REPORT

Generic Human Health and Ecological Risk Assessment Study

Durham – York Residual Waste Study

REPORT NO. 1009497.02

REPORT NO. 1009497.02

REPORT TO **Durham – York Residual Waste Study**

ON **Generic Human Health and Ecological Risk Assessment Study**

June 14, 2007

Jacques Whitford
3430 South Service Road, Suite 203
Burlington, Ontario,
L7N 3T9

Phone: 905-631-8684
Fax: 905-631-8960

www.jacqueswhitford.com

Executive Summary

The Regions of Durham and York in Ontario, Canada are undertaking an Individual Environmental Assessment (EA) termed the "Residual Waste Study". The purpose of the Environmental Assessment is to establish the preferred treatment (physical, biological and/or thermal treatment) of the waste that remains after the application of at source waste diversion programs in order to recover resources – both material and energy – and to minimize the amount of material requiring landfilling disposal.

At this point in the EA a thermal treatment energy-from-waste (EFW) facility has been determined to be the preferred option. However, at this point there are five potential short listed sites and no vendor or specific technology has been selected for implementation. Through the EA public consultation process, concerns have been raised about the potential for emissions from an EFW facility to adversely impact human and environmental health.

Although previous human health and ecological risk assessments of thermal treatment conducted in Ontario have concluded that there would be no significant impact on the environment, recent regulatory changes have prompted a re-examination of these findings. The purpose of this report is to study the potential health and environmental impacts and feasibility of siting an EFW facility in the Durham and York Regions. Given that a specific site has not been selected, nor has a vendor or technology been chosen, a regional generic risk assessment was conducted based on emissions data from an existing facility and Ontario emissions guidelines.

This report is in no way meant to replace the requirement for a detailed site specific human health and ecological risk assessment to be conducted upon selection of the preferred site and selected vendor and technology. This report is meant as a feasibility study only and to identify potential issues of concern that should be closely examined during the conduct of the site-specific risk assessment.

Selection of Chemicals of Potential Concern

Given that a specific vendor and technology have not been selected the list of chemicals of potential (CoPCs) was derived from previous studies conducted on similar facilities in Ontario. The majority of the exhaust stack air emission estimates used in this study are based on pollutant emission concentration values obtained from annual stack testing of the 150,000 t/y KMS Peel thermal treatment facility located in Brampton, Ontario. Maximum emission concentrations for all selected COPCs were considered for the air dispersion modelling to illustrate a realistic worst-case scenario for the proposed technology.

For the eight air contaminants found in the Ontario Ministry of Environment (MOE) A-7 guideline (i.e., particulate matter, cadmium, lead, mercury, dioxins and furans, hydrochloric acid, sulphur dioxide, and nitrogen oxides), guideline emission concentration limits were used as default exhaust stack air emission estimates to evaluate the potential risk to the surrounding environment. Furthermore, in addition to stack emissions, vehicular traffic for waste delivery and ash removal were also considered a source of emissions that could potentially impact human and ecological health.

Chemicals of Potential Concern Evaluated in the Risk Assessment

Metals	Chlorinated Monocyclic Aromatics	Chlorinated Polycyclic Aromatics	Polycyclic Aromatic Hydrocarbons	Volatile Organic Compounds
Antimony Arsenic✓ Barium Beryllium Boron Cadmium✓+ Chromium ✓ Cobalt Lead✓+ Mercury✓+ Nickel Phosphorus Silver Vanadium Zinc	1,2-Dichlorobenzene 1,2,4-Trichlorobenzene 1,2,4,5-Tetrachlorobenzene Pentachlorobenzene Hexachlorobenzene 2,4-Dichlorophenol 2,4,6-Trichlorophenol 2,3,4,6-Tetrachlorophenol Pentachlorophenol	PCBs 2,3,7,8-TCDD - (dioxin/furan)TEQ✓+ **Combustion Gases** PM_{10} + $PM_{2.5}$ + CO HCl✓+ HF NO_x✓+ SO_x✓+	*Benzo(a)pyrene group* Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(g,h,i)perylene Benzo(k)fluoranthene Chrysene Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Anthracene Napthalene Phenanthrene	Benzene✓ Chloroform Dichloromethane Formaldehyde Tetrachloroethylene Vinyl chloride✓

Notes: Chemical list derived from Cantox Report for Human Health Risk Assessment for the Proposed Expansion of the KMS Peel, Inc. Brampton, Energy-From-Waste Facility (2000)

✓ Chemicals also reviewed by MOE in Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration (1999)

\+ Chemical also included in GUIDELINE A-7 Combustion and Air Pollution Requirements for New Municipal Waste Incinerators (MOE 2004)

Air Quality Baseline and Modelling

The Residual Waste Study is examining thermal treatment EFW options of processing up to 400,000 t/y of municipal solid waste (MSW). It is important to note that the final annual throughput of MSW has yet to be decided. To that end, three facility scenarios were modelled for both their aerial emissions from the stack, as well as for vehiclar truck traffic that would be required to operate the facility. The three scenarios were as follows:

Operating Scenario 1: 3 process units running at full capacity - 400,000 t/y

Operating Scenario 2: 2 process units running at full capacity - 266,666 t/y

Operating Scenario 3: 1 process units running at full capacity - 133,333 t/y

The physical layout of this theoretical facility is based on current design of EFW facilities operating around the world. The Durham/York facility would occupy a space of 6.1 hectares (256 m by 240 m property) and is assumed to have a single emissions stack with a stack height of 65 meters.

Air Dispersion Modelling

The MOE approved air dispersion model AERMOD (version 04300) was used together with MOE regional MET files (meteorology) modified to include precipitation for the air dispersion modelling of the emissions released from the theoretical MSW thermal treatment facility. Particle phase and vapour phase average concentrations, as well as dry depositions and wet depositions of the selected COPCs were determined at all ground level receptor locations.

The air dispersion modelling included estimates of the 1-hour, 24-hour and annual averaging periods of the COPCs from the facility at the maximum point of impingement (MPOI). The MPOI represents the maximum concentration of COPC at the nearest point where air contamination emitted by a source will fall at or beyond the property line.

The 1-hour maximum ground level concentrations of COPCs were located approximately 700 m from the EFW facility stack or over 680 m outside the fenceline of the facility, while the 24-hour concentrations were typically located within 300 m of the facility. The annual average MPOI concentrations were located between approximately 280 m and 340 m from the fenceline of the facility.

Regional Background Air Quality

To evaluate the potential cumulative risk of exposure to airborne contaminants in this study, background ambient air concentrations of the relevant COPCs were collected. Although the specific location of the facility was unknown at the time of preparation of this report, there are several MOE ambient air quality stations located within the Durham and York Regions. These ambient air quality stations were used to assess the potential background, existing ambient air quality for the Regions.

Results of Air Quality Modelling

The modeled air results were all below the acceptable concentrations provided in Ontario Regulation 419/05. Moreover, even with the addition of ambient concentrations from Durham/York air quality monitoring stations concentrations were below air standards.

Estimating Exposure Point Concentrations in Environmental Media

Maximum predicted ground-level concentrations and wet and dry deposition rates of each CoPC at the MPOI were calculated and carried forward into the risk assessment. The air dispersion modeling and multi-exposure pathway fate and transport of chemicals in the environment was carried out using guidance provided by the United States Environmental Protection Agency (US EPA)

> *US EPA. 2005. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP), Final. EPA520-R-05-006*

Although Canadian regulatory authorities do not publish specific guidance for these types of risk assessments, standard MOE, Health Canada, and Environment Canada protocols for contaminated site risk assessment were adopted in this assessment where guidance exists.

Concentrations of CoPCs were modeled for air, soil, water, sediment, vegetation, produce, agricultural products, fish, and breast milk. These concentrations were then used in exposure estimates in the human health and ecological risk assessment (HHERA).

Human Health Risk Assessment

Selection of exposure scenarios for use in this risk assessment was in general accordance with the recommended exposure scenarios proposed by US EPA (2005) with the addition of a First Nations receptor. All receptors were assumed to live full-time at the MPOI (highest ground level concentration of contaminants), with the exception of the Commercial receptor, who was assumed to commute in only to work at a commercial building located at the MPOI. A brief description of each receptor scenario, highlighting significant assumptions, is provided below.

In general, for carcinogenic CoPCs, intakes were averaged over a lifetime of exposure from birth to 75 years old. For non-carcinogenic CoPCs, an infant, toddler and lifetime-averaged receptor were modelled.

Summary of Receptors and Exposure Pathways Modelled

	Receptors			
Exposure Pathways	**Durham – York Resident [1]**	**Durham – York Subsistence Farmer [2]**	**Durham – York First Nations/ Métis [3]**	**Durham – York Worker[4]**
Direct inhalation	✓	✓	✓	✓
Soil contact	✓	✓	✓	✓
Drinking water	✓	✓	✓	✓
Garden produce	✓	✓	✓	
Fish	✓	✓	✓	
Breast Milk	✓	✓	✓	
Wild game			✓	
Agriculture (meat, poultry)		✓		

1) Resident includes an adult, toddler, and nursing infant.
2) Subsistence Farmer includes an adult, toddler, and nursing infant.
3) First Nations and Métis includes an adult, toddler, and nursing infant.
4) Commercial includes an adult worker and a toddler at a daycare facility

Toxicity Assessment

The purpose of a toxicity assessment is to weigh available evidence regarding the potential for the environmental contaminants to cause adverse effects in exposed populations and to provide an estimate of the relationship between the extent of exposure and the increased likelihood and/or severity of those adverse effects.

Toxicity reference values (TRVs) were reviewed from a number of credible international agencies. In this study preference was first given to US EPA and Health Canada values, whereby the date of the review and validity of the studies were used for selection of TRVs. In the event that IRIS or Health Canada values were not available, or more current TRVs had been established by reputable agencies based on sound toxicological studies they were selected for use.

Chemical Mixtures

In order to properly assess health risks to the human receptors, certain groups of chemicals were assessed as mixtures. For the purposes of this assessment, the carcinogenic PAHs have been assessed as a mixture as have the dioxin and furans as 2,3,7,8 TCDD TEQ.

Additivity of Risks

Combined toxic effects may be produced in a receptor due to exposure to interacting CoPC. Such combined effects may be additive, synergistic (greater than additive), or antagonistic (less than additive). In order to assess these combined effects quantitatively, however, detailed studies of the interactions between CoPC are required, and little information is available in this regard. However, the additive risk of CoPC with the same target organ and toxicological endpoint has been evaluated as part of this risk assessment.

Exposure Assessment

The exposure assessment estimated the amount of a CoPC each receptor may take into his or her body (i.e., a dose) through all applicable exposure pathways. For the purposes of this assessment, the dose of a CoPC depends on the concentrations in air, water, soil, agriculture, (e.g., poultry, cows, milk) fish, plants, breast milk and wild game; the amount of time a person is in contact with these media; and the characteristics of the receptor (e.g., ingestion rate, inhalation rate, body weight, food preferences).

Risk Characterization

Risk characterization is essentially a comparison of the predicted human intake of a CoPC to the toxicity reference value (TRV) for that CoPC to estimate the potential risks to human health from the CoPC evaluated. In Ontario the regulated acceptable level of risk for non-carcinogens is a hazard quotient <0.2, and an incremental lifetime cancer risk of <1 predicted cancer case in an exposed population of one million (1E-06).

Results

Commercial Worker and Daycare Toddler

The Durham-York commercial receptor is assumed to live outside of the region but work full time in the vicinity of the facility. All risk estimates for the Durham-York commercial receptor met the appropriate benchmarks. This suggests that up to a 400,000 t/y EFW facility could be located within a commercial zone of land use without appreciable risk to receptors over its 35 year timeframe.

Durham-York Resident

The Durham-York resident is assumed to live full time in the region, have a backyard garden, and eat some locally caught fish. All risk estimates for the Durham-York resident met the appropriate benchmarks.

Durham-York Subsistence Farmer

The Durham-York subsistence farmer is assumed to live full time in the region and obtain 100% of their food (e.g., meat, fish, poultry, eggs, milk, produce) year-round from their farm. All risk estimates for the Durham-York subsistence farmer met the appropriate benchmarks, with the

exception of the potential risk from dioxins to an infant arising from ingestion of breast milk. When actual dioxin emission rates from the KMS Peel facility were modeled this risk was reduced to below the acceptable regulatory benchmark of HQ=0.2.

Durham-York First Nations and Métis

The Durham-York First Nations and Métis receptor is assumed to live full time in the region, have a backyard garden, and eat locally caught fish and wild game. All risk estimates for the Durham-York First Nations and Métis receptor met the appropriate benchmarks, with the exception of the potential risk from dioxins to an infant arising from ingestion of breast milk and the potential risk from methyl mercury to a toddler arising from ingestion of fish.

There were several uncertainties associated with this risk assessment. These are discussed in detail within the report. A qualitative analysis of uncertainties associated with the risk assessment process supports the conclusion that the risk estimates provided in this report are conservative and likely overstate the potential risks to the local community.

Ecological Risk Assessment

A generic ecological risk assessment was undertaken to help classify potential ecological impacts of EFW facility activities by identifying CoPC, the likely pathways leading to wildlife exposure, and the possible population effects of such exposure. Considering this pro-active approach, results of the ERA will be used to determine if the proposed EFW facility is potentially environmentally acceptable. Furthermore, results of the ERA can be used to guide monitoring and mitigation programs, and guide the site-specific risk assessment priorities.

Problem Formulation

During the problem formulation stage, the chemicals to be assessed in the ERA were identified as being the same as those for the HHRA. The terrestrial ecological receptors selected for evaluation in the ERA were: Masked Shrew, Meadow Vole, Muskrat, Mink, Red Fox, American Robin, Belted Kingfisher, Mallard, and Red-Tailed Hawk. For some ecological receptors it is more appropriate to evaluate risk at the population level (rather than species level). This method was used to evaluate risks to fish, terrestrial plants, soil invertebrates, and benthic (aquatic) invertebrates.

Exposure Assessment

For this generic ERA, oral ingestion of contaminated foods/substances is considered the major source of CoPC exposure. Exposure estimates were also calculated for: soil/sediment ingestion; ingestion of terrestrial vegetation, soil invertebrates, and mammalian prey; water ingestion; ingestion of aquatic invertebrates and fish.

Exposure to ecological receptors was calculated for each of the three Operating Scenarios. To minimize the likelihood of underestimating risks in the ERA, the exposure assessment was conducted in a manner that is likely to lead to an overestimation of actual exposure levels.

Toxicity Assessment

The toxicity assessment identified the potential adverse ecological health effects associated with oral exposure for each CoPC. TRVs were established for each CoPC by reviewing toxicological literature from a variety of sources (i.e., Oakridges National Laboratory (ORNL), US EPA, Agency for Toxic Substances and Disease Registry (ATSDR), primary scientific literature, etc.). TRVs define the amount of each CoPC, a specific ecological receptor can be exposed to on a daily basis below which unacceptable adverse effects are not expected to occur.

Risk Characterization

Risk Characterization combines the information developed in the toxicity and exposure assessments to identify potential sources of unacceptable ecological risk. The likelihood of unacceptable risk is established through the calculation of a Hazard Quotient. HQs are calculated as the ratio of the predicted exposure to the toxicity reference value. For this generic ERA, HQs were calculated at the EFW facility for all three operating scenarios. Typically, a HQ greater than 1.0 (daily exposure greater than TRV) is considered an indication that unacceptable adverse effects could be expected in ecological receptors. However, for this ERA a HQ value of 0.2 was used, in acknowledgement of the fact that existing concentrations of CoPCs in the environment were not incorporated into the exposure assessment.

