL	Chronic	11	Perinatal mortality	NiPERA, 2000a;b	NOAEL: 1.1 mg/kg/day	100	Cal EPA, 2012	2012
RfD	Chronic	20	No adverse effects on body weight and organ weight	Ambrose *et al*., 1976	NOAEL: 5 mg/kg/day	300	US EPA IRIS, 1996	1996
TDI	Chronic	50	NA	NA	NOAEL: 5 mg/kg/day	NA	RIVM, 2001	2001

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.30 Nitrogen Dioxide (NO_2)

The 24-hour acute inhalation exposure limit of 200 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 40 µg/m^3 proposed by the WHO (2006) were used for the assessment of nitrogen dioxide (Table A-47). These values were chosen based on their level of conservatism and considering the date of their most recent validation.

Table A-47 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	200 [b]	Respiratory tract irritation	NA	NA	NA	MOE, 2012	NA
NAAQO MAL; 24-hour	Acute	200	Health based	NA	NA	NA	CCME, 1999	published 1975; reviewed 1989
MDL; Annual Average	Chronic	60	Health based	NA	NA	NA	CCME, 1999	published 1975; reviewed 1989
NAAQS; Annual Average	Chronic	100	Health based	NA	NA	NA	US EPA, 2010	1993
AQG; Annual Average	Chronic	40	Respiratory effects	NA	NA	NA	WHO, 2006	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.

[a] Units of µg/m^3 unless otherwise noted.

[b] Exposure limit of NOx (Sum of NO and NO_2).

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A-2.1.31 Ozone

The 8-hour acute inhalation exposure limit of 100 µg/m^3 proposed by WHO (2005) was used for the assessment of ozone (Table A-48). The acute exposure limit was chosen based on the level of conservatism relative to the other available values.

Table A-48 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
Acute; 8-hour	Acute	0.075 ppm (160 µg/m^3)	NA	NA	NA	NA	US EPA, 2008	2008
Acute; 8-hour	Acute	100	Estimated 1-2% increase in daily mortality (based on findings of daily time series studies)	NA	NA	NA	WHO, 2005	2005

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.32 Carcinogenic Polycyclic Aromatic Hydrocarbons.

As indicated in Health Canada (2010), as well as most other regulatory guidance, including the US EPA (1993), the assessment of risks related to exposures to carcinogenic PAHs is primarily conducted through the use of potency or toxicity equivalence factors (PEF or TEF). TEFs allow large groups of compounds with a common mechanism of action such as PAHs to be assessed when limited data is available for all but one of the compounds (*i.e.*, B(a)P). Through this approach, exposures to each of the carcinogenic PAHs are adjusted by their carcinogenic potency relative to B(a)P. These potency-adjusted exposures can then be summed to provide an overall exposure to the group of carcinogenic PAHs, based on B(a)P as the primary surrogate.

Methods used in the determination of the relative potency factors for individual PAHs vary according to the information available for the compound and the agency conducting the evaluation. Consequently, many sets of TEFs for carcinogenic PAHs have been developed over the last decade (CCME, 2008). For example, relative potencies developed by the US EPA (1993) for seven individual PAHs were derived in increments of order of magnitude by comparison to B(a)P. According to US EPA methods, only the results of carcinogenicity bioassays in which B[a]P and individual PAHs were evaluated using the same protocol and within the same time frame were considered, and maximum likelihood estimates from a two-stage model were used for comparison. Due to limitations in the derivation owing to consideration of only a small subset of the PAHs and reliance on a single oral pathway for B[a]P exposure (US EPA, 2000), the US EPA recommended that the values only be used in the assessment of carcinogenicity and not when evaluating inhaled PAH mixtures (WHO, 1998). Subsequent derivations of relative potency values have been largely intended to address carcinogenic effects associated with inhalation of PAHs and/or direct application trials (CCME, 2008), and are most often based on the modeling of datasets consisting of all available appropriate assays to determine a central tendency estimate. The central tendency estimate is then used in potency comparison between B(a)P and the specific PAH (WHO, 1998). In the derivation of equivalence factors by Cal EPA (2005) preference was given to tumour data from the inhalation of PAHs, next only to cancer potency values specific to an individual PAH derived from a health effects evaluation and quantitative risk assessment (Cal EPA, 2005). Potency values recommended by Health Canada (2010) are primarily those adopted from CCME (2008) and EEI (2006).

The toxic equivalents approach was adopted in the current assessment. Table A-49 shows the TEF of each of the carcinogenic PAH evaluated in the current assessment, and the respective TEFs selected for use with this approach. Potency values recommended by Health Canada (2010) were selected when available. TEFs recommended by WHO (1998) and RIVM (2001) were considered in the absence of equivalence factors from Health Canada.

Table A-49 Relative Potency of Individual PAHs Compared with Benzo(a)pyrene		
PAH	***Toxic Equivalency Factor***	***Agency/Source***
Acenaphthylene	0.01	RIVM, 2001
Acenaphthene	0.001	RIVM, 2001
Benzo(a)anthracene	0.1	Health Canada, 2010
Benzo(b)fluoranthene	0.1	Health Canada, 2010
Benzo(k)fluoranthene	0.1	Health Canada, 2010
Benzo(ghi)perylene	0.01	Health Canada, 2010
Benzo(a)pyrene	1	Health Canada, 2010
Benzo(e)pyrene	0.01	WHO, 1998
Chrysene	0.01	Health Canada, 2010
Dibenzo(a,c)anthracene	0.1	WHO, 1998
Dibenzo(a,h)anthracene	1	Health Canada, 2010
Fluoranthene	0.001	Health Canada, 2010
Indeno(1,2,3 – cd)pyrene	0.1	Health Canada, 2010
Perylene	0.001	WHO, 1998
Phenanthrene	0.001	Health Canada, 2010
Pyrene	0.001	RIVM, 2001

The UR of $1.1x10^{-3}$ $(\mu g/m^3)^{-1}$ proposed by the Cal EPA (2011) was used for the assessment of carcinogenic PAHs (as toxic equivalents of benzo(a)pyrene) (Table A-50). This value was adopted and endorsed by the MOE (2011).

The SF of $7.3x10^{-3}$ $(\mu g/kg/d)^{-1}$ proposed by US EPA IRIS (1994) was used for the assessment of carcinogenic PAHs (as toxic equivalents of benzo(a)pyrene) (Table A-51). The SF was selected based on its level of conservatism relative to other available values.

Table A-50 Inhalation Toxicity Reference Values (PAHs as Benzo(a)pyrene Toxic Equivalents)								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
UR	Chronic	$8.7x10^{-2}$	Incidence of lung cancer	WHO, 1998	NA	NA	WHO, 2000	2000
UR	Chronic	$3.1x10^{-5}$	Respiratory tract tumours	Thyssen *et al*., 1981	NA	NA	Health Canada, 2010	2010
UR	Chronic	$1.1x10^{-3}$	Respiratory tract tumours	Thyssen *et al*., 1981	NA	NA	Cal EPA, 2011	2009
UR	Chronic	$1.1x10^{-3}$	NA	Cal EPA, 2011	NA	NA	MOE, 2011	2011

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $(\mu g/m^3)^{-1}$ unless otherwise noted.

Table A-51 Oral Toxicity Reference Values (PAHs as Benzo(a)pyrene Toxic Equivalents)								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
SF	Chronic	$7.3x10^{-3}$	Forestomach, squamous cell papillomas and carcinomas	Neal and Rigdon, 1967; Rabstein *et al*., 1973; Brune *et al*., 1981	NA	NA	US EPA IRIS, 1994	1994
SF	Chronic	$7.3x10^{-3}$	NA	NA	NA	NA	MOE, 2011	2011
SF	Chronic	$2.3x10^{-3}$	Gastric tumours (mostly squamous cell papillomas, with a few carcinomas)	Neal and Rigdon, 1967	NA	NA	Health Canada, 2010	2010
SF	Chronic	$1.2x10^{-2}$	Gastric tumours (paillomas and squamous cell carcinomas)	Neal and Rigdon, 1967	NA	NA	Cal EPA, 2011	2009
SF	Chronic	$2x10^{-4}$	Tumours in liver and forestomach	Kroese *et al*., 1999	NA	NA	RIVM, 2001	2001

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $(\mu g/kg/d)^{-1}$ unless otherwise noted.

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A-2.1.33 Particulate Matter ($PM_{2.5}$)

The 24-hour acute inhalation exposure limit of 27 µg/m³ and the annual inhalation exposure limit of 8.8 µg/m³, both proposed by the CCME (2012) were used for the assessment of $PM_{2.5}$ (Table A-52). These values were chosen based on their level of conservatism and considering the date of their most recent validation.

Table A-52 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	30	NA	NA	NA	NA	MOE, 2012	NA
Reference Level; 24-hour	Acute	15	Health based	NA	NA	NA	CCME, 1999	published 1998
CAAQS; 24-hour	Acute	27[b]	Respiratory tract irritation	NA	NA	NA	CCME, 2012	2012
NAAQS; 24-hour	Acute	35	Mortality and morbidity	NA	NA	NA	US EPA, 2010	NA
AQG; 24-hour	Acute	25	NA	NA	NA	NA	WHO, 2006	NA
CAAQS	Chronic	8.8[b]	Cardiopulmonary and lung cancer mortality increase	NA	NA	NA	CCME, 2012	2012
NAAQS	Chronic	12	Various adverse health effects; Increased risk of mortality, cardiovascular-related effects, respiratory morbidity	NA	NA	NA	US EPA, 2010	NA
AQG	Chronic	10	Lowest levels at which total, cardiopulmonary and lung cancer mortality has been shown to increase	NA	NA	NA	WHO, 2006	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m³ unless otherwise noted.
[b] Compliance by 2020

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A-2.1.34 Particulate Matter (PM_{10})

The 24-hour acute inhalation exposure limit of 50 µg/m³ and the annual inhalation exposure limit of 20 µg/m³, both proposed by the WHO (2006) were used for the assessment of PM_{10} (Table A-53). The 24-hour AQG recommended by WHO (2006) is consistent with the MOE (2012) 24-hour AAQC. These values were chosen based on their level of conservatism and considering the date of their most recent validation, and in the case of the annual exposure limit, the absence of other available values.

Table A-53 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC (interim); 24-hour	Acute	50	NA	NA	NA	NA	MOE, 2012	NA
Reference Level; 24-hour	Acute	25	Health based	NA	NA	NA	CCME, 1999	published 1998
NAAQS; 24-hour	Acute	150	Cardiovascular and respiratory hospital admissions and respiratory symptoms	NA	NA	NA	US EPA, 2010	NA
AQG; 24-hour	Acute	50	Respiratory tract irritation	NA	NA	NA	WHO, 2006	NA
AQG	Chronic	20	Lowest levels at which total, cardiopulmonary and lung cancer mortality has been shown to increase	NA	NA	NA	WHO, 2006	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m³ unless otherwise noted.

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WHO. 2006. Air Quality Guidelines: Global Update 2005. Particulate matter, ozone, nitrogen dioxide and sulphur dioxide. World Health Organization. ISBN 92 890 2192 6.

A-2.1.35 Total PCBs

The 24-hour acute and chronic inhalation exposure limits of 0.15 µg/m^3 and 0.035 µg/m^3, respectively, proposed by the MOE (2012), were used for the non-cancer assessment of total PCBs (Table A-54). The acute exposure limits were chosen as the only available values. The chronic exposure limit was selected based on its level of conservatism relative to MOE (2011) and considering the date of publication.

The UR of 1.0x10^{-4} (µg/m^3)$^{-1}$ proposed by the US EPA IRIS (1997) was the only available UR and was used for the carcinogenic assessment of total PCBs.