The highest HQ for a terrestrial ecological receptor was 0.17 for the Belted Kingfisher, as a result of exposure to methyl mercury under scenario one conditions (three process units in operation). Hazard quotients for the remaining ecological receptors did not exceed 0.1, indicating that unacceptable adverse effects were not expected to occur.

The highest HQ for an aquatic receptor was 0.8 for dioxins exposure to aquatic organisms under scenario one operating conditions. This HQ was calculated on the basis of exposure levels resulting from maximum allowable emission rates as defined by the MOE. When dioxins emission rates from a similar EFW facility were used, the HQ decreased substantially, to 0.1

Study Limitations

There are a number of limitations to conducting a human and ecological risk assessment feasibility study for a theoretical facility. These limitations should be taken into consideration in the event that Durham and York Regions pursue a thermal treatment EFW facility, as one option for dealing with their residual municipal solid waste.

The greatest source of uncertainty and the principal limitations for this study are two fold:

1. The final preferred site for the thermal treatment EFW facility has yet to be determined.
2. The final technology and vendor have not yet been selected.

Conclusions

A limited number of potential human health and ecological concerns were identified in this conservative, generic EFW facility risk assessment. These include exposure of Subsistence Farm and First Nations infants and aquatic receptors to dioxin and furans (2,3,7,8-TCDD TEQ) if the concentration being emitted from the stack was at the MOE A-7 Guideline. In addition, methyl mercury posed a potential risk to the First Nations toddler and approached a level of

concern for the Belted Kingfisher. These are issues that deserve particular attention in the site-specific risk assessment. These potential estimates of risk were based on a very conservative set of assumptions that were carried through all phases of the assessment.

Overall, it was determined that a thermal treatment EFW facility could be sited in the Durham and York Regions.

Next Steps

Environmental Baseline Chemical Collection

This generic risk assessment did not account for existing baseline chemical concentrations in the environment. In any site-specific risk assessment this information will be critical to understand the potential cumulative impact that the EFW facility would have on health and the environment. At the time of preparation of this report, a baseline monitoring program for a suite of contaminants of potential concern had been initiated in Durham and York Regions. Once the preferred site has been selected there are plans to conduct an extensive baseline chemical analysis of soil, water, sediment and biota in the area.

Site Specific Risk Assessment

A detailed site specific human health and ecological risk assessment and air dispersion modelling project should be undertaken once a preferred site and vendor is selected. This detailed site specific HHERA should address the concerns raised in this generic risk assessment and should include, at a minimum, consideration of cumulative environmental effects.

In the event that the initial results of the site-specific risk assessment reveal an unacceptable risk to either health and the environment, this does not automatically suggest that the facility could not still be built. Rather, discussions between the risk assessment team and the pollution control engineers could take place to enhance the performance of the technology to reduce the emission of chemicals to the environment.

Ultimately, prior to regulatory approval of the project, it will need to be clearly demonstrated that on a site-specific basis the emissions from the facility would not pose an unacceptable regulatory risk to either humans or the environment.

Table of Contents

List of Tables

List of Figures

List of Appendices

GLOSSARY OF TERMS

AAQC	Ontario Ministry of the Environment Air Quality Criteria
ADD	Average Daily Dose
Additive Interaction	Chemicals have similar targets and modes of action but do not interact. The hazard for exposure to the mixture is simply the sum of hazards for the individual chemicals.
AERMOD	Ontario Ministry of the Environment Approved Air Dispersion Model (Version 04300)
AF	Absorption Factor
Antagonistic Interaction	There is a negative interaction among the chemicals such that the response is less than would be expected if the chemicals acted independently.
ATSDR	Agency for Toxic Substances and Disease Registry
BAF_{fish}	Bioaccumulation Factor in fish
B[a]PTEQ	Benzo[a]pyrene Toxic Equivalent Concentration
BCF	Bioconcentration Factor
Bioaccumulation	The accumulation of a substance in various tissues of a living organism. Bioaccumulation takes place within an organism when the rate of intake of a substance is greater than the rate of excretion or metabolic transformation of that substance.
Bioavailability	The degree to which a substance becomes available to the target tissue after administration or exposure.
Biomagnificaton	The increasing concentration of a substance in the tissues of organisms at successively higher levels in a food chain.
BW	Body Weight
BW_t	Mean body weight for test species
BW_r	Mean body weight for receptor species
CAC	Criteria Air Contaminant
CCME	Canadian Council of Ministers of the Environment
CDI	Chronic Daily Intake

COPC	Contaminants of Potential Concern
CSF	Cancer Slope Factor
EA	Environmental Assessment
EFW	Energy-From-Waste
EPA	Environmental Protection Agency
EPC	Exposure Point Concentration
ERA	Ecological Risk Assessment
f_{site}	Fraction of the total ingestion rate from the site
Ontario Ministry of the Environment Guideline A-7	Combustion and Air Pollution Requirements for New Municipal Waste Incinerators: Emission Limits from a Generic EFW facility
HHRA	Human Health Risk Assessment
HQ	Hazard Quotient
IARC	International Agency for Research on Cancer. An organization of the WHO.
IF	Intake Factor
ILCR	Incremental Lifetime Cancer Risk
IRIS	Integrated Risk Information System
JWMG	Joint Waste Management Group
LADD	Lifetime Average Daily Dose
LD_{50}	Median lethal dose of a toxic substance or radiation is the dose that results in mortality half the members of a tested population.
LOAEL	Lowest-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the lowest dose results in observed adverse health effects. May be used in place of a NOAEL where a cannot be determined.
MET	Meteorology
MOE	Ontario Ministry of the Environment
MPOI	Maximum Point of Impingement. The nearest point where air contamination emitted by a source will impinge on a building or beyond the property line.

	Defined in the MOE's ESDM Procedure as "any point on the ground or on a receptor, such as nearby buildings, located outside the company's property boundaries at which the highest concentration of a contaminant caused by the aggregate emission of that contaminant from a facility is expected to occur."
MSW	Municipal Solid Waste
Non-Interacting	Chemicals have no effect in combination with each other. The toxicity of the mixture is the same as the toxicity of the most toxic component of the mixture.
Non-Threshold Mechanism	A chemical has a non-threshold mechanism if a NOAEL cannot be identified, a lowest observed adverse effects level (LOAEL), being the minimum dose, at which (usually minor) adverse effects are observed, may be used to derive a TRV instead. The application of an extra uncertainty factor to a LOAEL is warranted when deriving a TRV, since the “safe” dose level below that LOAEL may not have been identified.
NOAEL	No-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the highest dose does not result in adverse effects.
OTR_{98}	Ontario "Background" Soil Concentrations
PAH	Polyaromatic hydrocarbons
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzo-p-dioxins
PCDF	Polychlorinated dibenzofurans
PEF	Potency Equivalent Factor
RfD	Reference Dose. The RfD is an estimate of lifetime daily exposure to a non-carcinogen for the general human population that appears to be without appreciable risk of deleterious effects expressed in mg chemical/kg body weight-day.
SF	Slope factor. The SF is a plausible upper bound estimate of the probability of a response per unit intake of a chemical over a lifetime expressed as (mg chemical/kg body weight-day)$^{-1}$ and is used to express carcinogenic effects.
Synergistic Interaction	There is a positive interaction among the chemicals such that the response is greater than would be expected if the chemicals acted independently.
TC	Tolerable Concentration. A term used by Health Canada to describe concentrations in air that a person may be continuously exposed to over a lifetime without adverse effects.
TCDD	Tetrachlorodibenzo-*p*-Dioxin

TEF	Toxic Equivalency Factor
Threshold Mechanism	A chemical as a threshold mechanism id a specific dose level can be identified, at which no adverse effects are observed. This dose, known as a No Observed Adverse Effects Level (NOAEL), adjusted by uncertainty factors, serves as the basis for many TRVs.
TR	Target Risk
TRV	Toxicity Reference Value
UF	Uncertainty Factor. A factor that is applied to NOAELs or LOAELs to yield a RfC or RfD. For example, the UF can be used to account for intra-species and inter-species extrapolations.
Unit Risk	Units risks estimate the upper bound probability of an individual developing cancer following exposure to a particular level (usually as 1 µg/L in water or 1 $\mu g/m^3$) of a potential carcinogen. For example, if the unit risk is 1.2×10^{-6} µg/L then it is expected that 1.2 excess tumours are expected to occur per 1,000,000 people exposed to 1 µg of that chemical in 1 L of drinking water.
UP	Uptake Factor
US EPA	United States Environmental Protection Agency
US EPA MOBILE 6.2 Emission Factors	A trip-based model for emission factors projected based on a typical trip of 7.5 miles and on average speeds for a typical trip.
VEC	Valuable Ecosystem Component
VOC	Volatile Organic Compound
WHO	World Health Organization

1.0 INTRODUCTION

The Regions of Durham and York in Ontario, Canada are undertaking an Individual Environmental Assessment (EA) termed the "Residual Waste Study". The EA was initiated under the Ontario Environmental Assessment Act in 2005. The purpose of the Environmental Assessment is to establish the preferred treatment (physical, biological and/or thermal treatment) of the waste that remains after the application of at source waste diversion programs in order to recover resources – both material and energy – and to minimize the amount of material requiring landfilling disposal.

In June of 2006, both Regional Councils endorsed the Durham/York Joint Waste Management Group (JWMG) and the consultants' recommendation to manage residual waste through a thermal treatment energy-from-waste (EFW) facility. This is the preferred alternative being examined further in the environmental assessment. At this point no vendor or specific technology has been selected for implementation.

In March of 2007, five potential alternative "short-listed" sites were identified through an initial thermal facility site selection process. One site is located in the Town of East Gwillimbury in York Region, while the four additional sites are situated in the Municipality of Clarington (Durham), south of the 401 Highway. The short listed sites are being evaluated using a series of criteria developed as part of the environmental assessment, with the goal of identifying a preferred site in September, 2007.

Through the EA public consultation process, concerns have been raised about the potential for emissions from an EFW facility to adversely impact human and environmental health. In 1999, the Ontario Ministry of the Environment (MOE) published a report entitled "*Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration*" (MOE, 1999). This report concluded that no significant human or ecological effects would be likely in a typical suburban community located near an incinerator. In addition, Cantox Environmental Inc. (now Intrinsik Environmental Inc.) conducted a human health risk assessment on the proposed expansion of the KMS Peel, Inc. Brampton, Energy-From-Waste Facility (Cantox, 2000). This facility is a 150,000 tonne per year (t/y) municipal solid waste thermal treatment EFW facility currently operating in the Region of Peel. Overall, the report concluded that there would unlikely be any significant health effects of residents in the local area.

In addition to the work that has been conducted in Ontario, there are numerous EFW facilities operated across Europe, Japan and the United States. These facilities have undergone rigorous site and technology selection and are considered to operate within acceptable limits within each of their respective legislative regimes.

Although previous human health and ecological risk assessments of thermal treatment conducted in Ontario have concluded that there would be no significant impact on the environment, recent regulatory changes have prompted a re-examination of these findings. The purpose of this report is to study the potential health and environmental impacts and feasibility of siting an EFW facility in the Durham and York Regions. Given that a specific site has not been selected, nor has a vendor or technology been chosen, a regional generic risk assessment

was conducted based on emissions data from an existing facility and Ontario emissions guidelines.

Ultimately, the findings of this report will be incorporated into the siting criteria used to select the preferred site and to identify chemicals or issues of particular concern that should be further scrutinized in any site-specific risk assessment to be completed under the EA.

1.1 Scope of Work

The objective of this report was to examine the potential risk to human and ecological receptors from a generic EFW facility capable of processing up to 400,000 t/y of non-hazardous municipal solid waste (MSW). This report was completed in conjunction with an air dispersion modelling exercise (**Appendix I**) that modeled emissions from a theoretical facility under the following three scenarios:

- Scenario 1 – 3 process units, facility processing 400,000 t/y
- Scenario 2 – 2 process units, facility processing 266,666 t/y
- Scenario 3 – 1 process unit, facility processing 133,333 t/y

Given that a final throughput of MSW has yet to be determined for the facility the additional two scenarios were considered important for consideration in the generic risk assessment. This provides a basis for comparison of relative risk posed by increasing the MSW capacity of the proposed EFW facility.

The Ontario MOE does not publish specific guidance on the assessment of potential risk from air emissions emitted by waste processing facilities. Therefore, the air dispersion modeling and multi-exposure pathway risk assessment was carried out using guidance provided by the United States Environmental Protection Agency (US EPA), specifically from the following documents:

US EPA. 2005. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP), Final. EPA520-R-05-006

US EPA. 2005. Hazardous Waste Companion Database, Microsoft Access™

Although Canadian regulatory authorities do not publish specific guidance for these types of risk assessments, standard MOE, Health Canada, and Environment Canada protocols for contaminated site risk assessment were adopted in this assessment where guidance exists.

The specific scope of this project included:

1. Completion of a multi-exposure pathway human health and ecological risk assessment based on the incremental loading of chemicals emitted from a generic EFW facilty over its estimated 35 year operation period.

2. Examination of the potential human health risk of varying land use scenarios at the maximum point of impingement (MPOI, highest ground level concentration) of air emissions from a generic EFW facility.

3. Determination of whether or not the Ontario MOE *Guideline A-7 Combustion and Air Pollution Requirements for New Municipal Waste Incinerators* emission limits from a generic EFW facility would pose an unacceptable risk to either human or ecological receptors.

4. Provision of information for the public on the potential health and environmental concerns that would be associated with emissions from an EFW facility.

5. Allowance for a model and protocol to be created that would later facilitate the site-specific risk assessment that would be required for the final site and vendor selected.

1.2 Limitations of the Project

This report is in no way meant to replace the requirement for a detailed site specific human health and ecological risk assessment to be conducted upon selection of the preferred site and selected vendor and technology. This report is meant as a feasibility study only and to identify potential issues of concern that should be closely examined during the conduct of the site-specific risk assessment.

Details of limitations of the report are found in each of the human health (Section 5) and ecological (Section 6) risk assessment sections. In addition, study limitations are expanded on in detail in Section 7 of the report.

This report and scope of work did not cover the issues of greenhouse gas (GHG) emissions. This topic has already been previous examined in this EA, with information being reported in the Residual Waste Study - *Annex E-5 Supporting Technical Documentation on Environmental Lifecycle Analysis*. The Annex E-5 also provides some information on relative levels of smog precursors that are emitted from EFW facilities. However, the air dispersion modelling exercise in this report does not address the issues of GHGs or smog formation. This should be undertaken once the final site has been chosen and the specific technology has been selected.

2.0 SELECTION OF CHEMICALS OF POTENTIAL CONCERN

Selection of the chemicals of potential concern (CoPCs) to be evaluated is a critical step in any risk assessment. There are numerous chemicals that are potentially emitted from the stack of an EFW facility. However, given that a specific vendor and technology have not been selected at this point, specific information on chemicals in stack emissions was not available. Figure 2-1 depicts the typical screening that would be employed in a site-specific risk assessment, where facility emissions would be known.

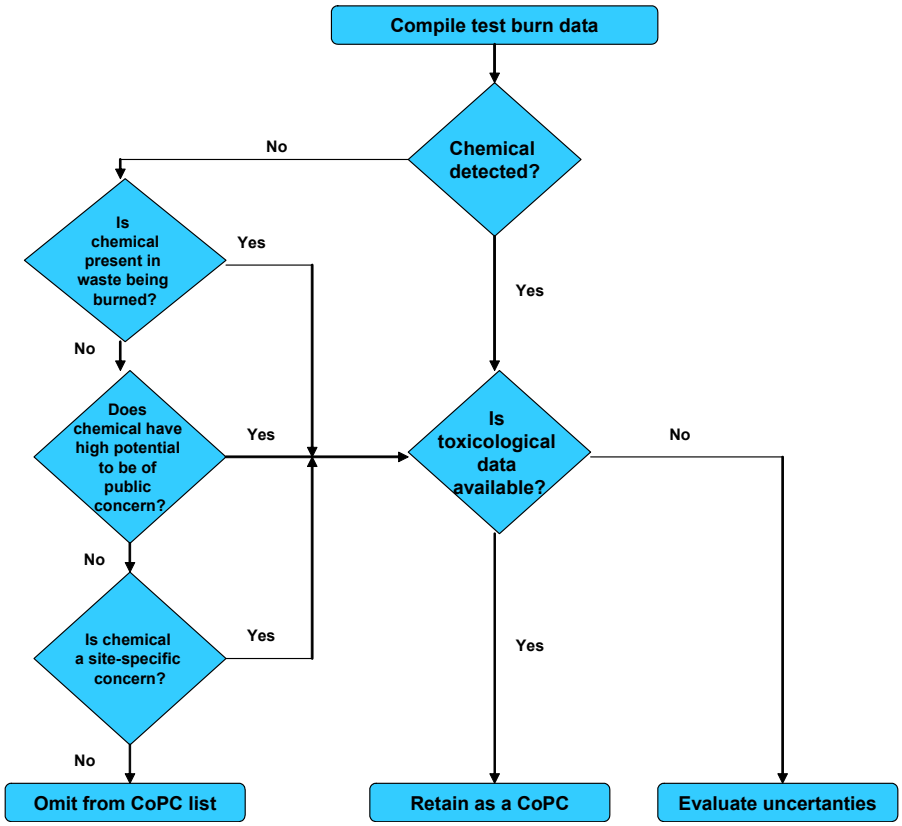


Figure 2-1 Typical Contaminant Screening Process to Develop a Preliminary List of CoPCs

Often in a site-specific risk assessment, those chemicals retained as CoPCs would be further screened based on their emission rate from the facility and their inhalation toxic potential to humans. Those chemicals that contribute to 99% of the cancer or non-cancer risk would then be carried forward into the quantitative assessment for potential risk to human and ecological receptors.

Given the limitation on not having facility specific information, the following sources were examined to derive the CoPC list for this study:

- MOE 1999, Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration
- Cantox Environmental Inc 2000, Human Health Risk Assessment for the Proposed Expansion of the KMS Peel , Inc. Brampton, Energy-From-Waste Facility.
- MOE 2004. Guideline A-7: Combustion and Air Pollution Control Requirements for New Municipal Waste Incinerators
- US EPA 2005, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.

From this list of reports the following list of chemicals of potential concern were derived (Table 2-1).

Table 2-1 Chemicals of Potential Concern from an EFW Facility Evaluated in this Study

Metals	Chlorinated Monocyclic Aromatics	Chlorinated Polycyclic Aromatics	Polycyclic Aromatic Hydrocarbons	Volatile Organic Compounds
Antimony Arsenic✓ Barium Beryllium Boron Cadmium✓+ Chromium ✓ Cobalt Lead✓+ Mercury✓+ Nickel Phosphorus Silver Vanadium Zinc	1,2-Dichlorobenzene 1,2,4-Trichlorobenzene 1,2,4,5-Tetrachlorobenzene Pentachlorobenzene Hexachlorobenzene 2,4-Dichlorophenol 2,4,6-Trichlorophenol 2,3,4,6-Tetrachlorophenol Pentachlorophenol	PCBs 2,3,7,8-TCDD - (dioxin/furan)TEQ✓+ **Combustion Gases** PM_{10}^{+} $PM_{2.5}^{+}$ CO $HCl^{✓+}$ HF $NO_x^{✓+}$ $SO_x^{✓+}$	*Benzo(a)pyrene group* Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(g,h,i)perylene Benzo(k)fluoranthene Chrysene Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Anthracene Napthalene Phenanthrene	Benzene✓ Chloroform Dichloromethane Formaldehyde Tetrachloroethylene Vinyl chloride✓

Notes: Chemical list derived from Cantox Report for Human Health Risk Assessment for the Proposed Expansion of the KMS Peel, Inc. Brampton, Energy-From-Waste Facility (2000)

✓ Chemicals also reviewed by MOE in Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration (1999)

\+ Chemical also included in GUIDELINE A-7 Combustion and Air Pollution Requirements for New Municipal Waste Incinerators (MOE 2004)

Of note is that specific information on DEHP that was evaluated in the Cantox (2000) report could not be located by the air dispersion modeling team, thus was not evaluated in this study. In addition, MOE 1999 chemicals that were not included for further assessment in this report include silicon, iron, tin, and 2-methylfluorene. These chemicals are not commonly evaluated for EFW facilities and were demonstrated by the Cantox screening process not to present a significant contribution to any overall potential risk during their chemical screening process from emission from the KMS Peel facility.

The lack of facility specific data is a limitation of this generic risk assessment that would be dealt with in the site-specific risk assessment through knowledge of technology specific emissions.

3.0 AIR QUALITY BASELINE AND MODELLING

3.1 Facility Overview and Air Dispersion Modelling

In support of the generic human health and ecological risk assessment an air modelling exercise was conducted to model emissions from a theoretical facility for inclusion in the risk assessment. The air modeling results are reported under separate cover "*Report on Air Dispersion Modelling*" (MacViro, 2007) and found in **Appenix I**. The following is a brief description of the facility parameters and air modeling results.

The Residual Waste Study is examining thermal treatment EFW options of processing up to 400,000 t/y of municipal solid waste. It is important to note that the final annual throughput of MSW has yet to be decided. To that end, three facility scenarios were modelled for both their aerial emissions from the stack, as well as for vehiclar truck traffic that would be required to operate the facility. The three scenarios were as follows:

Operating Scenario 1: 3 process units running at full capacity - 400,000 t/y

Operating Scenario 2: 2 process units running at full capacity - 266,666 t/y

Operating Scenario 3: 1 process units running at full capacity - 133,333 t/y

The physical layout of the facility itself was based on the anticipated maximum final stage capacity (400,000 t/y) of the proposed facility, as well as various North American EFW facilities' site characteristics. Figure 3-1 depicts the layout of the theoretical facility, with the facility occupying a 257 m by 240 m property (6.2 ha). The EFW facility was assumed to have a single emissions stack, located 40 m from the fenceline. The height of the exhaust stack was set at 65 m above grade.

3.1.1 Facility Emissions

The majority of the exhaust stack air emission estimates used in this study are based on pollutant emission concentration values obtained from annual stack testing of the 150,000 t/y KMS Peel thermal treatment facility located in Brampton, Ontario. Contaminants of potential concern that were modeled are listed in Table 2-1. The maximum emission concentrations from the 2003, 2004 and 2005 stack testing of the existing facility were prorated for the proposed 400,000 t/y thermal treatment facility and considered for the air dispersion modelling to illustrate a realistic worst-case scenario for the proposed technology.

The Guideline A-7 emission concentration limits were used as default exhaust stack air emission estimates for the eight (8) pollutants contained in the guideline to evaluate the potential risk to the surrounding environment.

Volatile organic compounds are not included in the annual stack testing of the existing facility. The air emissions of these six (6) pollutants were obtained from stack testing performed at the KMS Peel facility in December 1992 and March 1993.

Overall it is believed that the emissions estimated in this generic study are conservative in nature as it is likely that any modern MSW thermal treatment facility to be built would have better pollution control that what was used in the air dispersion modelling. Another level of

conservatism built into the air dispersion model was that all particulate matter above $PM_{2.5}$ would be captured in the air pollution control equipment and that the MOE Guideline A-7 emission concentration limit was comprised of soley $PM_{2.5}$.

In addition to stack emissions, vehicular traffic for waste delivery and ash removal were also considered a source of emissions that could potentially impact human and ecological health. These emissions were modeled based on US EPA MOBILE6.2 emission factors for heavy-duty diesel vehicles for the calendar year 2010.

The waste delivery traffic emission estimates were based on a predicted schedule of eighty-seven delivery trucks per day, with 80% of the deliveries occurring between the hours of 0800 to 1000 and 1400 to 1600, with the remaining 20% of the deliveries occurring between the hours of 1000 to 1400. The ash removal traffic emission estimates were based on a predicated schedule of ten removal trucks per day visiting the site.

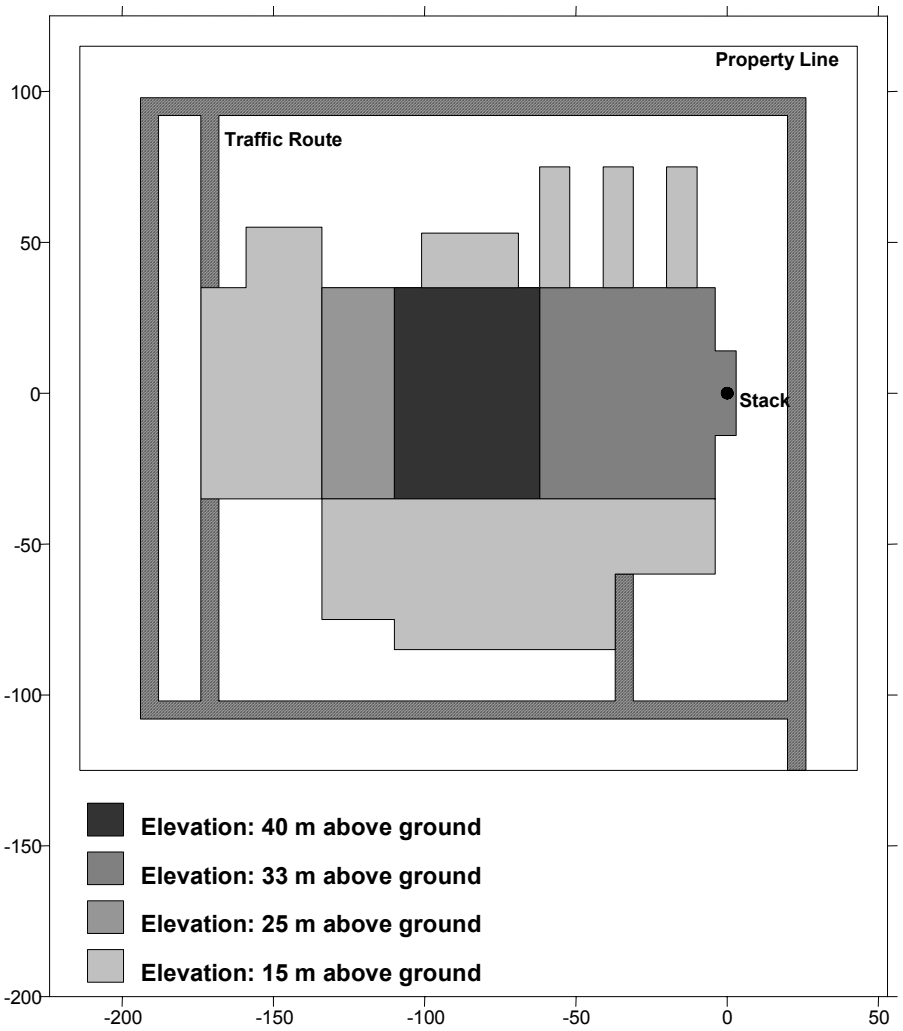


Figure 3-1 Site Plan Schematic of the Theoretical MSW Thermal Treatment Facility

3.1.2 Dispersion Modelling

The MOE approved air dispersion model AERMOD (version 04300) was used together with MOE regional MET files (meteorology) modified to include precipitation for the air dispersion modelling of the emissions released from the proposed MSW thermal treatment facility. Particle phase and vapour phase average concentrations, as well as dry depositions and wet depositions of the selected CoPCs were determined at all ground level receptors.

The air dispersion modelling included estimates of the 1-hour, 24-hour and annual averaging periods of the CoPCs from the facility at the maximum point of impingement (MPOI). Since the location of the proposed thermal treatment facility was unknown at this stage, the land characteristics considered in the modelling exercise were regionally based rather than site specific. The monthly vegetation categories considered for deposition modelling were transitional spring with partial green coverage or short annuals from March to May, midsummer with lush vegetation from June to August, autumn with unharvested cropland for September to November and winter with snow on ground for December to February. Since the topography of Durham and York Regions is relatively flat, a flat terrain was assumed in this generic modelling exercise.

3.1.3 Results of the Air Modelling Exercise

Table 3-1 provides the distances of the MPOI from the stack modeled for each of the three scenarios for the 1-hour, 24-hour and annual averages. The 1-hour maximum ground level concentrations of CoPCs were located approximately 700 m from the EFW facility stack or over 680 m outside the fenceline of the facility, while the 24-hour concentrations were typically located within 300 m of the facility. The annual average MPOI concentrations were located between approximately 280 m and 340 m from the fenceline of the facility.

Table 3-1 Maximum Point of Impingement Concentration from Stack.

Scenario	Distance of MPOI from Stack (m)					
	1-hour		24-hour		Annual Average	
	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour
Scenario 1: 400,000 t/y	728	728	316	316	381	381
Scenario 2: 266,666 t/y	762	762	316	316	381	381
Scenario 3: 133,333 t/y	728	728	316	316	316	316

Note: All distances are approximate as limited by the grid spacing of the air dispersion modelling exercise.

Table 3-2 provides the maximum ground level concentrations of CoPCs modeled as part of the generic facility emissions. As would be expected the highest CoPC concentrations were modeled in Operating Scenario 1, with concentrations decreasing with a decreasing throughput of MSW in the three scenarios. Dry and Wet deposition concentrations of CoPCs are also provided in Table 3-3 and Table 3-4.