The chronic oral exposure limit of 2.0x10^{-3} (µg/kg/d)$^{-1}$ proposed by US EPA IRIS (1996) was used for the oral non-carcinogenic assessment of total PCBs (Table A-55). The chronic oral exposure limit was selected based on the robustness of the study data. Further, ATSDR (2000), WHO (2003), and RIVM (2001) endorsed the same value with the use of the same study data.

The SF of 2.0x10^{-3} (µg/kg/d)$^{-1}$ proposed by US EPA IRIS (1997) was used for the oral carcinogenic assessment of PCBs. The SF was selected as it was the only available value.

Table A-54 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	0.15	Systemic effects	NA	NA	NA	MOE, 2012	NA
TCA	Chronic	0.5	marginal effects on various experimental animals	NA	LOAEC: 1,500 µg/m^3	300	RIVM, 2001	1999/ 2000
RfC	Chronic	0.5	NA	NA	RIVM, 2001	NA	MOE, 2011	NA
AAQC; annual	Chronic	0.035	Systemic effects	NA	NA	NA	MOE, 2012	NA
ESL; annual average	Chronic	0.01	Health based	NA	NA	NA	TCEQ, 2014	2003
UR	Chronic	1.0x10^{-4} (µg/m^3)$^{-1}$	Liver adenomas and carcinomas	NA	NA	NA	US EPA IRIS, 1997	1996

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-55 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	0.02	Immunological effects	Arnold *et al.*, 1993a; 1993b; Tryphonas *et al.*, 1989; 1991a; 1991b	LOAEL: 0.005 mg/kg/day	300	US EPA IRIS, 1996	1996
MRL	Chronic	0.02	Immunological effects	Arnold *et al.*, 1993a; 1993b; Tryphonas *et*	LOAEL: 0.005 mg/kg/day	300	ATSDR, 2000	2000

Table A-55 Oral Toxicity Reference Values								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
				al., 1989; 1991a; 1991b				
TDI	Chronic	0.02	Immunological effects	Arnold *et al.,* 1993a; 1993b; Tryphonas *et al.*, 1989; 1991a; 1991b	LOAEL: 0.005 mg/kg/day	300	WHO, 2003	2003
TDI	Chronic	0.02	Immunological effects	Arnold *et al.,* 1993a; 1993b; Tryphonas *et al.*, 1989; 1991a; 1991b	LOAEL: 0.005 mg/kg/day	270	RIVM, 2001	2001
TDI	Chronic	0.13	None observed	Bowman *et al.,* 1981.	NOEL: 0.013 mg/kg/day	100	Health Canada, 2010	2010
RfD	Chronic	0.02	NA	NA	NA	NA	MOE, 2011	2000
SF	Chronic	$2.0x10^{-3}$ $(\mu g/kg/d)^{-1}$	Liver hepatocellular adenomas, carcinomas	Brunner *et al*., 1996; Norback and Weltman, 1985	NA	NA	US EPA IRIS, 1997	1997

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.36 Selenium

The 24-hour acute inhalation exposure limit of 10 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 0.2 µg/m^3 proposed by the TCEQ (2014) were used for the assessment of selenium (Table A-56). The acute exposure limits were chosen as the only available values. The chronic exposure limit was chosen as it was the most conservative value and because the Cal EPA (2001) value was extrapolated from the ingestion pathway.

The chronic oral exposure limit of 5 µg/kg/d proposed by US EPA IRIS (1991b) was used for the assessment of selenium (Table A-57). The chronic exposure limit was chosen as it was the most conservative value. Further, MOE (2011), ATSDR (2003), and Cal EPA (2001) endorses the value.

Table A-56 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	10	Respiratory irritation	NA	NA	NA	MOE, 2012	NA
REL	Chronic	20 [b]	Selenosis	US EPA IRIS, 1991a; Yang *et al.,* 1989 a,b	0.005 mg/kg/day	3	Cal EPA, 2001	NA
ESL; Annual Average	Chronic	0.2	Eye and upper respiratory tract irritation	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.
[b] Derived using an inhalation extrapolation factor of 3,500 µg/m^3 per mg/kg-day (*i.e.,* assuming a body weight of 70 kg, an inhalation rate of 20 m^3/day)

Table A-57 Oral Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
TDI	Chronic	5.7 (+20 years)	Selenium in breast milk and selenosis	IOM, 2002; Shearer and Hadjimarkos 1975; Yang and Zhou 1994b	Upper intake of 0.4 mg/kg for adults	2	Health Canada, 2010	NA
RfD	Chronic	5	Selenosis	Yang *et al.,* 1989b	NOAEL: 0.015 mg/kg /day	3	US EPA IRIS, 1991b	1991
RfD	Chronic	5	NA	NA	NA	NA	MOE, 2011	NA
MRL	Chronic	5	Selenosis	Yang and Zhou 1994	NOAEL: 0.015 mg/kg /day	3	ATSDR, 2003	2003
REL	Chronic	5	Selenosis	US EPA IRIS, 1991b;	NOAEL: 0.015 mg/kg /day	3	Cal EPA, 2001	NA
Upper tolerable intake level	Chronic	6	NA	NA	NA	NA	WHO, 2011	NA

Table A-57 Oral Toxicity Reference Values								
Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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Yang, G., and Zhou, R.H. 1994. Further observations on the human maximum safe dietary selenium intake in a seleniferous area of China. J Trace Elem Electrolytes Health Dis. 8(3-4):159-165. Cited in: ATSD, 2003; Health Canada, 2010.

A-2.1.37 Strontium

The 24-hour acute inhalation exposure limit of 120 µg/m^3 proposed by the MOE (2012) and the annual inhalation exposure limit of 2 µg/m^3 proposed by the TCEQ (2014) were used for the assessment of strontium (Table A-58). The acute and chronic exposure limits were chosen as they were the only available values.

The chronic oral exposure limit of 600 µg/kg/d proposed by US EPA IRIS (1996) was used for the oral assessment of strontium (Table A-59). The chronic exposure limit was chosen as it was the only available value.

Table A-58 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	120	Respiratory irritation based on particulate levels	NA	NA	NA	MOE, 2012	NA
ESL; Annual Average	Chronic	2 (in PM_{10})	Respiratory inflammation	NA	NA	NA	TCEQ, 2014	2003

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-59 Oral Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	600	Rachitic bone	Storey, 1961; Marie *et al*., 1985; Skoryna, 1981	NOAEL: 190,000 µg/kg/d	300	US EPA IRIS, 1996	1996

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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A-2.1.38 Sulphur Dioxide (SO_2)

The 24-hour acute inhalation exposure limit of 275 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 29 µg/m^3 were used for the assessment of sulphur dioxide (Table A-60). The 1-hour and annual exposure limits were chosen based on their level of conservatism.

While the WHO (2006) 24-hour exposure limit is a more conservative value than the MOE (2012) value chosen for use in this assessment, some concerns were raised regarding its scientific defensibility. The WHO (2006) 24-hour value of 20 µg/m^3 was derived using a precautionary approach that considered various factors, including the uncertainty in SO_2 causality, the practical difficulty in attaining levels associated with no effect, the need for greater public protection than the previous guideline, and the assumption that future SO_2 exposures will be reduced with reduced concentrations (WHO, 2005a,b). It must be recognized that the SO_2 air quality guidelines recommended by WHO (2005a,b) may be very difficult, if not practically impossible, for some countries to attain; these guidelines represent desirable levels and in some instances achieving these guidelines requires a step-wise progression that may take years.

Further, the basis of the CCME (1999) 24-hour exposure limit, derived in 1974, could not be ascertained. Although no publically available information was identified describing the derivation of the MOE SO_2 24-hour AAQC, it is our understanding the MOE standards for SO_2 (*i.e.,* 24-hour and annual) were not developed using individual toxicological endpoints that vary with exposure duration, but rather converted (using meteorological based conversion factors) from the 1-hour AAQC. The MOE 1-hour AAQC is consistent with the Cal EPA (2008) 1-hour SO_2 REL of 0.25 ppm (690 µg/m^3) that was designed to protect sensitive individuals (*i.e.*, exercising asthmatics) from lower respiratory effects following acute exposure. A NOAEL for sensitive individuals of 0.25 ppm SO_2 (from multiple studies) was adopted as the Cal EPA acute REL for SO_2 that would not result in discomforting respiratory effects among sensitive individuals after a 1-hour exposure event. Accordingly, the MOE (2012) 24-hour exposure limit was considered appropriate for this assessment.

Table A-60 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
AAQC; 24-hour	Acute	275	Respiratory tract irritation	NA	NA	NA	MOE, 2012	NA
AQG; 24-hour	Acute	20	NA	NA	NA	NA	WHO, 2006	NA
NAAQO MDL; 24-hour	Acute	150	Health based	NA	NA	NA	CCME, 1999	published 1974; reviewed 1989
MRL; 14 days or less	Acute	26	Respiratory irritation	Sheppard *et al*., 1981	LOAEL: 0.1 ppm (262 µg/m^3)	9	ATSDR, 1998	NA
NAAQO MDL	Chronic	30	Health based	NA	NA	NA	CCME, 1999	published 1974; reviewed 1989
NAAQO MDL	Chronic	29	Respiratory inflammation	NA	NA	NA	Health Canada, 2006	NA

Table A-60 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value*** [a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
AAQC	Chronic	55	NA	NA	NA	NA	MOE, 2012	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.39 Tetrachloroethylene

The 24-hour acute inhalation exposure limit of 360 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 40 µg/m^3 proposed by the US EPA IRIS (2012) were used for the assessment of tetrachloroethylene (Table A-61). The acute exposure limit was chosen as it was the most conservative value. The chronic inhalation exposure limit of 40 µg/m³ derived by US EPA IRIS (2012) was selected in the assessment given that it was the most conservative and scientifically defensible value.

The IUR of 2.6 x 10-7 (µg/m^3)$^{-1}$ derived by US EPA IRIS (2012) was selected in the assessment due to its scientific defensibility.

Table A-61 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
REL; 1-hour	Acute	20,000	Loss of normal coordination in addition to eye, nose and throat irritation, headache, and light-headedness	Stewart *et al*., 1970	LOAEL (ADJ): 1,200 mg/m^3 (1,200,000 µg/m^3)	60	Cal EPA, 2008	2008
ReV; 1-hour	Acute	6,800	Latency of pattern reversal visual-evoked potential and performance deficits for eye-hand coordination	Altmann *et al*., 1992	NOAEL: 10 ppm (68,000 µg/m^3)	10	TCEQ, 2008	2008
MRL; 14 days or less	Acute	1,400	Pattern reversal visual-evoked potential latencies	Altmann *et al*., 1992	NOAEL (ADJ): 2 ppm (14,000 µg/m^3)	10	ATSDR, 1997	1997
AAQC; 24-hour	Acute	360	Central nervous system depression and respiratory system effects	Health Canada, 1996	LOAEL of 678 mg/m^3	1,000	MOE, 2012	2012
RfC	Chronic	40	Neurotoxicity (human)	Echeverria *et al*., 1995; Cavalleri *et al*., 1994	LOAEL: 56; LOAEL: 15	1,000	US EPA IRIS, 2012	2012
ReV	Chronic	370	Behavioural effects: increased reaction times	Ferroni *et al*., 1992	POD (HEC): 5.4 ppm (37,000 µg/m^3)	100	TCEQ, 2008	2008
MRL	Chronic	270	Prolonged reaction times	Ferroni *et al*., 1992	LOAEL: 15 ppm	100	ATSDR, 1997	1997
AQG	Chronic	250	Kidney effects	NA	LOAEL (ADJ): 24.3 mg/m^3 (24,300 µg/m^3)	100	WHO, 2000	2000
RfC	Chronic	250	NA	NA	NA	NA	MOE, 2011	2011

Table A-61 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
TCA	Chronic	250	NA	NA	NA	NA	RIVM, 2001	2001
REL	Chronic	35	NA	NA	NA	NA	Cal EPA, 1991	1991
TC	Chronic	360	Nephrotoxic, hepatotoxic, lung congestion, mononuclear cell leukemia	NTP, 1986	LOAEL (ADJ): 363 mg/m^3 (363,000 µg/m^3)	1000	Health Canada, 2010	2010
Unit risk	Chronic	2.6 x10^{-7} (µg/m^3)$^{-1}$	Hepatocellular adenomas or carcinomas (mice)	JISA , 1993	NA	NA	US EPA IRIS, 2012	2012
Unit risk	Chronic	3.8 x 10^{-7} (µg/m^3)$^{-1}$	Increase in incidences of hepatocellular carcinomas	NTP, 1986	NA	NA	TCEQ, 2008	2008
Unit risk	Chronic	5.9 x 10^{-6} (µg/m^3)$^{-1}$	Hepatocellular adenoma and carcinoma incidences	NTP, 1986	NA	NA	Cal EPA, 2005	2005
Unit risk	Chronic	5.2 x 10^{-6} (µg/m^3)$^{-1}$	Mononuclear cell leukemia; Benign and malignant liver tumours	Nagano *et al*., 1998a; Nagano *et al*., 1998b	NA	NA	WHO, 2006	2006

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.40 Toluene

The 24-hour acute inhalation exposure limit of 3,800 µg/m^3 proposed by the ATSDR (2000), and the annual inhalation exposure limit of 5,000 µg/m^3 proposed by the US EPA IRIS (2005) were used for the assessment of toluene (Table A-62). The acute exposure limit was chosen as it was the most conservative value. The chronic exposure limit of 5,000 µg/m^3 was selected in the assessment as it represents the most recent analysis of the available scientific literature. The MOE (2011) has recommended this value of 5,000 µg/m^3 that was derived by the US EPA IRIS (2005) and has endorsed it as an RfC.

Table A-62 Inhalation Toxicity Reference Values

Type	*Duration*	*Value* [a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; 1-hour	Acute	4,500 (HQ = 0.3)	Eye and nose irritation; increased occurrence of headache, dizziness, and intoxication	Andersen *et al*., 1983	NOAEL: 40 ppm (150,000 µg/m^3)	10	TCEQ, 2014	2008
ReV; 1-hour	Acute	15,000	Eye and nose irritation; increased occurrence of headache, dizziness, and intoxication	Andersen *et al*., 1983	NOAEL: 40 ppm (150,000 µg/m^3)	10	TCEQ, 2014	2008
REL; 1-hour	Acute	37,000	Headache, dizziness, slight eye and nose irritation	Andersen *et al*., 1983	NOAEL (ADJ): 98 ppm (370,000 µg/m^3)	10	Cal EPA, 2008	2008
MRL; 24-hour	Acute	3,800	Neurological effects (human)	Andersen *et al*., 1983	NOAEL (ADJ): 1 ppm (38,000 µg/m^3)	10	ATSDR, 2000	2000
RfC	Chronic	5,000	Neurological effects in occupationally-exposed workers	Multiple human studies	NOAEL (ADJ): 46,000 µg/m^3	10	US EPA IRIS, 2005	2005
RfC	Chronic	5,000	NA	US EPA IRIS, 2005	NA	NA	MOE, 2011	NA
MRL	Chronic	300	Alcohol- and age-adjusted colour vision impairment	Zavalic *et al*., 1998	LOAEL (ADJ): 8 ppm (30,000 µg/m^3)	100	ATSDR, 2000	2000
TC	Chronic	3,750	Increased relative liver and kidney	Andersen *et al*., 1983	NOAEL (ADJ): 37.5 mg/m^3	10	Health Canada, 2010	2010

Table A-62 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value*** [a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
			weight neurotoxic, irritation of the respiratory tract		(37,500 µg/m^3)			
REL	Chronic	300	Decreased brain (subcortical limbic area) weight, altered dopamine receptor (caudate-putamen) binding	Hillefors-Berglund *et al*., 1995; Foo *et al*., 1990	NOAEL (ADJ): 7 ppm (26,000 µg/m^3)	100	Cal EPA, 2008	2000
ReV	Chronic	4,100	Colour vision impairment	Zavalic *et al*., 1998	NOAEL: 11 ppm (41,000 µg/m^3)	10	TCEQ, 2014	2008
TCA	Chronic	400	Neurological effects	NA	NA	NA	RIVM, 2001	1999/2000

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

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A-2.1.41 Trichloroethylene

The 24-hour acute inhalation exposure limit of 12 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 2 µg/m^3 proposed by the US EPA (2011) were used for the assessment of trichloroethylene (Table A-63). The acute exposure limit was chosen as it was the most conservative value. The chronic exposure limit of 2 µg/m^3 derived by US EPA IRIS (2011) was selected as it was the most scientifically defensible and conservative value.

The IUR of 4.1 x 10-6 (µg/m^3)$^{-1}$ derived by US EPA IRIS (2011) was selected for use in this assessment as it was the most scientifically defensible and conservative value.

Table A-63 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; 1-hour	Acute	540	NA	NA	NA	NA	TCEQ, 2013	2003
AAQC; 24-hour	Acute	12	Central nervous system, eye, and respiratory system effects	NA	NA	NA	MOE, 2012	NA
MRL;< 14 days	Acute	10,000 (2ppm)	Mild subjective neurological effects (eye and throat irritation, headache, fatigue, drowsiness)	Stewart *et al.,* 1970	LOAEL(ADJ): 58 ppm (~300,000 µg/m^3)	30	ATSDR, 1997	1997
AAQC	Chronic	2.3	Health	NA	NA	NA	MOE, 2012	NA
RfC	Chronic	40	NA	NA	NA	NA	MOE, 2011	NA
RfC	Chronic	2	Decreased thymus weights and fetal heart malformations (mouse)	Keil *et al.,* 2009; Johnson *et al.,* 2003	Keil *et al*., 2009; $HEC_{99,\ LOAEL}$: 0.19 mg/m^3 Johnson *et al.,* 2003; $HEC_{99,\ BMDL01}$ 0.021	Keil *et al*., 2009; 100 Johnson *et al.,* 2003; 30	US EPA IRIS, 2011	2011
MRL	Chronic	2	NA	US EPA, 2011	NA	NA	ATSDR, 2013	NA
REL	Chronic	600	Neurotoxicological effects (drowsiness, fatigue and headache) and eye irritation	Vandervort and Polnkoff, 1973	LOAEL(ADJ): 11.4 ppm (~60,000 µg/m^3)	100	Cal EPA, 2008	NA
pTCA	Chronic	200	Hypatotoxicity	Kjellstrand *et al.,* 1983	LOAEL: 200 mg/m^3 (200,000 µg/m^3)	1,000	RIVM, 2001	NA
ESL	Chronic	54	NA	NA	NA	NA	TCEQ, 2013	NA

Table A-63 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
Unit risk	Chronic	6.1 x 10^{-7} ($\mu g/m^3$)$^{-1}$	Pulmonary adenomas and adenocarcinomas (mice); testicular tumours (rats)	Maltoni *et al.*, 1986 ; 1988 ; Fukuda *et al.*, 1983 ; NTP, 1988	NA	NA	Health Canada, 2010	NA
Unit risk	Chronic	2.0 x 10^{-6} ($\mu g/m^3$)$^{-1}$	Hepatocellular adenomas and carcinomas (males); lung adenocarcinomas and malignant lymphomas (females)	Bell *et al.*, 1978; Henschler *et al.*, 1980; Fukuda *et al.*, 1983; Maltoni *et al.*, 1986	NA	NA	Cal EPA, 2009	NA
Unit risk	Chronic	2.0 x 10^{-6} ($\mu g/m^3$)$^{-1}$	NA	NA	NA	NA	MOE, 2011	NA
Unit risk	Chronic	4.3 x 10^{-7} ($\mu g/m^3$)$^{-1}$	Leydig-cell tumours in testes	Maltoni *et al.*, 1986	NA	NA	WHO, 2000	NA
Unit risk	Chronic	4.1 x 10^{-6} ($\mu g/m^3$)$^{-1}$	Renal cell carcinoma (human)	Charbotel *et al.*, 2006	NA	NA	US EPA IRIS, 2011	2011

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.42 Vinyl Chloride

The 24-hour acute inhalation exposure limit of 1 µg/m^3 proposed by the MOE (2012), and the annual inhalation exposure limit of 60 µg/m^3 proposed by TCEQ (2009) were used for the assessment of vinyl chloride (Table A-64). The acute exposure limit was chosen as it was the most conservative value. The chronic exposure limit was selected for use in the current assessment given that it was based on a more recent study and it was a more conservative exposure limit.

The UR of 7.8 x 10^{-5} (µg/m^3)$^{-1}$ proposed by the Cal EPA (2009) was used for the carcinogenic assessment of vinyl chloride. Cal EPA (2009) considered this value to provide adequate health protective estimates of human unit risks, which represent the 95% upper confidence limits for risk calculations. The IUR value of 7.8 x 10^{-5} (µg/m^3)$^{-1}$ derived by Cal EPA (2009) was selected for use in this assessment given that it was the most conservative value.

Table A-64 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ReV;1-hour	Acute	68,000	Mild headache and dryness of their eyes and nose	Baretta *et al*., 1969	NOAEL: 6.78 x 10^5	10	TCEQ, 2009	2011
MRL	Acute	0.5 ppm (1,278 µg/m^3)	Maternal and developmental toxicity	John *et al.* (1977, 1981).	NOAEL(ADJ): 15 ppm (38,344 µg/m^3).	30	ATSDR, 2006	NA
REL; 1-hour	Acute	180,000	Mild headache and dryness of their eyes and nose	Baretta *et al*., 1969	NOAEL (ADJ): 715 ppm (approx. 1.8 x 10^{-6} µg/m^3)	10	Cal EPA, 2008	NA
AAQC; 24-hour	Acute	1	Central nervous system depression	NA	NA	NA	MOE, 2012	1989
AAQC	Chronic	0.2	NA	NA	NA	NA	MOE, 2012	NA
RfC	Chronic	100	Liver cell polymorphism	Til *et al.,* 1983; 1991	NOAEL (HEC): 2,500	30	US EPA IRIS, 2000	2000
RfC	Chronic	100	Liver cell polymorphism	Til *et al.,* 1983; 1991	NOAEL (HEC): 2,500	30	MOE, 2011	NA
ReV; Annual Average	Chronic	60	Centrilobular hypertrophy in the liver (rat)	Thornton *et al*., 2002	BMCL10 (ADJ): 0.680 ppm (1,738 µg/m^3)	30	TCEQ, 2009	NA
Unit Risk	Chronic	8.8 x 10^{-6} (µg/m^3)$^{-1}$	Increased incidence of liver angiosarcomas, angiomas, hepatomas, and neoplastic nodules in female rats	Maltoni *et al.,* 1981, 1984	NA	NA	US EPA, 2000	NA
Unit Risk	Chronic	8.8 x 10^{-6} (µg/m^3)$^{-1}$	NA	US EPA, 2000	NA	NA	MOE, 2011	NA
Unit Risk	Chronic	7.8 x 10^{-5} (µg/m^3)$^{-1}$	Increased tumor incidence	Drew *et al.,* 1983	NA	NA	Cal EPA, 2011	1990
Unit Risk	Chronic	2.78 x 10^{-5} (µg/m^3)$^{-1}$	Increased incidence of liver angiosarcomas, angiomas, hepatomas, and neoplastic nodules	Maltoni *et al.,* 1981, 1984	NA	NA	RIVM, 2001	NA

Table A-64 Inhalation Toxicity Reference Values								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***
Unit Risk	Chronic	1.0×10^{-6} $(\mu g/m^3)^{-1}$	NA	NA	NA	NA	WHO, 2000	1987
Unit Risk	Chronic	8.4×10^{-6} $(\mu g/m^3)^{-1}$	NA	US EPA, 2000	NA	NA	TCEQ, 2009	NA

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of $\mu g/m^3$ unless otherwise noted.