Table3-2 Maximum CoPC Ground Level Concentrations (µg/m^3)

Pollutant	Operating Scenario 1: Three (3) process units running 100% of the time						Operating Scenario 2: Two (2) process units running 100% of the time						Operating Scenario 3: One (1) process unit running 100% of the time					
	1-hour average		24-hour average		annual average		1-hour average		24-hour average		annual average		1-hour average		24-hour average		annual average	
	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour
Location of maxima (x,y) ***	(200,700)	(200,700)	(100,-300)	(100,-300)	(150,-350)	(150,-350)	(300,700)	(300,700)	(100,-300)	(100,-300)	(150,-350)	(150,-350)	(200,700)	(200,700)	(100,-300)	(100,-300)	(100,-300)	(100,-300)
Metals																		
Antimony	8.22E-04	0.00E+00	3.65E-04	0.00E+00	3.76E-05	0.00E+00	6.50E-04	0.00E+00	2.79E-04	0.00E+00	3.20E-05	0.00E+00	4.19E-04	0.00E+00	1.69E-04	0.00E+00	2.35E-05	0.00E+00
Arsenic	1.80E-04	1.09E-06	7.99E-05	4.81E-07	8.24E-06	4.97E-08	1.43E-04	8.60E-07	6.11E-05	3.68E-07	7.00E-06	4.22E-08	9.17E-05	5.54E-07	3.70E-05	2.22E-07	5.14E-06	3.10E-08
Barium	1.41E-03	1.28E-05	6.25E-04	5.66E-06	6.45E-05	5.85E-07	1.11E-03	1.01E-05	4.78E-04	4.33E-06	5.48E-05	4.97E-07	7.18E-04	6.52E-06	2.89E-04	2.62E-06	4.02E-05	3.65E-07
Beryllium	1.90E-05	1.73E-07	8.44E-06	7.65E-08	8.71E-07	7.90E-09	1.51E-05	1.37E-07	6.46E-06	5.84E-08	7.40E-07	6.71E-09	9.69E-06	8.80E-08	3.91E-06	3.53E-08	5.43E-07	4.92E-09
Boron	4.59E-02	0.00E+00	2.04E-02	0.00E+00	2.10E-03	0.00E+00	3.63E-02	0.00E+00	1.56E-02	0.00E+00	1.78E-03	0.00E+00	2.34E-02	0.00E+00	9.42E-03	0.00E+00	1.31E-03	0.00E+00
Cadmium *	4.16E-03	3.78E-05	1.85E-03	1.67E-05	1.90E-04	1.73E-06	3.29E-03	2.99E-05	1.41E-03	1.28E-05	1.62E-04	1.47E-06	2.12E-03	1.92E-05	8.55E-04	7.73E-06	1.19E-04	1.08E-06
Chromium	3.78E-04	3.43E-06	1.68E-04	1.52E-06	1.73E-05	1.57E-07	2.99E-04	2.71E-06	1.28E-04	1.16E-06	1.47E-05	1.33E-07	1.92E-04	1.75E-06	7.75E-05	7.01E-07	1.08E-05	9.77E-08
Cobalt	3.54E-05	0.00E+00	1.57E-05	0.00E+00	1.62E-06	0.00E+00	2.80E-05	0.00E+00	1.20E-05	0.00E+00	1.38E-06	0.00E+00	1.80E-05	0.00E+00	7.27E-06	0.00E+00	1.01E-06	0.00E+00
Lead *	4.23E-02	2.98E-04	1.88E-02	1.32E-04	1.94E-03	1.36E-05	3.35E-02	2.36E-04	1.44E-02	1.01E-04	1.64E-03	1.16E-05	2.15E-02	1.52E-04	8.69E-03	6.10E-05	1.21E-03	8.50E-06
Mercury *	9.00E-04	5.10E-03	3.99E-04	2.26E-03	4.12E-05	2.33E-04	7.12E-04	4.03E-03	3.05E-04	1.72E-03	3.50E-05	1.98E-04	4.58E-04	2.60E-03	1.85E-04	1.04E-03	2.57E-05	1.45E-04
Nickel	9.99E-04	9.07E-06	4.43E-04	4.01E-06	4.57E-05	4.15E-07	7.90E-04	7.18E-06	3.39E-04	3.07E-06	3.88E-05	3.52E-07	5.09E-04	4.62E-06	2.05E-04	1.86E-06	2.85E-05	2.59E-07
Phosphorus	6.93E-03	0.00E+00	3.07E-03	0.00E+00	3.17E-04	0.00E+00	5.48E-03	0.00E+00	2.35E-03	0.00E+00	2.69E-04	0.00E+00	3.53E-03	0.00E+00	1.42E-03	0.00E+00	1.98E-04	0.00E+00
Silver	1.40E-04	1.27E-06	6.20E-05	5.61E-07	6.39E-06	5.80E-08	1.11E-04	1.00E-06	4.74E-05	4.29E-07	5.43E-06	4.93E-08	7.12E-05	6.46E-07	2.87E-05	2.60E-07	3.99E-06	3.62E-08
Vanadium	2.01E-04	0.00E+00	8.92E-05	0.00E+00	9.20E-06	0.00E+00	1.59E-04	0.00E+00	6.82E-05	0.00E+00	7.81E-06	0.00E+00	1.02E-04	0.00E+00	4.13E-05	0.00E+00	5.73E-06	0.00E+00
Zinc	1.48E-02	1.19E-04	6.56E-03	5.28E-05	6.77E-04	5.45E-06	1.17E-02	9.44E-05	5.02E-03	4.03E-05	5.75E-04	4.63E-06	7.53E-03	6.07E-05	3.04E-03	2.44E-05	4.22E-04	3.40E-06
Chlorinated Monocyclic Aromatics																		
1,2-Dichlorobenzene	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
1,2,4-Trichlorodibenzene	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
1,2,4,5-Tetrachlorobenzene	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
Pentachlorobenzene	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
Hexachlorobenzene	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
2,4-Dichlorophenol	0.00E+00	3.09E-05	0.00E+00	1.37E-05	0.00E+00	1.41E-06	0.00E+00	2.44E-05	0.00E+00	1.04E-05	0.00E+00	1.20E-06	0.00E+00	1.57E-05	0.00E+00	6.32E-06	0.00E+00	8.81E-07
2,4,6-Trichlorophenol	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
2,3,4,6-Tetrachlorophenol	0.00E+00	1.55E-05	0.00E+00	6.84E-06	0.00E+00	7.06E-07	0.00E+00	1.22E-05	0.00E+00	5.22E-06	0.00E+00	6.00E-07	0.00E+00	7.87E-06	0.00E+00	3.16E-06	0.00E+00	4.40E-07
Pentachlorophenol	1.85E-08	1.85E-05	8.20E-09	8.17E-06	8.46E-10	8.44E-07	1.46E-08	1.46E-05	6.27E-09	6.24E-06	7.18E-10	7.17E-07	9.41E-09	9.40E-06	3.79E-09	3.78E-06	5.27E-10	5.26E-07
Combustion Gases																		
Total Particulate Matter PM ** (see note 1)	7.81E+00	0.00E+00	2.27E+00	0.00E+00	2.35E-01	0.00E+00	7.81E+00	0.00E+00	1.73E+00	0.00E+00	2.00E-01	0.00E+00	7.81E+00	0.00E+00	1.05E+00	0.00E+00	1.48E-01	0.00E+00
Particulate Matter PM10 ** (see note 1)	7.81E+00	0.00E+00	2.27E+00	0.00E+00	2.35E-01	0.00E+00	7.81E+00	0.00E+00	1.73E+00	0.00E+00	2.00E-01	0.00E+00	7.81E+00	0.00E+00	1.05E+00	0.00E+00	1.48E-01	0.00E+00
Particulate Matter PM2.5 */** (see note 1)	6.92E+00	0.00E+00	2.27E+00	0.00E+00	2.35E-01	0.00E+00	6.92E+00	0.00E+00	1.73E+00	0.00E+00	2.00E-01	0.00E+00	6.92E+00	0.00E+00	1.05E+00	0.00E+00	1.48E-01	0.00E+00
Carbon Monoxide ** (see note 2)	0.00E+00	1.61E+01	0.00E+00	3.03E+00	0.00E+00	3.19E-01	0.00E+00	1.61E+01	0.00E+00	2.31E+00	0.00E+00	2.71E-01	0.00E+00	1.61E+01	0.00E+00	1.47E+00	0.00E+00	2.04E-01
Hydrogen Chloride *	0.00E+00	8.10E+00	0.00E+00	3.58E+00	0.00E+00	3.70E-01	0.00E+00	6.41E+00	0.00E+00	2.74E+00	0.00E+00	3.14E-01	0.00E+00	4.12E+00	0.00E+00	1.66E+00	0.00E+00	2.31E-01
Hydrogen Fluoride	0.00E+00	7.23E-03	0.00E+00	3.20E-03	0.00E+00	3.30E-04	0.00E+00	5.72E-03	0.00E+00	2.44E-03	0.00E+00	2.81E-04	0.00E+00	3.68E-03	0.00E+00	1.48E-03	0.00E+00	2.06E-04
Nitrogen Oxides (as NO2) */** (see note 3)	0.00E+00	6.38E+01	0.00E+00	2.76E+01	0.00E+00	2.87E+00	0.00E+00	6.38E+01	0.00E+00	2.12E+01	0.00E+00	2.45E+00	0.00E+00	6.38E+01	0.00E+00	1.28E+01	0.00E+00	1.81E+00
Sulphur Oxides */**	0.00E+00	1.68E+01	0.00E+00	7.46E+00	0.00E+00	7.69E-01	0.00E+00	1.33E+01	0.00E+00	5.69E+00	0.00E+00	6.52E-01	0.00E+00	8.55E+00	0.00E+00	3.45E+00	0.00E+00	4.79E-01
Chlorinated Polycyclic Aromatics																		
PCB	9.75E-08	2.16E-05	4.32E-08	9.54E-06	4.46E-09	9.86E-07	7.71E-08	1.71E-05	3.31E-08	7.29E-06	3.79E-09	8.37E-07	4.96E-08	1.10E-05	2.00E-08	4.41E-06	2.78E-09	6.14E-07
2,3,7,8-TCDD TEQ *	8.07E-09	1.59E-08	3.58E-09	7.05E-09	3.69E-10	7.28E-10	6.38E-09	1.26E-08	2.74E-09	5.39E-09	3.13E-10	6.19E-10	4.11E-09	8.11E-09	1.66E-09	3.26E-09	2.30E-10	4.54E-10
2,3,7,8-TCDD TEQ	1.02E-09	2.01E-09	4.52E-10	8.90E-10	4.66E-11	9.20E-11	8.05E-10	1.59E-09	3.45E-10	6.80E-10	3.96E-11	7.81E-11	5.18E-10	1.02E-09	2.09E-10	4.11E-10	2.90E-11	5.73E-11
Polycyclic Aromatic Hydrocarbons																		
Benzo(a)pyrene	5.68E-06	2.36E-06	2.52E-06	1.05E-06	2.60E-07	1.08E-07	4.49E-06	1.87E-06	1.93E-06	7.99E-07	2.21E-07	9.18E-08	2.89E-06	1.20E-06	1.17E-06	4.83E-07	1.62E-07	6.74E-08
Benzo(a)anthracene	4.16E-06	3.88E-06	1.84E-06	1.72E-06	1.90E-07	1.77E-07	3.29E-06	3.07E-06	1.41E-06	1.31E-06	1.62E-07	1.51E-07	2.12E-06	1.98E-06	8.53E-07	7.94E-07	1.19E-07	1.11E-07
Benzo(b)fluoranthene	2.73E-07	7.77E-06	1.21E-07	3.44E-06	1.25E-08	3.55E-07	2.16E-07	6.14E-06	9.27E-08	2.63E-06	1.06E-08	3.02E-07	1.39E-07	3.95E-06	5.61E-08	1.59E-06	7.80E-09	2.21E-07
Benzo(g,h,i)perylene	5.68E-06	2.36E-06	2.52E-06	1.05E-06	2.60E-07	1.08E-07	4.49E-06	1.87E-06	1.93E-06	7.99E-07	2.21E-07	9.18E-08	2.89E-06	1.20E-06	1.17E-06	4.83E-07	1.62E-07	6.74E-08
Benzo(k)fluoranthene	5.85E-06	2.20E-06	2.59E-06	9.71E-07	2.67E-07	1.00E-07	4.62E-06	1.74E-06	1.98E-06	7.42E-07	2.27E-07	8.52E-08	2.98E-06	1.12E-06	1.20E-06	4.49E-07	1.67E-07	6.25E-08
Chrysene	2.06E-06	5.98E-06	9.13E-07	2.65E-06	9.42E-08	2.73E-07	1.63E-06	4.73E-06	6.98E-07	2.02E-06	8.00E-08	2.32E-07	1.05E-06	3.05E-06	4.23E-07	1.22E-06	5.87E-08	1.70E-07
Dibenzo(a,h)anthracene	7.60E-06	4.42E-07	3.37E-06	1.96E-07	3.48E-07	2.02E-08	6.01E-06	3.50E-07	2.58E-06	1.50E-07	2.95E-07	1.72E-08	3.87E-06	2.25E-07	1.56E-06	9.04E-08	2.17E-07	1.26E-08
Indeno(1,2,3-c,d)pyrene	8.00E-06	4.02E-08	3.55E-06	1.78E-08	3.66E-07	1.84E-09	6.33E-06	3.18E-08	2.71E-06	1.36E-08	3.11E-07	1.56E-09	4.07E-06	2.05E-08	1.64E-06	8.22E-09	2.28E-07	1.15E-09
Anthracene	1.61E-08	8.02E-06	7.13E-09	3.55E-06	7.36E-10	3.67E-07	1.27E-08	6.35E-06	5.46E-09	2.71E-06	6.25E-10	3.12E-07	8.19E-09	4.09E-06	3.30E-09	1.64E-06	4.59E-10	2.29E-07
Naphthalene	0.00E+00	4.92E-05	0.00E+00	2.18E-05	0.00E+00	2.25E-06	0.00E+00	3.89E-05	0.00E+00	1.66E-05	0.00E+00	1.91E-06	0.00E+00	2.51E-05	0.00E+00	1.01E-05	0.00E+00	1.40E-06
Phenanthrene	2.46E-08	2.46E-05	1.09E-08	1.09E-05	1.13E-09	1.12E-06	1.95E-08	1.95E-05	8.36E-09	8.32E-06	9.57E-10	9.55E-07	1.25E-08	1.25E-05	5.06E-09	5.03E-06	7.03E-10	7.01E-07
Volatile Organic Compounds																		
Benzene ** (see note 4)	0.00E+00	1.08E-01	0.00E+00	9.85E-03	0.00E+00	7.10E-04	0.00E+00	1.08E-01	0.00E+00	9.85E-03	0.00E+00	6.10E-04	0.00E+00	1.08E-01	0.00E+00	9.85E-03	0.00E+00	5.30E-04
Chloroform	0.00E+00	1.53E-04	0.00E+00	6.77E-05	0.00E+00	6.99E-06	0.00E+00	1.21E-04	0.00E+00	5.17E-05	0.00E+00	5.94E-06	0.00E+00	7.79E-05	0.00E+00	3.13E-05	0.00E+00	4.36E-06
Dichloromethane	0.00E+00	5.27E-02	0.00E+00	2.33E-02	0.00E+00	2.41E-03	0.00E+00	4.17E-02	0.00E+00	1.78E-02	0.00E+00	2.05E-03	0.00E+00	2.68E-02	0.00E+00	1.08E-02	0.00E+00	1.50E-03
Formaldehyde	0.00E+00	1.42E-02	0.00E+00	6.30E-03	0.00E+00	6.51E-04	0.00E+00	1.13E-02	0.00E+00	4.82E-03	0.00E+00	5.53E-04	0.00E+00	7.25E-03	0.00E+00	2.91E-03	0.00E+00	4.06E-04
Tetrachloroethylene	0.00E+00	1.70E-03	0.00E+00	7.53E-04	0.00E+00	7.77E-05	0.00E+00	1.35E-03	0.00E+00	5.75E-04	0.00E+00	6.60E-05	0.00E+00	8.66E-04	0.00E+00	3.48E-04	0.00E+00	4.85E-05
Vinyl Chloride	0.00E+00	1.79E-04	0.00E+00	7.90E-05	0.00E+00	8.16E-06	0.00E+00	1.41E-04	0.00E+00	6.04E-05	0.00E+00	6.93E-06	0.00E+00	9.09E-05	0.00E+00	3.65E-05	0.00E+00	5.09E-06