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A-2.1.43 Zinc

The annual inhalation exposure limit of 2 µg/m^3 also proposed by the TCEQ (2014) were used for the assessment of zinc (Table A-65). These exposure limits were chosen as the only available values.

The chronic oral exposure limit of 300 µg/kg/d proposed by US EPA IRIS (2005) was used for the oral assessment of zinc (Table A-66). This chronic exposure limit was selected based on the robustness of supporting study data. Further, MOE (2011) and ATSDR (2005) both endorse the same value.

Table A-65 Inhalation Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
ESL; Annual average	Chronic	2 (in PM_{10})	Health based	NA	NA	NA	TCEQ, 2014	2010

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/m^3 unless otherwise noted.

Table A-66 Oral Toxicity Reference Values

Type	*Duration*	*Value*[a]	*Critical Effect*	*Reference*	*Point of Departure*	*UF*	*Source*	*Derived*
RfD	Chronic	300	Decrease in erythrocyte Cu, Zn-super oxide dismutase (ESOD) activity	Fischer *et al.,* 1984; Yadrick *et al.,* 1989; Davis *et al*., 2000; Milne *et al*., 2001	LOAEL: 910 µg/kg/day	3	US EPA IRIS, 2005	NA
RfD	Chronic	300	NA	NA	NA	NA	MOE, 2011	NA
MRL	Chronic	300	Decreases in ESOD activity; changes in serum ferritin in females	Yadrick *et al*., 1989	NOAEL: 830 µg/kg/day	3	ATSDR, 2005	NA
TDI	Chronic	600	Reduced iron and copper status; increased growth of infant: length, weight, and head circumference	Walravens and Hambidge, 1976; Yadrick *et al.,* 1989	LOAEL (adult): 60 mg/day; NOAEL (infant and child): 4.5 mg/day	1.5 (Yadrick *et al*., 1989) for adult; no UF (Walravens and Hambidge, 1976) for infant and child	Health Canada , 2010	NA
TDI	Chronic	500	Reduced ESOD Activity	NA	LOAEL: 1,000 µg/kg/day	2	RIVM, 2001	NA
pTDI	Chronic	100	NA	NA	NA	NA	WHO, 2003	NA

Table A-66 Oral Toxicity Reference Values								
Type	***Duration***	***Value***[a]	***Critical Effect***	***Reference***	***Point of Departure***	***UF***	***Source***	***Derived***

Shaded exposure limits were selected as toxicological reference values for the current risk assessment.
NA Not available.
[a] Units of µg/kg/d unless otherwise noted.

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APPENDIX B

WORKED EXAMPLE FOR THE HUMAN HEALTH MULTIPLE PATHWAY EXPOSURE MODEL

APPENDIX B: WORKED EXAMPLE FOR THE HUMAN HEALTH MULTIPLE PATHWAY EXPOSURE MODEL

B-1.0 INTRODUCTION

The human health risk assessment (HHRA) focused on both direct and indirect health risks associated with air emissions from the Highland Creek Treatment Plant (hereafter referred to as "the HCTP"). The HCTP will emit chemicals of concern (COCs) directly into air from various sources, thus people residing near the HCTP, as well as people visiting the area could be directly exposed to the COCs *via* inhalation.

The primary pathway of exposure is inhalation; however, people that reside in the area might be exposed to the COCs *via* secondary exposure pathways. Some COCs emitted to the atmosphere *via* air emissions may be deposited onto the soils and plants surrounding the HCTP site. Depending on the fate, transport, and persistence of the COCs in the environment, chemical deposition could affect the chemical concentrations in local soils and foods (*i.e.,* locally grown produce).

Health risks from air emissions were characterized by comparing modelled long-term air concentrations of COCs with regulatory criteria considered protective of human health and these air concentrations were incorporated into the multimedia exposure model. Health risks associated with indirect exposure pathways such as consumption of locally grown produce and fruits were characterized through a detailed multimedia or multiple pathway exposure model used to predict long term exposures from persistent and/or bioaccumulative COCs. Estimated long-term exposures were also compared with oral COC exposure limits considered protective of human health.

This appendix provides summaries of the calculations used to estimate media concentrations and human exposures to the COCs from long-term (chronic) multiple pathway exposures from the HCTP, along with example calculations. Many of the methods, equations and assumptions used to predict concentrations in various environmental media were obtained from the United States Environmental Protection Agency Office of Solid Waste (US EPA, 2005), Health Canada (2012), and the Ontario Ministry of the Environment and Climate Change (MOE, 2011). Potential multiple pathway exposures to the COCs were predicted for residents using the highest annual average concentrations and the highest incremental increase in concentrations.

B-2.0 ENVIRONMENTAL MEDIA CONCENTRATIONS

In order to quantify potential human exposures (and associated health impacts) through the oral and dermal pathways as a result of emissions from the HCTP, predicted chemical concentrations in various environmental media were required to estimate exposures and characterize risks. Chemical concentrations in the following media were estimated for the multiple pathway exposure model:

- Soil;
- Dust; and,
- Garden vegetables (above and below ground plants).

The worked example is presented for a resident toddler exposed to polychlorinated dibenzo-p-dioxins and furans (as 2,3,7,8-TCDD), as toddlers typically represent the most sensitive lifestage due to their body weight and behavioural characteristics.

B-2.1 Chemical Concentrations in Air

Table B-1 presents the polychlorinated dibenzo-p-dioxins and furans (as 2,3,7,8-TCDD) air concentration that was used to estimate media concentrations for the human health risk assessment (HHRA) model for the Base Case Scenario (*i.e.*, existing multiple hearth incineration) at the maximum air concentration and deposition rates (Base Case (Max)).

Table B-1 Air Concentration used in the Worked Example

Chemical of Concern	*Concentration [µg/m³]*
2,3,7,8-TCDD	3.87E-11

B-2.2 Chemical Deposition

Atmospheric deposition is based on two forms of deposition (*i.e.,* dry and wet) and two chemical phases (*i.e.,* vapour and particulate). The maximum and average atmospheric deposition rates were used in the multiple pathway exposure model to predict COC concentrations in various media. Deposition rates were modelled by Golder Associates Ltd. (Golder). Table B-2 presents the dry, wet, and total deposition rates for 2,3,7,8-TCDD that were used in the HHRA model for Base Case (Max).

Table B-2 Deposition Rates used in the Worked Example [mg/m²/year]

Chemical of Concern	*Dry Deposition Rate*	*Wet Deposition Rate*	*Total Deposition Rate*
2,3,7,8-TCDD	3.57E-09	8.85E-06	8.85E-06

B-2.3 Chemical Concentration in Soil (C_s)

This section presents the equations used for the calculation of cumulative COC concentrations in soil.

B-2.3.1 Cumulative COC Concentration in Soil

US EPA (2005) recommended three (3) equations for the calculation of cumulative soil concentrations. Two (2) of these equations are recommended for the calculation of carcinogens:

Equation 1 – *For $T_2 \leq tD$:*

$$C_s = \frac{D_s}{ks \cdot (tD - T_1)} \cdot \left[\left(tD + \frac{\exp(-ks \cdot tD)}{ks} \right) - \left(T_1 + \frac{\exp(-ks \cdot T_1)}{ks} \right) \right]$$

Equation 2 – *For $T_1 < tD < T_2$:*

$$C_s = \frac{\left(\frac{D_s \cdot tD - Cs_{tD}}{ks} \right) + \left(\frac{Cs_{tD}}{ks} \right) \cdot \left(1 - \exp\left[- ks \cdot (T_2 - tD) \right]\right)}{(T_2 - T_1)}$$

Where:

C_s	=	Average soil concentration over exposure duration (mg/kg)
D_s	=	Deposition term (mg/kg/yr)
ks	=	COC soil loss constant due to all processes (yr^{-1})
tD	=	Time period over which deposition occurs (yr)
T_1	=	Time period at the beginning of combustion (yr)
Cs_{tD}	=	Soil concentration at time tD (mg/kg)
T_2	=	Length of exposure duration (yr)

US EPA (2005) recommended the following equation for calculating cumulative soil concentrations for noncarcinogenic COCs:

Equation 3:

$$C_s = \frac{D_s \times \left[1 - \exp(- ks \times tD)\right]}{ks}$$

Where:

C_s	=	Average soil concentration over exposure duration (mg/kg)
D_s	=	Deposition term (mg/kg/yr)
ks	=	COC soil loss constant due to all processes (yr^{-1})
tD	=	Time period over which deposition occurs (yr)

The operating lifetime of the HCTP is anticipated to be 30 years. Equation 1 is recommended when the exposure duration being modelled is less than or equal to the operating lifetime of the HCTP. Equation 2 is recommended when the exposure duration being modelled is greater than the operating lifetime of the HCTP. Equation 3 is used to predict the COC concentration in soil over the operating lifetime of the HCTP (*i.e.,* 30 years). For the purposes of calculating cumulative COC soil concentrations, the US EPA (2005) recommended equation for noncarcinogenic COCs (*i.e.,* Equation 3) was selected for the current assessment given that it results in the most conservative prediction of COC concentrations in soil.

The calculation of the deposition term (D_s) and the soil loss constant (*ks)* are presented in the sections below.

As part of the *Ds* calculation, the soil mixing zone depth is considered. The soil mixing zone depth is an important variable when calculating an appropriate soil concentration. Tilled soil will generally have lower COC concentrations than untilled soil given that tilling activities allow

deposited COCs to mix with a greater volume of soil. US EPA (2005) recommended soil mixing zone depths of 0.2 m for tilled soil and 0.02 m for untilled soil. Soil concentrations in the HHRA model were modelled using both mixing zones.

Example 1 *2,3,7,8-TCDD Concentration in Soil for tilled soil under Base Case (Max)*

$$C_s = \frac{2.95E-08 \times [1 - \exp(-0.03 \times 30)]}{0.03}$$

$$C_s = 5.83E-07$$

The 2,3,7,8-TCDD concentration in soil for tilled soil under Base Case (Max) was 5.83E-07 mg/kg.

Example 2 *2,3,7,8-TCDD Concentration in Soil for untilled soil under Base Case (Max)*

$$C_s = \frac{2.95E-07 \times [1 - \exp(-0.03 \times 30)]}{0.03}$$

$$C_s = 5.83E-06$$

The 2,3,7,8-TCDD concentration in soil for untilled soil under Base Case (Max) was 5.83E-06 mg/kg.

B-2.3.2 Deposition Term (D_s)

US EPA (2005) recommended the following equation to calculate D_s:

$$D_s = Hg_{AF-Soil} \cdot \left[\frac{100 \times Q}{Z_s \times BD}\right] \cdot [F_v \times (Dydv + Dywv) + (Dydp + Dywp) \times (1 - F_v)]$$

Where:

D_s	=	Deposition term (mg/kg/yr)
$Hg_{AF-Soil}$	=	Mercury adjustment factor (unitless) – Inorganic mercury: 0.48 x 0.98; All other COCs: 1
100	=	Unit conversion factor (mg-cm²/kg-cm²)
Q	=	COC-specific emission rate (g/s)
Z_s	=	Soil mixing zone depth (cm)
BD	=	Bulk density (1.5 g/cm³)
F_v	=	Fraction of COC air concentration in vapour phase (unitless)
Dydv	=	Unitized yearly average dry deposition from vapour phase (s/m²-yr)

Dywv	=	Unitized yearly average wet deposition from vapour phase (s/m²-yr)
Dydp	=	Unitized yearly average dry deposition from particle phase (s/m²-yr)
Dywp	=	Unitized yearly average wet deposition from particle phase (s/m²-yr)

US EPA (2005) considered 48% of total mercury emitted was deposited in soil and it was assumed that the mercury speciation in soil was 98% divalent mercury. This is considered in the $Hg_{AF\text{-}Soil}$ term.

Deposition rates were provided by Golder. Therefore, D_s was calculated using the following equation:

$$D_s = HG_{AF-Soil} \cdot \frac{Dep}{Z_s \times BD}$$

Where:

D_s	=	Deposition term (mg/kg/yr)
$Hg_{AF\text{-}Soil}$	=	Mercury adjustment factor (unitless) – Inorganic mercury: 0.48 x 0.98; All other COCs: 1
Dep	=	Total deposition rate (mg/m²/yr)
Z_s	=	Soil mixing zone depth (m)
BD	=	Bulk density (1500 kg/m³)

US EPA (2005) provided default values for *BD*. As previously discussed, US EPA (2005) recommended soil mixing zone depths of 0.2 m for tilled soil and 0.02 m for untilled soil. The deposition term for both mixing zones were used.

Example 3 *Deposition term for 2,3,7,8-TCDD for tilled soil (0.2 m mixing zone) under Base Case (Max)*

$$D_s = 1 \cdot \frac{8.85E-06}{0.2 \times 1500}$$

$$D_s = 2.95E-08 mg/kg/yr$$

The deposition term for 2,3,7,8-TCDD for tilled soil under Base Case (Max) is 2.95E-08 mg/kg/year.

Example 4 *Deposition term for 2,3,7,8-TCDD for untilled soil (0.02 m mixing zone) under Base Case (Max)*

$$D_s = 1 \cdot \frac{8.85E-06}{0.02 \times 1500}$$

$$D_s = 2.95E-07 mg/kg/yr$$

The deposition term for 2,3,7,8-TCDD for untilled soil under Base Case (Max) is 2.95E-07 mg/kg/year.

B-2.3.3 Soil Loss Constant (ks)

Chemicals may be lost from soil by leaching, runoff, erosion, biotic and abiotic degradation, and volatilization. The COC soil loss constant (*ks*) accounts for these processes using the following equation (US EPA 2005):

$$Ks = Ksg + Kse + Ksr + Ksl + Ksv$$

Where:

Ks	=	Soil loss constant due to all processes (yr^{-1})
Ksg	=	Soil loss constant due to biotic and abiotic degradation (yr^{-1})
Kse	=	Soil loss constant due to soil erosion (yr^{-1})
Ksr	=	Soil loss constant due to surface runoff (yr^{-1})
Ksl	=	Soil loss constant due to leaching (yr^{-1})
Ksv	=	Soil loss constant due to volatilization (yr^{-1})

The calculation of each COC loss constant is described in the sections below.

Example 5 Soil Loss Constant due to All Processes for 2,3,7,8-TCDD

$$Ks = 0.03 + 0 + 0 + 0 + 4.62E - 05$$

$$Ks = 0.03$$

The *Ks* for 2,3,7,8-TCDD was calculated to be 0.03 yr^{-1}.

B-2.3.3.1 Soil Loss Constant due to Biotic and Abiotic Degradation (*Ksg*)

The US EPA (2005) Companion Database provides *Ksg* values for many of the COCs assessed in the HHRA. For those COCs not presented in the Companion Database, *Ksg* values were calculated using the US EPA (2005) recommended equation presented below. The COC soil half-life values used in the HHRA were provided by US EPA (2012) EPI Suite database.

For metals, the calculation of *Ksg* was required given that *Kse*, *Ksr*, *Ksl*, and *Ksv* for metals are equal to zero (0) for this assessment (discussed in sections below). In general, five (5) half-lives are sufficient to reach 99.9% of equilibrium with first order kinetics. Therefore, the *Ksg* of metals were calculated using 5 half lives of 80 years.

$$Ksg = \frac{0.693}{t_{1/2}}$$

Where:

Ksg	=	Soil loss constant due to biotic and abiotic degradation (yr^{-1})
$t_{1/2}$	=	COC half life in soil (yr)

The US EPA (2005) Companion Database provided a *Ksg* value of 0.03 yr^{-1} for 2,3,7,8-TCDD.

B-2.3.3.2 Soil Loss Constant due to Soil Erosion (*Kse*)

US EPA (2005) recommended that *Kse* should be equal to zero (0). This is because contaminated soil erodes both onto and off the site. Therefore, the *Kse* for 2,3,7,8-TCDD was set to zero (0).

B-2.3.3.3 Soil Loss Constant Due to Surface Runoff (*Ksr*)

US EPA (2005) recommended the following equation for the calculating *Ksr*:

$$Ksr = \frac{RO}{\theta_{sw} \cdot Z_s} \cdot \left(\frac{1}{1 + \left(Kd_s \cdot BD / \theta_{sw} \right)} \right)$$

Where:

Ksr	=	Soil loss constant due to surface runoff (yr^{-1})
RO	=	Average annual surface runoff from pervious areas (cm/yr)
Θ_{sw}	=	Soil volumetric water content (0.2 ml/cm^3)
Z_s	=	Soil mixing zone depth (cm)
Kd_s	=	Soil/water partition coefficient (ml/g)
BD	=	Soil bulk density (1.5 g/cm^3)

US EPA (2005) provided default values for Θ_{sw} and *BD.*

For this assessment, *Ksr* was conservatively assumed to be zero (0) for all COCs.

B-2.3.3.4 Soil Loss Constant Due to Leaching (*Ksl*)

US EPA (2005) recommended the following equation for the calculating *Ksl*:

$$Ksl = \frac{P + I - RO - E_v}{\theta_{sw} \cdot Z_s \cdot \left[1.0 + \left(BD \cdot Kd_s / \theta_{sw} \right) \right]}$$

Where:

Ksl	=	Soil loss constant due to leaching (yr^{-1})
P	=	Average annual precipitation (cm/yr)
I	=	Average annual irrigation (cm/yr)
RO	=	Average annual surface runoff from pervious areas (cm/yr)
E_V	=	Average annual evapotranspiration (cm/yr)
Θ_{sw}	=	Soil volumetric water content (0.2 ml/cm^3)
Z_s	=	Soil mixing zone depth (cm)
BD	=	Soil bulk density (1.5 g/cm^3)
Kd_s	=	Soil/water partition coefficient (cm^3/g)

US EPA (2005) provided default values for Θ_{sw} and *BD.*

For this assessment, *Ksl* was conservatively assumed to be zero (0) for all COCs.

B-2.3.3.5 Soil Loss Constant Due to Volatilization (*Ksv*)

US EPA (2005) recommended the following equation for the calculating *Ksv*:

$$Ksv = \left(\frac{CF \cdot H}{Z_s \cdot Kd_s \cdot R \cdot T_a \cdot BD}\right) \cdot \left(\frac{D_a}{Z_s}\right) \cdot \left[1 - \left(\frac{BD}{\rho_{soil}}\right) - \theta_{sw}\right]$$

Where:

Ksv	=	Soil loss constant due to volatilization (yr^{-1})
CF	=	Unit conversion factor (3.1536E+07 s/yr)
H	=	Henry's Law constant (atm-m^3/mol)
Z_s	=	Soil mixing zone depth (20 cm)
Kd_s	=	Soil/water partition coefficient (ml/g)
R	=	Universal gas constant (8.205 E-05 atm-m^3/mol-K)
T_a	=	Ambient air temperature (298.1 K)
BD	=	Soil bulk density (1.5 g/cm^3)
D_a	=	Diffusivity of COC in air (cm^2/s)
ρ_{soil}	=	Solids particle density (2.7 g/cm^3)
Θ_{sw}	=	Soil volumetric water content (0.2 ml/cm^3)

US EPA (2005) provided default values for T_a, *BD*, ρ_{soil} and, Θ_{sw}. This soil loss constant was calculated using the more conservative mixing zone depth of 20cm.

Example 6 Soil Loss Constant due to Volatilization for 2,3,7,8-TCDD

$$Ksv = \left(\frac{3.1536E+07 \cdot 3.29E-05}{20 \cdot 3.89E+04 \cdot 8.205E-05 \cdot 298.1 \cdot 1.5}\right) \cdot \left(\frac{0.104}{20}\right) \cdot \left[1 - \left(\frac{1.5}{2.7}\right) - 0.2\right]$$

$$Ksv = 4.62E-05$$

The *Ksv* for 2,3,7,8-TCDD was calculated to be 4.62E-05 yr^{-1}.

B-2.4 Chemical Concentration in Dust

Concentrations of COCs in fugitive dust were calculated based on the following equation:

$$C_d = C_s \cdot DL \cdot CF$$

Where:

C_d	=	COC Concentration in dust (µg/m³)
C_s	=	Average soil concentration over exposure duration (mg/kg)
DL	=	Airborne respirable particulate matter concentration (µg/m³)
CF	=	Unit conversion factor (0.000001 kg/mg)

Health Canada (2012) provided an average airborne respirable particulate matter concentration of 0.76 µg/m³. This value was selected for use as *DL* in the above equation.

Example 7 2,3,7,8-TCDD Concentration in Dust

$$C_d = 5.83E-06 \cdot 0.76 \cdot 0.000001$$
$$C_d = 4.43E-12$$

The 2,3,7,8-TCDD concentration in dust was calculated to be 4.43E-12 µg/m³.

B-2.5 Chemical Concentrations in Plants

The methodology used to estimate the contribution from each route of the chemical uptake in plants are described in the following sections. Four (4) plant groups were modelled for the HHRA: exposed aboveground produce, protected aboveground produce, belowground produce, and fruit. Table B-3 provides a summary of the mechanisms that were included when estimating the uptake of COCs into the tissue of each plant group.

Table B-3 Summary of Mechanisms Included in the Estimation of COC Uptake into Plants

Plant Group	*Direct Deposition*	*Vapour Uptake*	*Root Uptake*
Exposed Aboveground Produce	x	x	x
Protected Aboveground Produce			x
Belowground Produce			x
Fruit	x	x	x

The worked example is provided for exposed aboveground produce; however, Table B-4 presents the input parameters that were used for the remaining plant groups included in the HHRA model. The current assessment did not adjust concentrations in plants for human consumption with a washing and peeling factor to account for potential reduction in exposures where washing or peeling occurs. The predicted COC concentration in plants are on a wet weight (WW) basis for produce and fruits.

Table B-4 Input Parameters for Predicting COC Concentrations in Plants[a]					
Plant Group	***Intercept fraction (Rp) [unitless]***	***Plant surface loss coefficient (kp) [yr^{-1}]***	***Length of plant exposure (Tp) [yr]***	***Yield or productivity (Yp) [kg DW/m^2]***	***Water content of plant (WC) [unitless]***
Exposed Aboveground Produce	0.982	18	0.164	5.66	0.85
Protected Aboveground Produce	N/A	N/A	N/A	N/A	0.85
Belowground Produce	N/A	N/A	N/A	N/A	0.87
Fruits	0.053	18	0.164	0.25	0.85

[a] Input parameters provided by US EPA (2005).
N/A Not applicable.