* MOE Guideline A-7 criteria
** Includes emissions from facility traffic
*** Relative to stack location (0,0) in metres

NOTES:
1) The maximum 1-hour average concentrations occur at (-80,120) due to large influence of traffic sources
2) The maximum 1-hour average concentrations occur at (-20,120) due to large influence of traffic sources
3) The maximum 1-hour average concentrations occur at (-20,120) due to large influence of traffic sources
4) The maximum 1-hour average concentrations occur at (-20,120) due to large influence of traffic sources

Table3-3 Maximum Dry Deposition Concentrations (g/m^2)

Pollutant	Operating Scenario 1: Three (3) process units running 100% of the time						Operating Scenario 2: Two (2) process units running 100% of the time						Operating Scenario 3: One (1) process unit running 100% of the time					
	1-hour total		24-hour total		annual total		1-hour total		24-hour total		annual total		1-hour total		24-hour total		annual total	
	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour
Metals																		
Antimony	7.17E-08	0.00E+00	2.65E-07	0.00E+00	5.82E-07	0.00E+00	7.38E-08	0.00E+00	2.73E-07	0.00E+00	4.22E-07	0.00E+00	5.06E-08	0.00E+00	1.88E-07	0.00E+00	5.26E-09	0.00E+00
Arsenic	1.57E-08	2.29E-10	5.81E-08	3.10E-09	1.27E-07	4.24E-08	1.62E-08	1.71E-10	5.99E-08	2.30E-09	9.25E-08	3.43E-08	1.11E-08	9.93E-11	4.13E-08	1.34E-09	1.15E-09	2.36E-08
Barium	1.23E-07	2.69E-09	4.54E-07	3.64E-08	9.97E-07	4.99E-07	1.27E-07	2.01E-09	4.69E-07	2.71E-08	7.24E-07	4.04E-07	8.67E-08	1.17E-09	3.23E-07	1.57E-08	9.02E-09	2.78E-07
Beryllium	1.66E-09	3.63E-11	6.13E-09	4.92E-10	1.35E-08	6.73E-09	1.71E-09	2.72E-11	6.33E-09	3.66E-10	9.78E-09	5.46E-09	1.17E-09	1.58E-11	4.36E-09	2.13E-10	1.22E-10	3.75E-09
Boron	4.00E-06	0.00E+00	1.48E-05	0.00E+00	3.25E-05	0.00E+00	4.12E-06	0.00E+00	1.53E-05	0.00E+00	2.36E-05	0.00E+00	2.82E-06	0.00E+00	1.05E-05	0.00E+00	2.94E-07	0.00E+00
Cadmium *	3.63E-07	7.95E-09	1.34E-06	1.08E-07	2.94E-06	1.47E-06	3.74E-07	5.94E-09	1.38E-06	8.00E-08	2.14E-06	1.19E-06	2.56E-07	3.45E-09	9.54E-07	4.65E-08	2.66E-08	8.20E-07
Chromium	3.29E-08	7.21E-10	1.22E-07	9.76E-09	2.67E-07	1.34E-07	3.39E-08	5.39E-10	1.26E-07	7.26E-09	1.94E-07	1.08E-07	2.32E-08	3.13E-10	8.65E-08	4.22E-09	2.42E-09	7.44E-08
Cobalt	3.09E-09	0.00E+00	1.14E-08	0.00E+00	2.50E-08	0.00E+00	3.18E-09	0.00E+00	1.18E-08	0.00E+00	1.82E-08	0.00E+00	2.18E-09	0.00E+00	8.11E-09	0.00E+00	2.27E-10	0.00E+00
Lead *	3.69E-06	6.27E-08	1.36E-05	8.49E-07	2.99E-05	1.16E-05	3.80E-06	4.69E-08	1.41E-05	6.31E-07	2.17E-05	9.41E-06	2.60E-06	2.72E-08	9.70E-06	3.67E-07	2.71E-07	6.47E-06
Mercury *	7.85E-08	2.77E-09	2.90E-07	3.41E-08	6.37E-07	7.60E-07	8.08E-08	1.90E-09	2.99E-07	2.53E-08	4.62E-07	6.23E-07	5.54E-08	1.07E-09	2.06E-07	1.47E-08	5.76E-09	4.32E-07
Nickel	8.71E-08	1.91E-09	3.22E-07	2.58E-08	7.07E-07	3.53E-07	8.97E-08	1.43E-09	3.32E-07	1.92E-08	5.13E-07	2.86E-07	6.15E-08	8.29E-10	2.29E-07	1.12E-08	6.40E-09	1.97E-07
Phosphorus	6.04E-07	0.00E+00	2.23E-06	0.00E+00	4.90E-06	0.00E+00	6.23E-07	0.00E+00	2.30E-06	0.00E+00	3.56E-06	0.00E+00	4.26E-07	0.00E+00	1.59E-06	0.00E+00	4.44E-08	0.00E+00
Silver	1.22E-08	2.67E-10	4.50E-08	3.61E-09	9.89E-08	4.94E-08	1.26E-08	1.99E-10	4.65E-08	2.69E-09	7.18E-08	4.01E-08	8.60E-09	1.16E-10	3.20E-08	1.56E-09	8.95E-10	2.75E-08
Vanadium	1.75E-08	0.00E+00	6.48E-08	0.00E+00	1.42E-07	0.00E+00	1.81E-08	0.00E+00	6.68E-08	0.00E+00	1.03E-07	0.00E+00	1.24E-08	0.00E+00	4.61E-08	0.00E+00	1.29E-09	0.00E+00
Zinc	1.29E-06	2.51E-08	4.77E-06	3.40E-07	1.05E-05	4.65E-06	1.33E-06	1.87E-08	4.92E-06	2.52E-07	7.60E-06	3.77E-06	9.10E-07	1.09E-08	3.39E-06	1.47E-07	9.47E-08	2.59E-06
Chlorinated Monocyclic Aromatics																		
1,2-Dichlorobenzene	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
1,2,4-Trichlorodibenzene	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
1,2,4,5-Tetrachlorobenzene	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
Pentachlorobenzene	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
Hexachlorobenzene	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
2,4-Dichlorophenol	0.00E+00	4.95E-09	0.00E+00	6.85E-08	0.00E+00	6.32E-07	0.00E+00	3.46E-09	0.00E+00	5.10E-08	0.00E+00	5.13E-07	0.00E+00	1.92E-09	0.00E+00	2.96E-08	0.00E+00	3.59E-07
2,4,6-Trichlorophenol	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
2,3,4,6-Tetrachlorophenol	0.00E+00	2.47E-09	0.00E+00	3.43E-08	0.00E+00	3.16E-07	0.00E+00	1.73E-09	0.00E+00	2.55E-08	0.00E+00	2.56E-07	0.00E+00	9.61E-10	0.00E+00	1.48E-08	0.00E+00	1.79E-07
Pentachlorophenol	1.61E-12	2.96E-09	5.96E-12	4.09E-08	1.31E-11	3.77E-07	1.66E-12	2.07E-09	6.15E-12	3.05E-08	9.49E-12	3.06E-07	1.14E-12	1.15E-09	4.24E-12	1.77E-08	1.18E-13	2.14E-07
Combustion Gases																		
Total Particulate Matter PM **	4.45E-04	0.00E+00	1.65E-03	0.00E+00	3.81E-03	0.00E+00	3.05E-04	0.00E+00	1.14E-03	0.00E+00	2.82E-03	0.00E+00	1.57E-04	0.00E+00	5.91E-04	0.00E+00	1.76E-03	0.00E+00
Particulate Matter PM10 **	4.45E-04	0.00E+00	1.65E-03	0.00E+00	3.81E-03	0.00E+00	3.05E-04	0.00E+00	1.14E-03	0.00E+00	2.82E-03	0.00E+00	1.57E-04	0.00E+00	5.91E-04	0.00E+00	1.76E-03	0.00E+00
Particulate Matter PM2.5 */**	4.45E-04	0.00E+00	1.65E-03	0.00E+00	3.62E-03	0.00E+00	3.05E-04	0.00E+00	1.13E-03	0.00E+00	2.63E-03	0.00E+00	1.57E-04	0.00E+00	5.85E-04	0.00E+00	1.53E-03	0.00E+00
Carbon Monoxide **	0.00E+00	4.75E-06	0.00E+00	5.65E-05	0.00E+00	1.24E-03	0.00E+00	3.25E-06	0.00E+00	4.19E-05	0.00E+00	1.01E-03	0.00E+00	1.82E-06	0.00E+00	2.45E-05	0.00E+00	7.08E-04
Hydrogen Chloride *	0.00E+00	1.43E-05	0.00E+00	2.07E-04	0.00E+00	4.56E-03	0.00E+00	9.78E-06	0.00E+00	1.48E-04	0.00E+00	3.65E-03	0.00E+00	5.32E-06	0.00E+00	8.17E-05	0.00E+00	2.42E-03
Hydrogen Fluoride	0.00E+00	1.27E-08	0.00E+00	1.85E-07	0.00E+00	4.07E-06	0.00E+00	8.73E-09	0.00E+00	1.32E-07	0.00E+00	3.26E-06	0.00E+00	4.75E-09	0.00E+00	7.29E-08	0.00E+00	2.16E-06
Nitrogen Oxides (as NO2) */**	0.00E+00	4.34E-05	0.00E+00	5.14E-04	0.00E+00	1.13E-02	0.00E+00	2.97E-05	0.00E+00	3.83E-04	0.00E+00	9.22E-03	0.00E+00	1.67E-05	0.00E+00	2.23E-04	0.00E+00	6.41E-03
Sulphur Oxides */**	0.00E+00	1.17E-05	0.00E+00	1.39E-04	0.00E+00	3.03E-03	0.00E+00	8.02E-06	0.00E+00	1.03E-04	0.00E+00	2.48E-03	0.00E+00	4.50E-06	0.00E+00	6.00E-05	0.00E+00	1.72E-03
Chlorinated Polycyclic Aromatics																		
PCB	8.50E-12	3.45E-09	3.14E-11	4.78E-08	6.90E-11	4.41E-07	8.76E-12	2.41E-09	3.24E-11	3.56E-08	5.01E-11	3.58E-07	6.00E-12	1.34E-09	2.23E-11	2.07E-08	6.24E-13	2.50E-07
2,3,7,8-TCDD TEQ *	7.03E-13	2.55E-12	2.60E-12	3.53E-11	5.71E-12	3.26E-10	7.24E-13	1.78E-12	2.68E-12	2.63E-11	4.14E-12	2.65E-10	4.96E-13	9.91E-13	1.85E-12	1.53E-11	5.16E-14	1.85E-10
2,3,7,8-TCDD TEQ	8.88E-14	3.22E-13	3.28E-13	4.46E-12	7.20E-13	4.11E-11	9.15E-14	2.25E-13	3.39E-13	3.32E-12	5.23E-13	3.34E-11	6.26E-14	1.25E-13	2.33E-13	1.93E-12	6.52E-15	2.34E-11
Polycyclic Aromatic Hydrocarbons																		
Benzo(a)pyrene	4.95E-10	3.94E-10	1.83E-09	5.38E-09	4.02E-09	6.23E-08	5.10E-10	2.94E-10	1.89E-09	4.00E-09	2.92E-09	5.06E-08	3.49E-10	1.71E-10	1.30E-09	2.33E-09	3.63E-11	3.49E-08
Benzo(a)anthracene	3.63E-10	6.47E-10	1.34E-09	8.84E-09	2.94E-09	1.02E-07	3.73E-10	4.83E-10	1.38E-09	6.58E-09	2.14E-09	8.31E-08	2.56E-10	2.81E-10	9.53E-10	3.82E-09	2.66E-11	5.73E-08
Benzo(b)fluoranthene	2.38E-11	1.29E-09	8.81E-11	1.77E-08	1.93E-10	2.05E-07	2.46E-11	9.67E-10	9.09E-11	1.32E-08	1.40E-10	1.66E-07	1.68E-11	5.62E-10	6.27E-11	7.64E-09	1.75E-12	1.15E-07
Benzo(g,h,i)perylene	4.95E-10	3.94E-10	1.83E-09	5.38E-09	4.02E-09	6.23E-08	5.10E-10	2.94E-10	1.89E-09	4.00E-09	2.92E-09	5.06E-08	3.49E-10	1.71E-10	1.30E-09	2.33E-09	3.63E-11	3.49E-08
Benzo(k)fluoranthene	5.10E-10	3.66E-10	1.88E-09	5.00E-09	4.14E-09	5.79E-08	5.25E-10	2.73E-10	1.94E-09	3.72E-09	3.00E-09	4.70E-08	3.60E-10	1.59E-10	1.34E-09	2.16E-09	3.74E-11	3.24E-08
Chrysene	1.80E-10	9.96E-10	6.63E-10	1.36E-08	1.46E-09	1.58E-07	1.85E-10	7.45E-10	6.84E-10	1.01E-08	1.06E-09	1.28E-07	1.27E-10	4.33E-10	4.72E-10	5.89E-09	1.32E-11	8.83E-08
Dibenzo(a,h)anthracene	6.63E-10	7.37E-11	2.45E-09	1.01E-09	5.38E-09	1.17E-08	6.83E-10	5.50E-11	2.53E-09	7.49E-10	3.90E-09	9.46E-09	4.67E-10	3.20E-11	1.74E-09	4.35E-10	4.86E-11	6.52E-09
Indeno(1,2,3-c,d)pyrene	6.98E-10	6.70E-12	2.58E-09	9.16E-11	5.66E-09	1.06E-09	7.19E-10	5.00E-12	2.66E-09	6.81E-11	4.11E-09	8.60E-10	4.92E-10	2.91E-12	1.83E-09	3.96E-11	5.12E-11	5.93E-10
Anthracene	1.40E-12	1.34E-09	5.18E-12	1.83E-08	1.14E-11	2.12E-07	1.44E-12	9.99E-10	5.35E-12	1.36E-08	8.26E-12	1.72E-07	9.89E-13	5.81E-10	3.69E-12	7.90E-09	1.03E-13	1.18E-07
Naphthalene	0.00E+00	8.20E-09	0.00E+00	1.12E-07	0.00E+00	1.30E-06	0.00E+00	6.12E-09	0.00E+00	8.33E-08	0.00E+00	1.05E-06	0.00E+00	3.56E-09	0.00E+00	4.84E-08	0.00E+00	7.26E-07
Phenanthrene	2.15E-12	4.10E-09	7.94E-12	5.60E-08	1.74E-11	6.49E-07	2.21E-12	3.06E-09	8.19E-12	4.17E-08	1.27E-11	5.26E-07	1.52E-12	1.78E-09	5.65E-12	2.42E-08	1.58E-13	3.63E-07
Volatile Organic Compounds																		
Benzene **	0.00E+00	3.19E-06	0.00E+00	3.12E-05	0.00E+00	3.24E-04	0.00E+00	2.22E-06	0.00E+00	2.33E-05	0.00E+00	2.71E-04	0.00E+00	1.29E-06	0.00E+00	1.38E-05	0.00E+00	1.93E-04
Chloroform	0.00E+00	3.38E-08	0.00E+00	3.28E-07	0.00E+00	3.39E-06	0.00E+00	2.36E-08	0.00E+00	2.44E-07	0.00E+00	2.79E-06	0.00E+00	1.24E-08	0.00E+00	1.42E-07	0.00E+00	1.96E-06
Dichloromethane	0.00E+00	1.17E-05	0.00E+00	1.13E-04	0.00E+00	1.17E-03	0.00E+00	8.13E-06	0.00E+00	8.42E-05	0.00E+00	9.62E-04	0.00E+00	4.29E-06	0.00E+00	4.90E-05	0.00E+00	6.77E-04
Formaldehyde	0.00E+00	3.15E-06	0.00E+00	3.05E-05	0.00E+00	3.16E-04	0.00E+00	2.20E-06	0.00E+00	2.27E-05	0.00E+00	2.60E-04	0.00E+00	1.16E-06	0.00E+00	1.33E-05	0.00E+00	1.83E-04
Tetrachloroethylene	0.00E+00	3.76E-07	0.00E+00	3.65E-06	0.00E+00	3.77E-05	0.00E+00	2.62E-07	0.00E+00	2.72E-06	0.00E+00	3.10E-05	0.00E+00	1.38E-07	0.00E+00	1.58E-06	0.00E+00	2.18E-05
Vinyl Chloride	0.00E+00	3.95E-08	0.00E+00	3.83E-07	0.00E+00	3.96E-06	0.00E+00	2.75E-08	0.00E+00	2.85E-07	0.00E+00	3.26E-06	0.00E+00	1.45E-08	0.00E+00	1.66E-07	0.00E+00	2.29E-06