B-2.5.1 Plant Concentrations as a Result of Direct Deposition

US EPA (2005) recommended the following equation to calculate COC concentrations in plants as a result of direct deposition:

$$Pd = \frac{Hg_{AG-Plant} \cdot CF \cdot Q \cdot (1 - F_v) \cdot [Dydp + (Fw \cdot Dywp)] \cdot Rp \cdot [1.0 - \exp(-kp \cdot Tp)]}{Yp \cdot kp} \cdot (1 - WC)$$

Where:

Pd	=	COC concentration in plants as a result of direct (wet and dry) deposition (mg/kg)
$Hg_{AF-Plant}$	=	Mercury adjustment factor (unitless) – Inorganic mercury: 0.48 x 0.78; All other COCs: 1
CF	=	Unit conversion factor (1000 mg/g)
Q	=	COC emission rate (g/s)
F_v	=	Fraction of COC air concentration in vapour phase (unitless)
Dydp	=	Unitized yearly average dry deposition from particle phase (s/m^2/yr)
Fw	=	0.2 for anions, 0.6 for cations & most organics (unitless)
Dywp	=	Unitized yearly wet deposition from particle phase (s/m^2/yr)
Rp	=	Interception fraction of edible portion of plant (unitless)
kp	=	Plant surface loss coefficient (yr^{-1})
Tp	=	Length of plant exposure to deposition per harvest of the edible portion of the plant group (yr)
Yp	=	Yield or standing crop biomass of the edible portion of the plant (productivity) (kg DW/m^2)
WC	=	Water content of plant (unitless)

US EPA (2005) considered 48% of total mercury emitted was deposited in soil and it was assumed that the mercury speciation in plants was 78% divalent mercury. This is considered in the $Hg_{AF-Plant}$ term.

Since deposition rates were provided by Golder, the deposition term was calculated using the following equation:

$$Pd = \frac{Hg_{AF-Plant} \cdot [D_d + (D_w \cdot Fw)] \cdot Rp \cdot [1.0 - \exp(-kp \cdot Tp)]}{Yp \cdot kp} \cdot (1 - WC)$$

Where:

Pd	=	COC concentration in plants as a result of direct (wet and dry) deposition (mg/kg)
$Hg_{AF\text{-}Plant}$	=	Mercury adjustment factor (unitless) - Inorganic mercury: 0.48 x 0.78; All other COCs: 1
D_d	=	Dry deposition rate (mg/m²/yr)
D_w	=	Wet deposition rate (mg/m²/yr)
Fw	=	0.2 for anions, 0.6 for cations & most organics (unitless)
Rp	=	Interception fraction of edible portion of plant (unitless)
kp	=	Plant surface loss coefficient (yr^{-1})
Tp	=	Length of plant exposure to deposition per harvest of the edible portion of the plant group (yr)
Yp	=	Yield or standing crop biomass of the edible portion of the plant (productivity) (kg DW/m²)
WC	=	Water content of plant (unitless)

Example 8 *2,3,7,8-TCDD Concentration in Exposed Aboveground Produce as a Result of Direct Deposition*

$$Pd = \frac{1 \cdot [3.57E-09 + (8.85E-06 \cdot 0.6)] \cdot 0.982 \cdot [1.0 - \exp(-18 \cdot 0.164)]}{5.66 \cdot 18} \cdot (1 - 0.85)$$

$$Pd = 7.28E-09$$

The 2,3,7,8-TCDD concentration in exposed aboveground produce as a result of direct deposition under Base Case (Max) is 7.28E-09 mg/kg WW.

B-2.5.2 Plant Concentrations as a Result of Vapour Uptake

US EPA (2005) recommended the following equation to calculate COC concentrations in plants as a result of vapour uptake:

$$Pv = Hg_{AF-Plant} \cdot Q \cdot F_v \cdot \frac{Cyv \cdot Bv_{ag} \cdot VG_{ag}}{\rho_{air}} \cdot (1 - WC)$$

Where:

Pv	=	COC concentration in plants as a result of vapour uptake (mg/kg)
$Hg_{AF\text{-}Plant}$	=	Mercury adjustment factor (unitless) - Inorganic mercury: 0.48 x 0.78; All other COCs: 1
Q	=	COC emission rate (g/s)
F_v	=	Fraction of COC in vapour phase (unitless)
Cyv	=	Unitized yearly average air concentration from vapour phase (µg-s/g-m³)

Bv_{ag}	=	COC mass-based air-to-plant biotransfer factor (µg/g DW plant / µg/g air)
VG_{ag}	=	Empirical correction factor for aboveground plants (unitless)
ρ_{air}	=	Density of air (1,200 g/m³; Weast 1981)
WC	=	Water content of plant (unitless)

US EPA (2005) considered 48% of total mercury emitted was deposited in soil and it was assumed that the mercury speciation in plants was 78% divalent mercury. This is considered in the $Hg_{AF\text{-}Plant}$ term.

Since air concentrations were provided by Golder, P_v was calculated using the following equation:

$$Pv = Hg_{AF-Plant} \cdot \frac{C_{air} \cdot F_v \cdot \left(\frac{B_v}{RF}\right) \cdot VG_{ag}}{\rho_{air}} \cdot (1 - WC)$$

Where:

Pv	=	COC concentration in plants as a result of vapour uptake (mg/kg)
$Hg_{AF\text{-}Plant}$	=	Mercury adjustment factor (unitless) - Inorganic mercury: 0.48 x 0.78; All other COCs: 1
C_{air}	=	COC concentration in air (µg/m³)
F_v	=	Fraction of COC in vapour phase (unitless)
B_v	=	COC mass-based air-to-plant biotransfer factor (µg/g DW plant / µg/g air)
RF	=	Reduction factor (unitless)
VG_{ag}	=	Empirical correction factor for aboveground plants (unitless)
ρ_{air}	=	Density of air (1,200 g/m³; Weast 1981)
WC	=	Water content of plant (unitless)

As recommended by the US EPA (2005), the biotransfer factor for organics (except dioxin and furan) should be reduced by a factor of 100. Additionally, US EPA (2005) recommended an empirical correction factor (*i.e., VG_{ag}*) of 0.01 for COCs with a log K_{ow} greater than 4 and an empirical correction factor of 1 for COCs with a log K_{ow} less than 4.

The concentration of COCs in plants from direct vapour uptake was calculated using a mass-based air-to-plant biotransfer factor (B_v), which was derived from the volumetric air-to-plant biotransfer factor (B_{vol}) (US EPA, 2005). The equations used to calculate B_v *and* B_{vol} are presented below.

Example 9 *2,3,7,8-TCDD Concentration in Exposed Aboveground Produce as a Result of Vapour Uptake*

$$Pv = 1 \cdot \frac{3.87E-11 \cdot 0.664 \cdot \left(\frac{65500}{1}\right) \cdot 0.01}{1200} \cdot (1 - 0.85)$$

$$Pv = 2.1E-08$$

The 2,3,7,8-TCDD concentration in exposed aboveground produce as a result of vapour uptake under Base Case (Max) is 2.11E-12 mg/kg WW.

B-2.5.2.1 Volumetric Air-to-Plant Biotransfer Factor (B_{vol})

US EPA (2005) recommended the following equation to calculate chemical-specific B_{vol} on a wet weight basis:

$$\log B_{vol} = 1.065 \cdot \log K_{ow} - \log\left(\frac{H}{R \cdot T}\right) - 1.654$$

Where:

B_{vol}	=	Volumetric air-to-plant biotransfer factor (unitless; WW basis)
$log\ K_{ow}$	=	Log of the octanol-water partition coefficient (unitless)
H	=	Henry's Law constant (atm-m³/mol)
R	=	Universal gas constant (8.205 E-05 atm-m³/mol-K)
T	=	Ambient temperature (298.1 K)

US EPA (2005) provided a default value for *R* and *T.*

An example calculation of B_{vol} for 2,3,7,8,-TCDD has not been presented given that B_v for this COC has been provided by the US EPA (2005) Companion Database.

B-2.5.2.2 Mass-Based Air-to-Plant Biotransfer Factor (B_v)

US EPA (2005) recommended the following equation to calculate chemical-specific B_v on a wet weight basis:

$$B_v = \frac{\rho_{air} \cdot B_{vol}}{(1 - WC) \cdot \rho_{forage}}$$

Where:

B_v	=	mass-based air-to-plant biotransfer factor (µg/g DW plant / µg/g air)
ρ_{air}	=	density of air (1.19 g/L; Weast 1981)
B_{vol}	=	volumetric air-to-plant biotransfer factor (unitless; WW basis)
WC	=	water or moisture content of plant (0.85)
ρ_{forage}	=	density of forage (770 g/L; McCrady and Maggard 1993)

An example calculation of B_v for 2,3,7,8-TCDD has not been presented given that it has been provided by the US EPA (2005) Companion Database.

B-2.5.3 Plant Concentrations as a Result of Root Uptake

US EPA (2005) recommended the following two (2) equations to calculate COC concentrations in plants as a result of vapour uptake:

For exposed and protected aboveground produce:

$$\mathrm{Pr} = Cs \cdot BCF \cdot (1 - WC)$$

Where:

Pr	=	COC concentration in plant as a result of root uptake (mg/kg)
Cs	=	Cumulative COC concentration in soil (mg/kg)
BCF	=	Plant-soil bioconcentration factor (kg soil/kg plant DW)
WC	=	Water content of plant (unitless)

For belowground produce:

$$\mathrm{Pr} = Cs \cdot BCF_{root} \cdot VG_{rootveg} \cdot (1 - WC)$$

Where:

Pr	=	COC concentration in plant as a result of root uptake (mg/kg WW)
Cs	=	Cumulative COC concentration in soil (mg/kg)
BCF_{root}	=	Root-soil concentration factor (kg soil/kg plant DW)
$VG_{rootveg}$	=	Empirical correction factor for belowground produce (unitless)
WC	=	Water content of plant (unitless)

US EPA (2005) recommended an empirical correction factor for belowground produce (*i.e.,* $VG_{rootveg}$) of 0.01 for COCs with a log K_{ow} greater than 4 and an empirical correction factor of 1 for COCs with a log K_{ow} less than 4.

Example 10 *2,3,7,8-TCDD Concentration in Exposed Aboveground Produce as a Result of Root Uptake*

$$\mathrm{Pr} = 5.83E-07 \cdot 0.00455 \cdot (1 - 0.85)$$

$$\mathrm{Pr} = 3.98E-10$$

The 2,3,7,8-TCDD concentration in exposed aboveground produce as a result of root uptake under Base Case (Max) is 3.98E-10 mg/kg WW.

B-2.5.3.1 Plant-Soil Bioconcentration Factor (*BCF*)

The US EPA (2005) Companion Database has provided COC-specific bioconcentration factors (BCFs) for each aboveground plant group and root concentration factors (RCF) for belowground produce assessed in the HHRA.

For metal COCs not presented in the Companion Database, BCF values were obtained from Baes *et al.* (1984). For other COCs, BCF values were calculated using the following US EPA (2005) recommended equation:

$$\log BCF = 1.588 - 0.578(\log K_{ow})$$

Where:

BCF	=	Plant-soil bioconcentration factor (kg soil/kg plant DW)
log K_{ow}	=	Log of the octanol-water partition coefficient (unitless)

The above equation was derived from experiments conducted on compounds with log K_{ow} values ranging from 1.15 to 9.35. Thus, *BCF* values for compounds with a log K_{ow} value less than 1.15 should be calculated using a log K_{ow} value of 1.15 and BCF values for compounds with a log K_{ow} greater than 9.35 should be calculated using a log K_{ow} value of 9.35 (US EPA 2005).

An example calculation of *BCF* for 2,3,7,8-TCDD has not been presented given that it has been provided by the US EPA (2005) Companion Database.

B-2.5.3.2 Root-Soil Concentration Factor (BCF_{root})

For metal COCs not presented in the Companion Database, *RCF* values were obtained from Baes *et al.* (1984). For other COCs, *RCF* values were calculated using the following US EPA (2005) recommended equations:

For COCs with log K_{ow} of 2.0 and greater:

$$\log RCF = 0.77 \cdot \log K_{ow} - 1.52$$

$$BCF_{root} = \frac{RCF}{Kd_s \cdot CF \cdot (1 - WC)}$$

Where:

RCF	=	Root concentration factor (kg soil/kg plant WW)
log K_{ow}	=	Log of the octanol-water partition coefficient (unitless)
BCF_{root}	=	Root -soil concentration factor (kg soil/kg plant DW)
Kd_s	=	Soil/water partition coefficient (L/kg)
CF	=	Unit conversion factor (1 kg/L)
WC	=	Water or moisture content of plant (0.87)

For COCs with log K_{ow} less than 2.0:

$$\log(RCF - 0.82) = 0.77 \cdot \log K_{ow} - 1.52$$

$$BCF_{root} = \frac{RCF}{Kd_s \cdot CF1 \cdot (1 - WC)}$$

Where:

RCF	=	Root concentration factor (kg soil/kg plant WW)
log K_{ow}	=	Log of the octanol-water partition coefficient (unitless)
BCF_{root}	=	Root -soil concentration factor (kg soil/kg plant DW)
Kd_s	=	Soil/water partition coefficient (L/kg)
CF1	=	Unit conversion factor (1 kg/L)
WC	=	Water or moisture content of plant (0.87)

As recommended by US EPA (2005), the *RCF* values calculated in the above equations were converted from fresh weight to dry weight using a moisture content of 87% in root vegetables.

An example calculation of *BCF_{root}* for 2,3,7,8-TCDD has not been presented given that it has been provided by the US EPA (2005) Companion Database.

B-2.5.4 Total COC Concentrations in Plants

The total COC concentration in plants was calculated by summing the contribution from direct deposition (if applicable), vapour uptake (if applicable), and root uptake:

$$C_{plant} = Pd + Pv + \mathrm{Pr}$$

Where:

C_{plant}	=	Total COC concentration in plants (mg/kg)
Pd	=	COC concentration in plants as a result of direct (wet and dry) deposition (mg/kg)
Pv	=	COC concentration in plants as a result of vapour uptake (mg/kg)
Pr	=	COC concentration in plants as a result of root uptake (mg/kg)

Example 11 Total 2,3,7,8-TCDD Concentration in Exposed Aboveground Produce

$$C_{forage} = 7.28E-09 + 2.11E-12 + 3.98E-10$$

$$C_{forage} = 7.68E-09$$

The total 2,3,7,8-TCDD concentration in exposed aboveground produce under Base Case (Max) is 7.68E-09 mg/kg WW.

B-3.0 HUMAN EXPOSURE ESTIMATES

As discussed in the main report, the following human receptors were assessed in the HHRA:

- Local residents

The following section presents the methodologies used to estimate COC exposures by human receptors. This worked example is presented for a resident toddler exposed to 2,3,7,8-TCDD as toddlers typically represent the most sensitive life stage due to their body weight and behavioural characteristics.

B-3.1 Human Receptor Characteristics

Human receptor characteristics are required for the purposes of predicting COC exposure. While certain receptor characteristics may vary between receptor groups, some receptor characteristics were assumed to be consistent amongst all human receptors groups. Table B-5 presents the general characteristics for all human receptors used in the HHRA.

Table B-5 Summary of General Characteristics for Human Receptor									
Receptor Lifestage	***Body weight (kg)***	***Soil Ingestion Rate (g/day)***	***Air Inhalation Rate (m^3/day)***	***Surface Area – Hands (cm^2)***	***Surface Area- Other (cm^2)***	***Soil Loading –Hands (g/cm^2/event)***	***Soil Loading – Other (g/cm^2/event)***	***Lifestage Duration (years)***	***Reference***
Adult	7.07E+01	2.00E-02	1.66E+01	8.90E+02	8.22E+03	1.00E-04	1.00E-05	60	Health Canada, 2012
Teen	5.97E+01	2.00E-02	1.56E+01	8.00E+02	7.20E+03	1.00E-04	1.00E-05	8	Health Canada, 2012
Child	3.29E+01	2.00E-02	1.45E+01	5.90E+02	4.55E+03	1.00E-04	1.00E-05	7	Health Canada, 2012
Toddler	1.65E+01	8.00E-02	8.30E+00	4.30E+02	2.58E+03	1.00E-04	1.00E-05	4.5	Health Canada, 2012
Infant	8.20E+00	2.00E-02	2.20E+00	3.20E+02	1.46E+03	1.00E-04	1.00E-05	0.5	Health Canada, 2012

The calculated estimated daily intakes were also adjusted to account for amount of time each receptor group was anticipated to spend in the Study Area. The exposure frequency, exposure duration, and averaging time for each receptor life stage is presented in Table B-6.

Table B-6 Exposure Adjustments Adopted in the Current Assessment[a]

Receptor	*Exposure Frequency (EF; days/year)*	*Exposure Frequency – Direct Soil/Dust Contact (EFs; days/year)[b]*	*Exposure Duration (ED; years)*	*Averaging Time (AT; days)*
Adult	365	274	60	21,900
Teen	365	274	8	2,920
Child	365	274	7	2,555
Toddler	365	274	4.5	1642.5
Infant	365	274	0.5	182.5

[a] Exposure adjustments recommended by Health Canada (2012), unless indicated otherwise.
[b] Number of non-snow covered days (MOE, 2011).

B-3.2 Dietary Ingestion Rates

Ingestion rates are important for the calculation of estimated daily intakes (EDIs). A number of recognized regulatory agencies have recommended ingestion rates for various media, including Health Canada (2012), US EPA (2005), and the US EPA (2011) Exposure Factors Handbook. A review of the available ingestion rates was conducted to determine the most appropriate values for this HHRA.

In accordance with US EPA (2005), the multiple pathway exposure model has predicted COC concentrations in soil, exposed above ground plants, protected above ground plants, belowground plants, and fruit. Ingestion rates are required for each of these food items.

Health Canada (2012) recommended ingestion rates for soil, root vegetables, and other vegetables for all five life stages (*i.e.,* infant, toddler, child, teen, and adult). The soil ingestion rate recommended by Health Canada (2012) was adopted for the current assessment. The root vegetable and other vegetable ingestion rates were based on a Canadian 24-hour recall survey conducted in 1970 to 1972. While this data was collected in Canada, food consumption patterns are anticipated to change over time. Since this data was collected approximately 40 years ago, it was not considered to be representative of present day food ingestion rates.

US EPA (2005) recommended ingestion rates for soil, exposed aboveground produce, protected aboveground produce, and belowground produce. The ingestion rates for these media were based on the US EPA (1997) Exposure Factors Handbook. The US EPA (1997) ingestion rates are based on the 1987-1988 USDA National Food Consumption Survey. The US EPA (2005) recommended food ingestion rates were adjusted for cooking and preparation losses.

US EPA (2011) recommended age-specific per capita and consumer-only ingestion rates on a wet weight basis for home-produced vegetables and fruits. Similar to US EPA (1997), the consumer-only ingestion rates were based on 1987-1988 USDA National Food Consumption Survey. The per capita ingestion rates were estimated by Phillips and Moya (2012) using the 1987-1988 USDA National Food Consumption Survey data and adjusted to account for preparation losses and post-cooking losses. While this data is also over 20 years old, the ingestion rates are considered more appropriate given that they are age-specific and on a per

capita basis. Therefore, the US EPA (2011) home-produced vegetable and fruit ingestion rates were adopted for the current assessment.

Unlike US EPA (2005), US EPA (2011) recommended a single home-produced vegetable ingestion rate, rather than ingestion rates for individual vegetable groups (*i.e.,* exposed aboveground produce, protected aboveground produce, and belowground produce). In order to use the US EPA (2011) recommended home-produced vegetable ingestion rate, it was divided between the three vegetable groups based on the ratio of the US EPA (2005) recommended ingestion rates. For the resident receptor scenario, the home-produced vegetable ingestion rate was assumed to consist of 29.9% as exposed aboveground produce, 57.0% as protected aboveground produce, and 13.1% as belowground produce.

Additionally, the ingestion rates were provided for age groups that do not match the life stages of the Health Canada (2012) guidance. In order to appropriately use these values, the US EPA (2011) were weighted based on the Health Canada (2012) age groups.

The home-produced exposed aboveground produce, protected aboveground produce, belowground produce, and fruit ingestion rates adopted in the current assessment are provided in Table B-7.

Table B-7 Daily Ingestion Rates Adopted for the Current Assessment

Receptor	*Home-Produced Exposed Aboveground Produce* [a]	*Home-Produced Protected Aboveground Produce* [a]	*Home-Produced Belowground Produce* [a]	*Home-Produced Fruit* [a]
Adult	1.74E-04	3.31E-04	7.60E-05	1.95E-04
Teen	1.67E-04	3.19E-04	7.34E-05	1.30E-04
Child	2.42E-04	4.61E-04	1.06E-04	4.16E-04
Toddler	3.59E-04	6.84E-04	1.57E-04	8.90E-04
Infant	0	0	0	0

[a] Daily ingestion rate is in units of kg WW/kg BW/day

B-3.3 Calculating Estimated Daily Intake of COCs

The following sections provide the equations used to predict estimated daily intake of COCs in the HHRA.

B-3.3.1 Incidental Ingestion of Soil

The following equation was used to estimate human exposure *via* incidental ingestion of soil. Soil ingestion rates, body weights, and equations used to predict exposures were based on recommendations from Health Canada (2012). The COC concentration in untilled soil is generally higher than tilled soil. As a conservative measure, the estimated daily intake of COCs *via* soil ingestion was based on chemical concentrations in untilled soil.

$$EDI_{soil} = \frac{C_s \cdot SIR \cdot CF \cdot EFs \cdot ED}{BW \cdot AT}$$

Where:

EDI_{soil} = Estimated daily intake of COC *via* ingestion of soil (mg/kg/day)
C_s = COC concentration in untilled soil (mg/kg)

SIR	=	Incidental soil ingestion rate (g/d)
CF	=	Unit conversion factor (0.001 kg/g)
EFs	=	Exposure frequency for direct soil/dust contact (d/yr)
ED	=	Exposure duration (yr)
BW	=	Receptor body weight (kg)
AT	=	Averaging time (d)

Example 12 *Estimated Daily Intake of 2,3,7,8-TCDD via Ingestion of Soil by the Resident Toddler Under Base Case (Max)*

$$EDI_{soil} = \frac{5.83E-06 \cdot 0.08 \cdot 0.001 \cdot 274 \cdot 4.5}{16.5 \cdot 1642.5}$$

$$EDI_{soil} = 2.12E-11$$

The estimated daily intake of 2,3,7,8-TCDD *via* ingestion of soil by the resident toddler under Base Case (Max) was 2.12E-11 mg/kg/day.