* MOE Guideline A-7 criteria

** Includes emissions from facility traffic

NOTES:

- No adjustments are made to deposition maximas as a result of lake effects (adjustments were not determined)

Table3-4 Maximum CoPC Wet Deposition Concentrations (g/m^2)

Pollutant	Operating Scenario 1: Three (3) process units running 100% of the time						Operating Scenario 2: Two (2) process units running 100% of the time						Operating Scenario 3: One (1) process unit running 100% of the time					
	1-hour total		24-hour total		annual total		1-hour total		24-hour total		annual total		1-hour total		24-hour total		annual total	
	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour	Particulate	Vapour
Metals																		
Antimony	2.75E-09	0.00E+00	2.75E-09	0.00E+00	1.57E-08	0.00E+00	3.01E-09	0.00E+00	4.17E-09	0.00E+00	1.07E-08	0.00E+00	2.33E-09	0.00E+00	2.92E-09	0.00E+00	5.26E-09	0.00E+00
Arsenic	6.03E-10	1.38E-09	6.03E-10	1.40E-09	3.45E-09	3.10E-09	6.58E-10	1.01E-09	9.14E-10	1.01E-09	2.34E-09	2.04E-09	5.11E-10	5.86E-10	6.41E-10	5.86E-10	1.15E-09	9.96E-10
Barium	4.72E-09	1.63E-08	4.72E-09	1.64E-08	2.70E-08	3.65E-08	5.15E-09	1.19E-08	7.15E-09	1.19E-08	1.83E-08	2.41E-08	4.00E-09	6.90E-09	5.01E-09	6.90E-09	9.02E-09	1.17E-08
Beryllium	6.37E-11	2.20E-10	6.37E-11	2.22E-10	3.65E-10	4.93E-10	6.96E-11	1.60E-10	9.66E-11	1.60E-10	2.47E-10	3.25E-10	5.40E-11	9.31E-11	6.77E-11	9.32E-11	1.22E-10	1.58E-10
Boron	1.54E-07	0.00E+00	1.54E-07	0.00E+00	8.79E-07	0.00E+00	1.68E-07	0.00E+00	2.33E-07	0.00E+00	5.95E-07	0.00E+00	1.30E-07	0.00E+00	1.63E-07	0.00E+00	2.94E-07	0.00E+00
Cadmium *	1.39E-08	4.81E-08	1.39E-08	4.85E-08	7.97E-08	1.08E-07	1.52E-08	3.50E-08	2.11E-08	3.50E-08	5.40E-08	7.10E-08	1.18E-08	2.04E-08	1.48E-08	2.04E-08	2.66E-08	3.46E-08
Chromium	1.26E-09	4.36E-09	1.26E-09	4.40E-09	7.23E-09	9.78E-09	1.38E-09	3.18E-09	1.92E-09	3.18E-09	4.90E-09	6.44E-09	1.07E-09	1.85E-09	1.34E-09	1.85E-09	2.42E-09	3.14E-09
Cobalt	1.19E-10	0.00E+00	1.19E-10	0.00E+00	6.78E-10	0.00E+00	1.29E-10	0.00E+00	1.80E-10	0.00E+00	4.59E-10	0.00E+00	1.00E-10	0.00E+00	1.26E-10	0.00E+00	2.27E-10	0.00E+00
Lead *	1.42E-07	3.80E-07	1.42E-07	3.83E-07	8.10E-07	8.51E-07	1.55E-07	2.76E-07	2.15E-07	2.76E-07	5.49E-07	5.60E-07	1.20E-07	1.61E-07	1.50E-07	1.61E-07	2.71E-07	2.73E-07
Mercury *	3.01E-09	9.48E-14	3.01E-09	9.48E-14	1.72E-08	1.47E-13	3.29E-09	6.90E-14	4.57E-09	6.90E-14	1.17E-08	9.77E-14	2.55E-09	4.02E-14	3.20E-09	4.02E-14	5.76E-09	5.17E-14
Nickel	3.35E-09	1.15E-08	3.35E-09	1.16E-08	1.91E-08	2.59E-08	3.65E-09	8.40E-09	5.07E-09	8.40E-09	1.30E-08	1.71E-08	2.83E-09	4.89E-09	3.55E-09	4.89E-09	6.40E-09	8.31E-09
Phosphorus	2.32E-08	0.00E+00	2.32E-08	0.00E+00	1.33E-07	0.00E+00	2.53E-08	0.00E+00	3.52E-08	0.00E+00	8.99E-08	0.00E+00	1.97E-08	0.00E+00	2.47E-08	0.00E+00	4.44E-08	0.00E+00
Silver	4.68E-10	1.62E-09	4.68E-10	1.63E-09	2.68E-09	3.62E-09	5.11E-10	1.18E-09	7.09E-10	1.18E-09	1.81E-09	2.39E-09	3.96E-10	6.84E-10	4.97E-10	6.84E-10	8.95E-10	1.16E-09
Vanadium	6.73E-10	0.00E+00	6.73E-10	0.00E+00	3.85E-09	0.00E+00	7.35E-10	0.00E+00	1.02E-09	0.00E+00	2.61E-09	0.00E+00	5.70E-10	0.00E+00	7.15E-10	0.00E+00	1.29E-09	0.00E+00
Zinc	4.95E-08	1.52E-07	4.95E-08	1.53E-07	2.83E-07	3.40E-07	5.41E-08	1.10E-07	7.51E-08	1.10E-07	1.92E-07	2.24E-07	4.20E-08	6.43E-08	5.26E-08	6.43E-08	9.47E-08	1.09E-07
Chlorinated Monocyclic Aromatics																		
1,2-Dichlorobenzene	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
1,2,4-Trichlorodibenzene	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
1,2,4,5-Tetrachlorobenzene	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
Pentachlorobenzene	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
Hexachlorobenzene	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
2,4-Dichlorophenol	0.00E+00	4.56E-11	0.00E+00	4.56E-11	0.00E+00	7.22E-11	0.00E+00	3.32E-11	0.00E+00	3.32E-11	0.00E+00	4.76E-11	0.00E+00	1.93E-11	0.00E+00	1.93E-11	0.00E+00	2.60E-11
2,4,6-Trichlorophenol	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
2,3,4,6-Tetrachlorophenol	0.00E+00	2.28E-11	0.00E+00	2.28E-11	0.00E+00	3.61E-11	0.00E+00	1.66E-11	0.00E+00	1.66E-11	0.00E+00	2.38E-11	0.00E+00	9.66E-12	0.00E+00	9.66E-12	0.00E+00	1.30E-11
Pentachlorophenol	6.19E-14	2.73E-11	6.19E-14	2.73E-11	3.54E-13	4.32E-11	6.76E-14	1.98E-11	9.38E-14	1.98E-11	2.40E-13	2.84E-11	5.24E-14	1.15E-11	6.57E-14	1.15E-11	1.18E-13	1.56E-11
Combustion Gases																		
Total Particulate Matter PM **	1.72E-05	0.00E+00	2.51E-05	0.00E+00	1.05E-04	0.00E+00	1.25E-05	0.00E+00	1.77E-05	0.00E+00	7.30E-05	0.00E+00	7.31E-06	0.00E+00	9.60E-06	0.00E+00	3.88E-05	0.00E+00
Particulate Matter PM10 **	1.72E-05	0.00E+00	2.51E-05	0.00E+00	1.05E-04	0.00E+00	1.25E-05	0.00E+00	1.77E-05	0.00E+00	7.30E-05	0.00E+00	7.31E-06	0.00E+00	9.60E-06	0.00E+00	3.88E-05	0.00E+00
Particulate Matter PM2.5 */**	1.71E-05	0.00E+00	2.47E-05	0.00E+00	9.78E-05	0.00E+00	1.24E-05	0.00E+00	1.72E-05	0.00E+00	6.61E-05	0.00E+00	7.23E-06	0.00E+00	9.07E-06	0.00E+00	3.27E-05	0.00E+00
Carbon Monoxide **	0.00E+00	8.93E-07	0.00E+00	1.12E-06	0.00E+00	4.09E-06	0.00E+00	6.48E-07	0.00E+00	7.77E-07	0.00E+00	2.76E-06	0.00E+00	3.77E-07	0.00E+00	4.07E-07	0.00E+00	1.37E-06
Hydrogen Chloride *	0.00E+00	3.99E-06	0.00E+00	4.98E-06	0.00E+00	1.82E-05	0.00E+00	2.90E-06	0.00E+00	3.48E-06	0.00E+00	1.23E-05	0.00E+00	1.69E-06	0.00E+00	1.82E-06	0.00E+00	6.12E-06
Hydrogen Fluoride	0.00E+00	3.56E-09	0.00E+00	4.44E-09	0.00E+00	1.63E-08	0.00E+00	2.59E-09	0.00E+00	3.11E-09	0.00E+00	1.10E-08	0.00E+00	1.51E-09	0.00E+00	1.62E-09	0.00E+00	5.47E-09
Nitrogen Oxides (as NO2) */**	0.00E+00	8.16E-06	0.00E+00	1.02E-05	0.00E+00	3.73E-05	0.00E+00	5.93E-06	0.00E+00	7.12E-06	0.00E+00	2.53E-05	0.00E+00	3.45E-06	0.00E+00	3.72E-06	0.00E+00	1.25E-05
Sulphur Oxides */**	0.00E+00	2.21E-06	0.00E+00	2.75E-06	0.00E+00	1.01E-05	0.00E+00	1.60E-06	0.00E+00	1.92E-06	0.00E+00	6.82E-06	0.00E+00	9.34E-07	0.00E+00	1.01E-06	0.00E+00	3.39E-06
Chlorinated Polycyclic Aromatics																		
PCB	3.26E-13	3.18E-11	3.26E-13	3.18E-11	1.87E-12	5.04E-11	3.56E-13	2.32E-11	4.95E-13	2.32E-11	1.26E-12	3.32E-11	2.77E-13	1.35E-11	3.47E-13	1.35E-11	6.24E-13	1.82E-11
2,3,7,8-TCDD TEQ *	2.70E-14	2.35E-14	2.70E-14	2.35E-14	1.54E-13	3.73E-14	2.95E-14	1.71E-14	4.09E-14	1.71E-14	1.05E-13	2.45E-14	2.29E-14	9.97E-15	2.87E-14	9.97E-15	5.16E-14	1.34E-14
2,3,7,8-TCDD TEQ	3.41E-15	2.97E-15	3.41E-15	2.97E-15	1.95E-14	4.70E-15	3.72E-15	2.16E-15	5.17E-15	2.16E-15	1.32E-14	3.10E-15	2.89E-15	1.26E-15	3.62E-15	1.26E-15	6.52E-15	1.70E-15
Polycyclic Aromatic Hydrocarbons																		
Benzo(a)pyrene	1.90E-11	2.48E-10	1.90E-11	2.48E-10	1.09E-10	3.94E-10	2.08E-11	1.81E-10	2.88E-11	1.81E-10	7.36E-11	2.60E-10	1.61E-11	1.05E-10	2.02E-11	1.05E-10	3.63E-11	1.42E-10
Benzo(a)anthracene	1.39E-11	4.08E-10	1.39E-11	4.08E-10	7.96E-11	6.47E-10	1.52E-11	2.97E-10	2.11E-11	2.97E-10	5.39E-11	4.26E-10	1.18E-11	1.73E-10	1.48E-11	1.73E-10	2.66E-11	2.33E-10
Benzo(b)fluoranthene	9.16E-13	8.16E-10	9.16E-13	8.16E-10	5.24E-12	1.29E-09	9.99E-13	5.94E-10	1.39E-12	5.94E-10	3.54E-12	8.53E-10	7.76E-13	3.46E-10	9.72E-13	3.46E-10	1.75E-12	4.66E-10
Benzo(g,h,i)perylene	1.90E-11	2.48E-10	1.90E-11	2.48E-10	1.09E-10	3.94E-10	2.08E-11	1.81E-10	2.88E-11	1.81E-10	7.36E-11	2.60E-10	1.61E-11	1.05E-10	2.02E-11	1.05E-10	3.63E-11	1.42E-10
Benzo(k)fluoranthene	1.96E-11	2.31E-10	1.96E-11	2.31E-10	1.12E-10	3.66E-10	2.14E-11	1.68E-10	2.97E-11	1.68E-10	7.58E-11	2.41E-10	1.66E-11	9.77E-11	2.08E-11	9.77E-11	3.74E-11	1.32E-10
Chrysene	6.89E-12	6.29E-10	6.89E-12	6.29E-10	3.94E-11	9.97E-10	7.52E-12	4.58E-10	1.04E-11	4.58E-10	2.67E-11	6.57E-10	5.84E-12	2.66E-10	7.32E-12	2.66E-10	1.32E-11	3.59E-10
Dibenzo(a,h)anthracene	2.54E-11	4.65E-11	2.54E-11	4.65E-11	1.46E-10	7.37E-11	2.78E-11	3.38E-11	3.86E-11	3.38E-11	9.85E-11	4.86E-11	2.16E-11	1.97E-11	2.70E-11	1.97E-11	4.86E-11	2.65E-11
Indeno(1,2,3-c,d)pyrene	2.68E-11	4.23E-12	2.68E-11	4.23E-12	1.53E-10	6.70E-12	2.92E-11	3.07E-12	4.06E-11	3.08E-12	1.04E-10	4.41E-12	2.27E-11	1.79E-12	2.85E-11	1.79E-12	5.12E-11	2.41E-12
Anthracene	5.39E-14	8.43E-10	5.39E-14	8.44E-10	3.08E-13	1.34E-09	5.88E-14	6.14E-10	8.16E-14	6.14E-10	2.09E-13	8.81E-10	4.56E-14	3.57E-10	5.72E-14	3.57E-10	1.03E-13	4.82E-10
Naphthalene	0.00E+00	5.17E-09	0.00E+00	5.17E-09	0.00E+00	8.20E-09	0.00E+00	3.76E-09	0.00E+00	3.76E-09	0.00E+00	5.40E-09	0.00E+00	2.19E-09	0.00E+00	2.19E-09	0.00E+00	2.95E-09
Phenanthrene	8.25E-14	2.59E-09	8.25E-14	2.59E-09	4.72E-13	4.10E-09	9.00E-14	1.88E-09	1.25E-13	1.88E-09	3.19E-13	2.70E-09	6.99E-14	1.10E-09	8.76E-14	1.10E-09	1.58E-13	1.48E-09
Volatile Organic Compounds																		
Benzene **	0.00E+00	2.54E-09	0.00E+00	2.54E-09	0.00E+00	4.03E-09	0.00E+00	1.85E-09	0.00E+00	1.85E-09	0.00E+00	2.65E-09	0.00E+00	1.08E-09	0.00E+00	1.08E-09	0.00E+00	1.45E-09
Chloroform	0.00E+00	2.70E-11	0.00E+00	2.70E-11	0.00E+00	4.28E-11	0.00E+00	1.97E-11	0.00E+00	1.97E-11	0.00E+00	2.82E-11	0.00E+00	1.14E-11	0.00E+00	1.14E-11	0.00E+00	1.54E-11
Dichloromethane	0.00E+00	9.31E-09	0.00E+00	9.31E-09	0.00E+00	1.47E-08	0.00E+00	6.77E-09	0.00E+00	6.77E-09	0.00E+00	9.71E-09	0.00E+00	3.94E-09	0.00E+00	3.94E-09	0.00E+00	5.31E-09
Formaldehyde	0.00E+00	2.52E-09	0.00E+00	2.52E-09	0.00E+00	3.98E-09	0.00E+00	1.83E-09	0.00E+00	1.83E-09	0.00E+00	2.62E-09	0.00E+00	1.07E-09	0.00E+00	1.07E-09	0.00E+00	1.44E-09
Tetrachloroethylene	0.00E+00	3.00E-10	0.00E+00	3.00E-10	0.00E+00	4.76E-10	0.00E+00	2.19E-10	0.00E+00	2.19E-10	0.00E+00	3.13E-10	0.00E+00	1.27E-10	0.00E+00	1.27E-10	0.00E+00	1.72E-10
Vinyl Chloride	0.00E+00	3.15E-11	0.00E+00	3.15E-11	0.00E+00	4.99E-11	0.00E+00	2.29E-11	0.00E+00	2.29E-11	0.00E+00	3.29E-11	0.00E+00	1.34E-11	0.00E+00	1.34E-11	0.00E+00	1.80E-11

* MOE Guideline A-7 criteria

** Includes emissions from facility traffic

NOTES:

- No adjustments are made to deposition maximas as a result of lake effects (adjustments were not determined)

3.1.4 Local Background Air Quality

Although the specific location of the facility was unknown at the time of preparation of this report, there are several MOE ambient air quality stations located within the Durham and York Regions (Table 3-5). These ambient air quality stations were used to assess the potential background, existing ambient air quality for the Regions. In the case of SO_2 there were no stations located within the study area that measured SO_2; therefore, the concentration of SO_2 from the Mississauga station was used.

Table3-5 Ambient Monitoring Data for 2004 from Durham / York MOE Air Monitoring Stations.

Station Number	Station ID	$PM_{2.5}$ (ug/m^3)		NO_2 (ug/m^3)		SO_2 (ug/m^3)		CO (ug/m^3)	
		90% Hourly	Annual	90% Hourly	Annual	90% Hourly	Annual	90% Hourly	Annual
45025	Newmarket	20	7.7	35.8	16.0			722	470
19114	Oshawa	20	8.1	26.3					
48002	Stouffville								
46109	Mississauga					13.1	6.8		
Background Values for Study		**20**	**8.1**	**35.8**	**16.0**	**13.1**	**6.8**	**722**	**470**

Notes:
1. Data for AQ Stations in Durham/York used unless there were no stations measuring a specific compound.
2. the 90th % (percentile) hourly data is used as short-term average background concentrations
3. The annual average concentration for long-term background concentrations
4. Based on the 2004 monitoring data, as 2005 data is not available for the selected stations.

The highest concentration from each of the three Durham/York stations was used to estimate the local ambient air quality for the study area. Given that the site specific location is not yet known, this is the most reasonable approach to determining the ambient air concentration for the study area. Unfortunately, volatile organic compound (VOC) concentrations were only available for the Stoffville station. Not all of the current study CoPCs were included in the Stoffville data set; however, those CoPCs that were reported were included as the existing ambient air quality for the generic risk assessment study.

These background or ambient concentrations of CoPCs were used to evaluate the potential cumulative risk of exposure to airborne contaminants in this study. The authors recognize that this was only a very limited review of potential background air concentrations in the Durham and York Regions. Other monitoring stations may exist, as well as additional data. However, in anticipation of the regulatory requirements and need for a site-specific risk assessment, plans are being developed by the EA study team to establish a network of air monitoring stations that encompass the short listed sites.

3.2 Air Quality Criteria

3.2.1 Ontario Air Quality Criteria

Ambient air quality criteria are established to define desired environmental quality that will protect public health and ecosystems. Depending on jurisdiction, air quality criteria can be referred to as objectives, guidelines or effects screening levels. Air quality criteria are generally established for 1-hour, 24-hour and annual mean averaging periods.

Ontario Regulation 419/05 falls under the *Environmental Protection Act* (*EPA*) and is the primary regulatory tool for creating standards for contaminants "that are protective of local air quality" in Ontario (MOE, 2005). The MOE has approached the development of the standards by developing AAQCs, or effects based levels in air, of varying averaging times (1-hour, 24-hour and annual) appropriate for the effects. The effects considered may be based on human health or on effects including odour, vegetation, soiling, visibility, corrosion, etc. To derive the POI guidelines, the most conservative half-hour value derived from AAQCs of variable averaging times was selected. MOE O.Reg. 419 Schedule 3 values are those that are specific to the type of EFW facility being considered in this EA and are based on a 24-hr value.

The modelled air results at the MPOI for Operating Scenario 1 (400,000 t/y) are provided in Table 3-6. Concentrations of all CoPCs were below the MOE criteria, even with the addition of ambient concentrations from Durham/York air quality monitoring stations. This was also true of the modeled air concentrations for Operating Scenario 2 and 3. This is a critical item in this generic assessment, because if the MPOI concentrations modeled did not meet these standard then the MOE would not grant a Certificate of Approval to operate this type of facility.

Again this section reviews only the air quality against MOE standards and regulations. Not all of the standards are health based, and in some cases those that are have additional inhalation toxicology known about them. Chronic exposure inhalation risk to human receptors is captured in the human health risk assessment. In addition, Section 5.0 of this report includes screening for potential acute risk and chronic exposure evaluation for the combustion gases.

Table 3-6 Comparison of Modelled Air Results to Ontario MOE O.Reg. 419 Schedule 3 and Ambient Air Quality Criteria

Pollutant	MOE Sch.3	MOE AAQC	MOE AAQC	Background		Operating Scenario 1: Three (3) process units running 100% of the time				
	24-hour	24-hour	1-hour	90th Percentile	Annual	1-hr average	24-hour average		annual average	
				(24-hour equiv.)		Total	Total	**Total + Bgd**	Total	**Total + Bgd**
	($\mu g/m^3$)									
Metals										
Antimony	25	--	--			8.22E-04	3.65E-04	3.65E-04	3.76E-05	3.76E-05
Arsenic	--	0.3	--			1.81E-04	8.04E-05	8.04E-05	8.29E-06	8.29E-06
Barium	--	10	--			1.42E-03	6.31E-04	6.31E-04	6.51E-05	6.51E-05
Beryllium	0.01	--	--			1.92E-05	8.52E-06	8.52E-06	8.79E-07	8.79E-07
Boron	--	120 [a]	--			4.59E-02	2.04E-02	2.04E-02	2.10E-03	2.10E-03
Cadmium	2	--	--			4.20E-03	1.86E-03	1.86E-03	1.92E-04	1.92E-04
Chromium (trivalent)	--	1.5	--			3.81E-04	1.69E-04	1.69E-04	1.74E-05	1.74E-05
Cobalt	--	0.1	--			3.54E-05	1.57E-05	1.57E-05	1.62E-06	1.62E-06
Lead	2	--	--			4.26E-02	1.89E-02	1.89E-02	1.95E-03	1.95E-03
Mercury	2	--	--			6.00E-03	2.66E-03	2.66E-03	2.74E-04	2.74E-04
Nickel	2 [b]	--	--			1.01E-03	4.47E-04	4.47E-04	4.61E-05	4.61E-05
Phosphorus	--	--	--			6.93E-03	3.07E-03	3.07E-03	3.17E-04	3.17E-04
Silver	--	1	--			1.41E-04	6.26E-05	6.26E-05	6.45E-06	6.45E-06
Vanadium	2	--	--			2.01E-04	8.92E-05	8.92E-05	9.20E-06	9.20E-06
Zinc	120 [a]	--	--			1.49E-02	6.62E-03	6.62E-03	6.82E-04	6.82E-04
Chlorinated Monocyclic Aromatics										
1,2-Dichlorobenzene	--	--	30500			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
1,2,4-Trichlorobenzene	--	400	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
1,2,4,5-Tetrachlorobenzene	--	--	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
Pentachlorobenzene	--	--	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
Hexachlorobenzene	--	--	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
2,4-Dichlorophenol	--	--	--			3.09E-05	1.37E-05	1.37E-05	1.41E-06	1.41E-06
2,4,6-Trichlorophenol	--	--	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
2,3,4,6-Tetrachlorophenol	--	--	--			1.55E-05	6.84E-06	6.84E-06	7.06E-07	7.06E-07
Pentachlorophenol	--	20	--			1.85E-05	8.18E-06	8.18E-06	8.45E-07	8.45E-07
Combustion Gases										
Particulate Matter PM10	--	--	--			7.81E+00	2.27E+00	2.27E+00	2.35E-01	2.35E-01
Particulate Matter PM2.5	--	--	--	20.0	8.1	6.92E+00	2.27E+00	2.23E+01	2.35E-01	8.33E+00
Carbon Monoxide	--	--	--	721.7	469.7	1.61E+01	3.03E+00	7.25E+02	3.19E-01	4.70E+02

Table 3-6 Comparison of Modelled Air Results to Ontario MOE O.Reg. 419 Schedule 3 and Ambient Air Quality Criteria

Pollutant	MOE Sch.3	MOE AAQC	MOE AAQC	Background		Operating Scenario 1: Three (3) process units running 100% of the time				
	24-hour	24-hour	1-hour	90th Percentile	Annual	1-hr average	24-hour average		annual average	
				(24-hour equiv.)		Total	Total	**Total + Bgd**	Total	**Total + Bgd**
	($\mu g/m^3$)									
Hydrogen Chloride	20	--	--			8.10E+00	3.58E+00	3.58E+00	3.70E-01	3.70E-01
Hydrogen Fluoride	--	--	--			7.23E-03	3.20E-03	3.20E-03	3.30E-04	3.30E-04
Nitrogen Oxides	200	--	--	45.0	22.0	6.38E+01	2.76E+01	7.26E+01	2.87E+00	2.48E+01
Sulphur Oxides [c]	275 [d]	--	--			1.68E+01	7.46E+00	7.46E+00	7.69E-01	7.69E-01
Chlorinated Polycyclic Aromatics										
PCB	--	0.15	--			2.17E-05	9.58E-06	9.58E-06	9.90E-07	9.90E-07
2,3,7,8-TCDD TEQ	--	5.00E-06	--			2.40E-08	1.06E-08	1.06E-08	1.10E-09	1.10E-09
Polycyclic Aromatic Hydrocarbons										
Benzo(a)pyrene	--	1.10E-03	--			8.04E-06	3.56E-06	3.56E-06	3.68E-07	3.68E-07
Benzo(a)anthracene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.68E-07	3.68E-07
Benzo(b)fluoranthene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.67E-07	3.67E-07
Benzo(g,h,I)perylene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.68E-07	3.68E-07
Benzo(k)fluoranthene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.68E-07	3.68E-07
Chrysene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.68E-07	3.68E-07
Dibenzo(a,h)anthracene	--	--	--			8.04E-06	3.57E-06	3.57E-06	3.68E-07	3.68E-07
Indeno(1,2,3-c,d)pyrene	--	--	--			8.04E-06	3.57E-06	3.57E-06	3.68E-07	3.68E-07
Anthracene	--	--	--			8.04E-06	3.56E-06	3.56E-06	3.67E-07	3.67E-07
Naphthalene	--	22.5	--	0.26	0.101	4.92E-05	2.18E-05	2.60E-01	2.25E-06	1.01E-01
Phenanthrene	--	--	--			2.46E-05	1.09E-05	1.09E-05	1.13E-06	1.13E-06
Volatile Organic Compounds										
Benzene	--	--	--			1.08E-01	9.85E-03	9.85E-03	7.10E-04	7.10E-04
Chloroform	1	--	--	0.113	0.088	1.53E-04	6.77E-05	1.13E-01	6.99E-06	8.80E-02
Dichloromethane	--	--	--			5.27E-02	2.33E-02	2.33E-02	2.41E-03	2.41E-03
Formaldehyde	65	--	--			1.42E-02	6.30E-03	6.30E-03	6.51E-04	6.51E-04
Tetrachloroethylene	--	--	--	0.503	0.1	1.70E-03	7.53E-04	5.04E-01	7.77E-05	1.00E-01
Vinyl Chloride	1	--	--			1.79E-04	7.90E-05	7.90E-05	8.16E-06	8.16E-06

NOTES:
[a] particulate-based value
[b] vegetation-based value
[c] sulphur dioxide value used
[d] health and vegetation-based value

4.0 EXPOSURE POINT CONCENTRATIONS

4.1 CoPC Air Concentrations and Deposition Rates

Air quality was assessed in the context of facility-related emissions and ground-level concentrations of the substances of interest. Air emissions of concern from the facility include criteria air contaminants (CACs), volatile organic compounds (VOCs), chlorinated monocyclic aromatics, chlorinated polycyclic aromatics, polycyclic aromatic hydrocarbons (PAHs), and trace metals. All of these substances have the potential to affect ambient air quality in the region surrounding the facility.

The major source of air emissions associated with the operation of an EFW thermal treatment facility from the exhaust stack. However, to ensure that all potential sources of impact were considered, air emissions from waste delivery and ash removal on-site traffic were also considered for commonly emitted pollutants in the air dispersion modelling of the proposed facility.

An air dispersion modelling assessment was conducted using the MOE approved air dispersion model AERMOD to determine the maximum ground level concentrations, as well as wet and dry depositions.

Again the following three operating scenarios were considered:

Operating Scenario 1: 3 process units running at full capacity - 400,000 t/y

Operating Scenario 2: 2 process units running at full capacity - 266,666 t/y

Operating Scenario 3: 1 process units running at full capacity - 133,333 t/y

Maximum predicted ground-level concentrations and wet and dry deposition rates of each CoPC at the MPOI were calculated and carried forward into the HHERA. Details of these predicted ground-level concentrations and deposition rates and predicted chemical release concentrations in water are found in the Report on Air Dispersion Modelling (MacViro, 2007), submitted under separate cover and included in **Appendix I** of this report.

4.2 Predicting Multi-Media Exposure Point Concentrations

This section describes the methodologies used in estimating exposure point concentrations (EPCs) of CoPCs in each exposure pathway. The equations and references used in calculating CoPC-specific EPCs are presented in **Appendix D**, with a brief description provided in the following sections .