B-3.3.2 Inhalation and Subsequent Ingestion of Dust

The following equation was used to estimate human exposure *via* incidental inhalation and ingestion of dust. Soil ingestion rates, body weights, and equations used to predict exposures were based on recommendations from Health Canada (2012).

$$EDI_{dust} = \frac{C_{dust} \cdot AIR \cdot CF \cdot EFs \cdot ED}{BW \cdot AT}$$

Where:

EDI_{dust}	=	Estimated daily intake of COC *via* ingestion of dust (mg/kg/day)
C_{dust}	=	COC concentration in dust (µg/m³)
AIR	=	Inhalation rate (m³/day)
CF	=	Unit conversion factor (0.001 mg/µg)
EFs	=	Exposure frequency for direct soil/dust contact (d/yr)
ED	=	Exposure duration (yr)
BW	=	Receptor body weight (kg)
AT	=	Averaging time (d)

Example 13 *Estimated Daily Intake of 2,3,7,8-TCDD via Inhalation and Subsequent Ingestion of Dust by the Resident Toddler Under Base Case (Max)*

$$EDI_{dust} = \frac{4.43E-12 \cdot 8.3 \cdot 0.001 \cdot 274 \cdot 4.5}{16.5 \cdot 1642.5}$$

$$EDI_{dust} = 1.67E-15$$

The estimated daily intake of 2,3,7,8-TCDD *via* inhalation and subsequent ingestion of dust by the resident toddler under Base Case (Max) was 1.67E-15 mg/kg/day.

B-3.3.3 Dermal Exposure

Potential dermal exposure was estimated by applying soil loading rates to exposed skin, skin surface areas, and dermal absorption factors to cumulative COC concentrations in soil. Dermal exposures were estimated separately for hands only and for surfaces other than hands (*e.g.,* arms and legs).

The Health Canada (2012) recommended skin soil loading rates and surface area values were adopted for the current assessment. The selected dermal absorption factors were based on recommendations from Health Canada (2010), US EPA (2004), and Risk Assessment Information System (RAIS, 2013).

B-3.3.3.1 Dermal Exposure to Hands

The following equation was used to estimate dermal exposure for hands only:

$$EDI_{Dermal\_h} = \frac{C_s \cdot SAH \cdot SLH \cdot DE \cdot RAF_{dermal} \cdot CF \cdot EFs \cdot ED}{BW \cdot AT}$$

Where:

EDI_{Dermal_h}	=	Estimated daily intake of COC from dermal contact of hands with untilled soil (mg/kg/day)
C_s	=	COC concentration in untilled soil (mg/kg)
SAH	=	Skin surface area of hands (cm²)
SLH	=	Soil loading rate to exposed skin on hands (g/cm²/event)
DE	=	Dermal events per day (1 event/d; Health Canada, 2012)
RAF_{dermal}	=	Relative dermal absorption factor (%)
CF	=	Unit conversion factor (0.001 kg/g)
EFs	=	Exposure frequency for direct soil/dust contact (d/yr)
ED	=	Exposure duration (yr)
BW	=	Receptor body weight (kg)
AT	=	Averaging time (d)

Example 14 Estimated Daily Intake of 2,3,7,8-TCDD via Dermal Contact of Hands by the Resident Toddler under Base Case (Max)

$$EDI_{Dermal\_h} = \frac{5.83E-06 \cdot 430 \cdot 1.00E-04 \cdot 1 \cdot 0.03 \cdot 0.001 \cdot 274 \cdot 4.5}{16.5 \cdot 1642.5}$$

$$EDI_{Dermal\_h} = 3.42E-13$$

The estimated daily intake of 2,3,7,8-TCDD *via* dermal contact of hands by the resident toddler under Base Case (Max) was 3.42E-13 mg/kg/day.

B-3.3.3.2 Dermal Exposure to Surfaces Other than Hands

The following equation was used to estimate dermal exposure of surfaces other than hands:

$$EDI_{Dermal\_o} = \frac{C_s \cdot SAO \cdot SLO \cdot DE \cdot RAF_{dermal} \cdot CF \cdot EFs \cdot ED}{BW \cdot AT}$$

Where:

EDI_{Dermal_o}	=	Estimated daily intake of COC from dermal contact of surfaces other than hands with untilled soil (mg/kg/day)
C_s	=	COC concentration in untilled soil (mg/kg)
SAO	=	Skin surface area other than hands (cm²)
SLO	=	Soil loading rate to exposed skin other than hands (g/cm²/event)
DE	=	Dermal events per day (1 event/d; Health Canada, 2012)
RAF_{dermal}	=	Relative dermal absorption factor (%)
CF	=	Unit conversion factor (0.001 kg/g)
EFs	=	Exposure frequency for direct soil/dust contact (d/yr)
ED	=	Exposure duration (yr)
BW	=	*Receptor b*ody weight (kg)
AT	=	Averaging time (d)

Example 15 Estimated Daily Intake of 2,3,7,8-TCDD via Dermal Contact of Surfaces Other than Hands by the Resident Toddler under Base Case (Max)

$$EDI_{Dermal\_o} = \frac{5.83E-06 \cdot 2580 \cdot 1.00E-05 \cdot 1 \cdot 0.03 \cdot 0.001 \cdot 274 \cdot 4.5}{16.5 \cdot 1642.5}$$

$$EDI_{Dermal\_o} = 2.05E-13$$

The estimated daily intake of 2,3,7,8-TCDD *via* dermal contact of surfaces other than hands by the resident toddler under Base Case (Max) was 2.05E-13 mg/kg/day.

B-3.3.4 Ingestion of Food Items

The following equation was used to estimate human exposure *via* ingestion of exposed aboveground produce, protected aboveground produce, belowground produce, and fruit.

$$EDI_i = \frac{C_i \cdot IR_i \cdot EF \cdot ED}{AT}$$

Where:

EDI_i	=	Estimated daily intake of COC *via* ingestion of food item *i* (mg/kg/day)
C_i	=	COC concentration in food item *i* (mg/kg)
IR_i	=	Ingestion rate of food item *i* (kg/kg BW/day)
EF	=	Exposure frequency (d/yr)
ED	=	Exposure duration (yr)
AT	=	Averaging time (d)

As discussed in Section B-3.3, the US EPA (2011) recommended ingestion rates were adopted and modified for this assessment. The food ingestion rates adopted for the current assessment are provided in Table B-6.

Example 16 *Estimated Daily Intake of 2,3,7,8-TCDD via Ingestion of Exposed Aboveground Produce by the Resident Toddler under Base Case (Max)*

$$EDI_{EAG} = \frac{7.68E-09 \cdot 3.59E-04 \cdot 365 \cdot 4.5}{1642.5}$$

$$EDI_{EAG} = 2.76E-12$$

The estimated daily intake of 2,3,7,8-TCDD *via* ingestion of exposed aboveground produce by the resident toddler under Base Case (Max) was 2.76E-12 mg/kg/day.

B-3.4 Total Estimated Daily Intake

The following equations were used to calculate the total estimated daily intake of COCs *via* incidental ingestion of soil and ingestion of food items.

$$EDI_{total} = EDI_{soil} + EDI_{dust} + EDI_{Dermal\_h} + EDI_{Dermal\_o} + EDI_{EAG} + EDI_{PAG} + EDI_{BG} + EDI_{fruit}$$

Where:

EDI_{total}	=	Total estimated daily intake of COC (mg/kg/day)
EDI_{soil}	=	Estimated daily intake of soil (mg/kg/day)
EDI_{dust}	=	Estimated daily intake of dust (mg/kg/day)
EDI_{dermal_h}	=	Estimated daily intake from dermal exposure to hands (mg/kg/day)
EDI_{dermal_o}	=	Estimated daily intake from dermal exposure from surfaces other than hands (mg/kg/day)
EDI_{EAG}	=	Estimated daily intake of exposed aboveground produce (mg/kg/day)
EDI_{PAG}	=	Estimated daily intake of protected aboveground produce (mg/kg/day)
EDI_{BG}	=	Estimated daily intake of belowground produce (mg/kg/day)
EDI_{fruit}	=	Estimated daily intake of fruit (mg/kg/day)

Example 17 *Total Estimated Daily Intake of 2,3,7,8-TCDD for the Resident Toddler under Base Case (Max)*

$$EDI_{total} = 2.12E-11 + 1.67E-15 + 3.42E-13 + 2.05E-13 + 2.76E-12 + 6.16E-11 + 1.23E-13 + 8.27E-12$$

$$EDI_{total} = 9.46E-11$$

The total estimated daily intake of 2,3,7,8-TCDD *via* all exposure routes for the resident toddler under Base Case (Max) was 9.46E-11 mg/kg/day.

B-4.0 RISK CHARACTERIZATION

The risk characterization step in an HHRA integrates the exposure and hazard assessments to provide a conservative estimate of human health risk for the receptors assessed in the various exposure scenarios. Potential risk was characterized through a comparison of the total estimated daily intake from all exposure pathways with the identified exposure limits.

For chemicals considered to be carcinogenic, exposures over a lifetime were evaluated since development of cancer is a long term process that may take many years to manifest. A special type of receptor called a "lifetime" or "composite" receptor was selected for the evaluation of potential carcinogenic risks for the local resident. This receptor is a "composite" of all relevant life stages for which exposure will be evaluated. Health risks associated with exposure to carcinogenic compounds will be expressed as an estimate of excess or incremental lifetime cancer risk (ILCR) resulting from exposures to chemicals released by the HCTP. Thus, risks associated with carcinogenic compounds will be predicted using the average daily dose over a human receptor's entire life span.

To allow a comprehensive assessment of carcinogenic COCs, all five lifestages were grouped as a composite receptor and evaluated (as per Health Canada, 2012):

- Infant (0 to 6 months);
- Preschool child or toddler (7 months to 4 years);
- Child (5 years to 11 years);
- Adolescent (12 to 19 years); and
- Adult (20 years and over).

To assess risks from exposure to non-carcinogenic COCs, the toddler life stage was selected since this life stage is generally regarded as being the most sensitive due to the elevated soil ingestion rate assumed for this age group (*i.e.*, 6 months to 5 years of age). .

The calculation of hazard quotient (HQ) values for non–carcinogenic COCs and ILCRs for carcinogenic COCs were estimated using the calculated exposure estimates and the equations presented below.

B-4.1 Non-Carcinogens

The following equation was used to calculate the hazard quotients for non–carcinogens (Health Canada, 2012):

$$HQ_i = \frac{EDI_{total}}{RfD}$$

Where:

HQ	=	Hazard quotient of COC for the 'i' lifestage of the residents (unitless)
EDI_{total}	=	Total estimated daily intake of COC *via* all exposure routes for the 'i' lifestage (mg/kg/day)
RfD	=	COC oral reference dose (mg/kg/day)

Example 18 *Hazard Quotient of 2,3,7,8-TCDD for the Resident Toddler under Base Case (Max)*

$$HQ_i = \frac{9.46E-11}{7.0E-10}$$

$$HQ_i = 1.4E-01$$

The estimated hazard quotient for exposure to 2,3,7,8-TCDD by the resident toddler under Base Case (Max) was 1.4E-01.

B-4.2 Carcinogens

The following equation was used to calculate incremental lifetime cancer risk (ILCR) for carcinogens (Health Canada, 2012):

$$ILCR = \sum \left(EDI_{total-i} \, x \, LAF_i \right) \cdot SF$$

Where:

ILCR	=	Incremental lifetime cancer risk (unitless)
$EDI_{total-i}$	=	Total estimated daily intake of COCs *via* all exposure routes for the 'i' lifestage (mg/kg bw/d)
SF	=	COC oral slope factor $(mg/kg/day)^{-1}$
LAF-i	=	Lifetime adjustment factor for the 'i' lifestage for general population (yr-life stage/yr-total)

For the resident receptor scenario, ILCR values are calculated for a composite receptor. A composite receptor is representative of total estimated daily intake of COCs by each lifestage (*i.e.,* infant, toddler, child, teen, and adult), weighted according to the duration of each life stage.

An ILCR was not calculated for 2,3,7,8-TCDD given that an appropriate oral slope factor was not identified.

B-5.0 REFERENCES

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