In accordance with US EPA (2005), CoPC are grouped into three broad categories for the assessment of potential exposure pathways, as follows:

- organics (e.g., polycyclic aromatic hydrocarbons [PAHs]);
- metals (excluding mercury); and
- mercuric compounds (Hg^{2+}, Hg^{0}, MeHg).

Each CoPC category is evaluated on an exposure pathway specific basis. For instance, elemental mercury (Hg^0) is assessed for direct inhalation exposure but is not included in possible food chain uptakes.

This risk assessment has evaluated exposure to three mercury species via varied pathways:

1. Elemental mercury (Hg^0) has been assessed only through direct inhalation of the vapour phase;
2. Divalent mercury (Hg^{2+}) has been assessed through both direct inhalation and indirect exposure to vapour and particle-bound mercuric chloride; and,
3. Methyl mercury (MHg) has been assessed only through indirect exposure.

US EPA (2005) states that air emissions of mercury contribute to local, regional, and global deposition. A portion of anthropogenic releases is in the form of elemental mercury and a portion in the oxidized form (e.g., Hg^{2+}). Total mercury exiting the stack is assumed to consist almost entirely of elemental and divalent species, with no emissions of methyl mercury. Much of the divalent mercury is thought to be mercuric chloride ($HgCl_2$) (US EPA, 1997).

As presented in US EPA (2005), based on a review of mercury emissions data presented for combustion sources in US EPA (1997) and published literature (Peterson et al., 1995), estimates for the percentage of vapour and particle-bound mercury emissions range widely from 20 to 80 percent. Therefore, in the absence of site-specific mercury sampling, a protective approach that assumes phase allocation of mercury emissions from waste combustion of 80 percent of total mercury in the vapour phase and 20 percent of total mercury in the particle-bound phase has been adopted. Section D.1.5.8 **(Appendix D)** discusses this issue further, and Figure D.1 (**Appendix D**) illustrates the phase allocation and speciation of mercury in air. This is generally presented below:

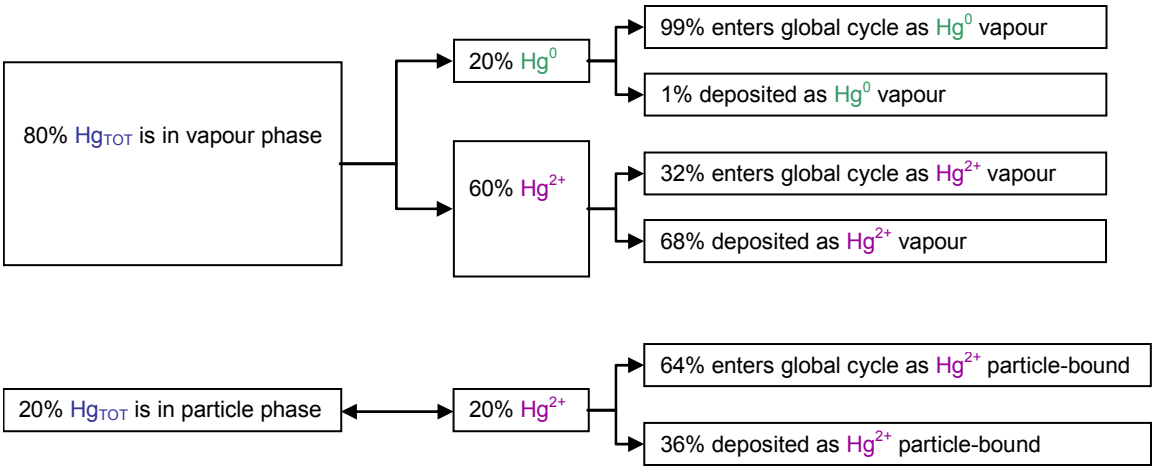


Figure 4-1 Phase allocation and speciation of mercury from stack emissions

This allocation is:

- Consistent with mercury emissions speciation data for hazardous waste combustions sources reported in the literature; and
- Believed to be reasonably protective, since it results in the highest percentage of total mercury being deposited in proximity to the source, and therefore, indicative of the maximum indirect risk.

Using this allocation, the percentage of total mercury deposited would be reduced to a total of 48.2 percent (40.8% as divalent vapour, 7.2% as divalent particle-bound, and 0.2% as elemental vapour). Also, to account for the remaining 51.8 percent of the total mercury mass that is not deposited, the deposition and media concentration equations for air (**Appendix D**, Section D.1.1) are multiplied by a mercury factor of 0.002 for elemental mercury and 0.482 for divalent mercury.

Methylation of Mercury

Based on the information in US EPA 1997, we have assumed that 98 percent of the deposited mercury remains divalent mercury, and two percent speciates to organic (or methyl) mercury in soil.

Both watershed erosion and direct atmospheric deposition can be important sources of mercury to a water body. A portion of the total mercury deposited into a waterbody is then assumed to be converted into the organic form – methyl mercury – in the water. Both forms of mercury are accumulated into fish at different rates and fish tissue mercury is assumed to be converted to 100% methyl mercury.

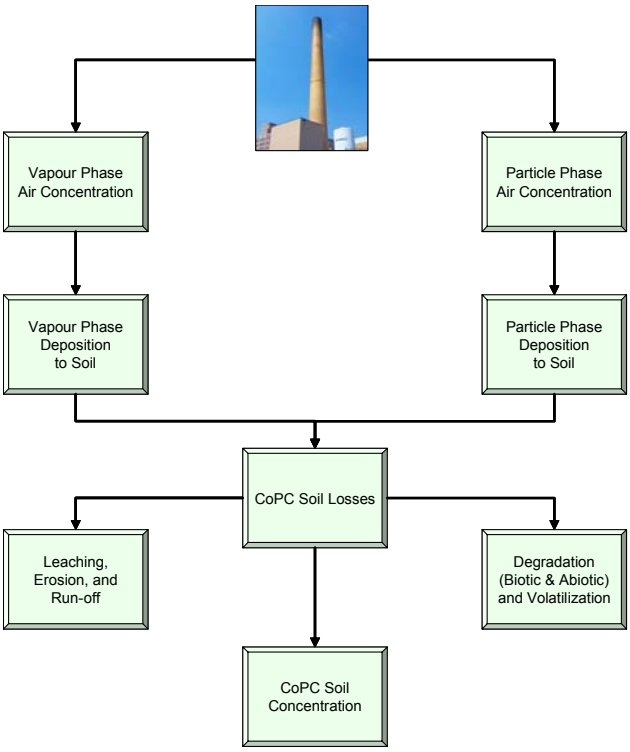
Final EPC values for all media are presented in **Appendix E** by Operating Scenario.

4.2.1 Ambient Air

Concentrations of CoPC in ambient air are primarily a function of the emission rate. The CoPC concentration in ambient air is directly inhaled and absorbed through the skin of a receptor. These concentrations are obtained directly from the air dispersion modelling results.

4.2.2 Soil

The first step in determining CoPC uptake is to estimate CoPC concentrations in soil, based on results from the dispersion and deposition modelling. The CoPC soil concentrations are used along with the air concentrations to calculate CoPC intakes resulting from all other exposure pathways, as each pathway is influenced by the initial concentration of CoPC in soil and air. Receptors are directly exposed to soil through inhalation of soil-derived dust, dermal contact with soil and soil-derived dust, and incidental ingestion.



In order to provide a layer of safety in the risk assessment, soil concentrations were based on maximum deposition rates and air concentrations within the modelled area.

For the purposes of this assessment, there are two main classes of chemicals, carcinogenic and non-carcinogenic (Section 5.4). Each class of chemicals is treated differently in the calculation of CoPC soil concentrations. For non-carcinogens, where risks are calculated for specific exposure durations, the soil concentration is calculated as the single highest annual soil concentration throughout the operating lifetime of the facility. Typically, this occurs at the end of the operating period. For those chemicals that manipulate the functions of genetic material and cell production in a non-threshold mechanism of action (i.e., carcinogens), risks are averaged over a lifetime of exposure. Therefore, the soil concentrations are also averaged over the operating lifetime of the facility (i.e., 35 years).

CoPC concentrations in soil were calculated by summing the vapour and particle phase deposition to the soil. Wet and dry deposition of particles was considered, with dry deposition of vapours calculated from the vapour air concentration and the dry deposition velocity. The calculation of soil concentration also incorporated a term (ks) that accounts for loss of CoPC by several mechanisms, including leaching, erosion, runoff, degradation (biotic and abiotic), and volatilization. For inorganic CoPC (metals), it is assumed that soil losses due to abiotic degradation and volatilization are zero as these elements are not biodegradable, nor volatile.

The US EPA model allows for variation of the soil mixing zone through which the contaminants would be deposited and then be distributed. The model provides soil depth ranges from 2 cm to 20 cm (till depth). Given that this facility will be operated for 35 years there will be downward migration of contaminants, at least in the top 10 cm of soil, over this period of time. In addition, the majority of the uptake of chemicals and subsequent exposure is from environmental receptors that would be exposed to tilled soil, such as garden produce and crops. Therefore, a 10 cm soil deposition zone was selected for use in the risk assessment.

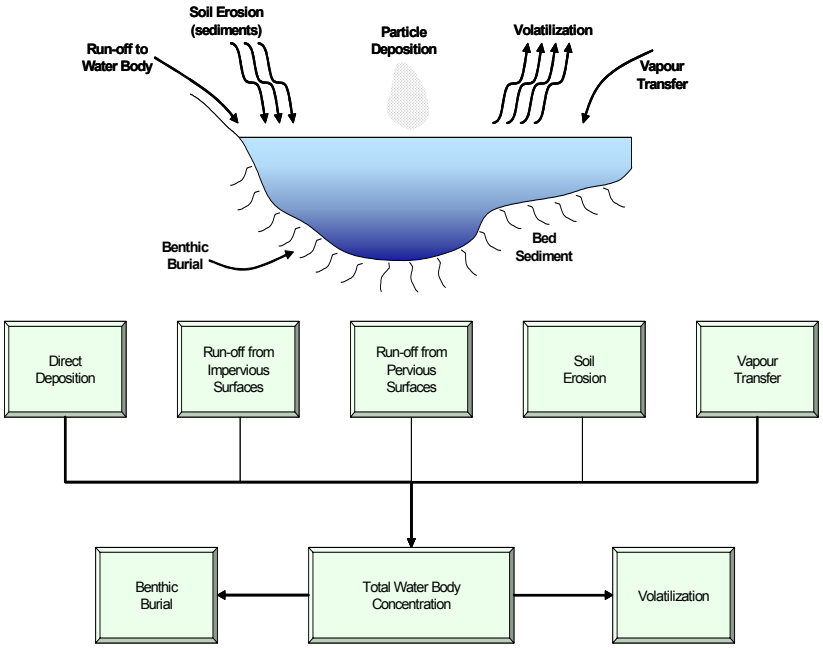
Table 4-1 presents the Ontario “background” or typical range values in soil known as the OTR_{98}. In addition, modelled soil loadings over 35 years of the life of the facility are provided, along with the percent change from the background concentrations. It should be noted that these are not site specific background concentrations. During the site-specific risk assessment, data should be collected with respect to the local background soil concentrations so that quantitative modeling of cumulative risk of loading of air contaminants to soil can be conducted.

Table 4-1 Background and Modelled Soil Concentrations after 35 years of Deposition

Analyte	Background OTR98	Scenario 1 (3 units)		Scenario 2 (2 units)		Scenario 3 (1 units)	
	Surface Soil Concentration (mg/Kg)	Surface Soil Concentration (mg/Kg)	% Loading	Surface Soil Concentration (mg/Kg)	% Loading	Surface Soil Concentration (mg/Kg)	% Loading
BTEX							
Benzene	4.00E-05	2.36E-07	0.6	1.97E-07	0.5	1.41E-07	0.4
PAHs							
Anthracene	6.00E-03	2.58E-06	0.04	2.09E-06	0.03	1.44E-06	0.02
Benzo(a)pyrene (TEQ)	1.19E-01	1.95E-06	0.002	1.57E-06	0.001	9.81E-07	0.001
Naphthalene	6.00E-03	9.89E-07	0.02	8.02E-07	0.01	5.52E-07	0.01
Phenanthrene	9.20E-02	3.43E-06	0.00	2.78E-06	0.003	1.91E-06	0.00
PCBs							
Aroclor 1254 (Total PCBs)	1.50E-02	6.35E-05	0.4	5.15E-05	0.3	3.60E-05	0.2
Dioxins and Furans							
2,3,7,8-TCDD Equivalent	4.80E-06	4.76E-08	1.0	3.86E-08	0.8	2.66E-08	0.6
2,3,7,8-TCDD Equivalent - Peel	4.80E-06	6.01E-09	0.1	4.87E-09	0.1	3.36E-09	0.1
VOCs							
Chloroform	2.20E-03	2.11E-09	0.0001	1.74E-09	0.0001	1.22E-09	0.0001
Dichloromethane	4.50E-04	3.78E-07	0.08	3.11E-07	0.07	2.19E-07	0.05
Di(2-ethylhexyl)phthalate							
Formaldehyde		9.57E-05		7.88E-05		5.54E-05	
Tetrachloroethylene	1.10E-03	2.70E-08	0.002	2.22E-08	0.002	1.56E-08	0.001
Vinyl Chloride	3.00E-05	1.53E-10	0.001	1.26E-10	0.0004	8.88E-11	0.0003
Chlorinated Monocyclic Aromatics							
1,2-Dichlorobenzene	3.00E-06	2.67E-08	0.89	2.17E-08	0.72	1.52E-08	0.51
1,2,4-Trichlorobenzene		7.56E-08		6.14E-08		4.29E-08	
1,2,4,5-Tetrachlorobenzene		1.00E-06		8.15E-07		5.70E-07	
Pentachlorobenzene		2.61E-06		2.12E-06		1.48E-06	
Hexachlorobenzene		1.32E-06		1.08E-06		7.52E-07	
2,4-Dichlorophenol	1.40E-02	5.34E-07	0.004	4.34E-07	0.003	3.03E-07	0.002
2,4,6-Trichlorophenol	6.00E-03	3.71E-07	0.01	3.01E-07	0.01	2.11E-07	0.004
2,3,4,6-Tetrachlorophenol	1.40E-02	1.04E-06	0.01	8.44E-07	0.01	5.90E-07	0.004
Pentachlorophenol	1.40E-02	9.07E-07	0.01	7.37E-07	0.01	5.15E-07	0.004
Inorganics							
Antimony	4.30E-01	7.51E-05	0.02	5.44E-05	0.01	1.32E-06	0.0003
Arsenic	1.10E+01	1.12E-05	0.0001	8.36E-06	0.0001	1.71E-06	0.00002
Barium	1.60E+02	1.86E-04	0.0001	1.40E-04	0.0001	3.67E-05	0.00002
Beryllium	1.10E+00	4.72E-06	0.0004	3.54E-06	0.0003	9.30E-07	0.0001
Boron	3.00E+01	3.87E-04	0.001	2.81E-04	0.001	6.82E-06	0.00002
Cadmium	7.10E-01	4.14E-04	0.06	3.11E-04	0.04	8.15E-05	0.0115
Chromium (Total)	5.80E+01	9.39E-05	0.0002	7.05E-05	0.0001	1.85E-05	0.00003
Cobalt	1.60E+01	3.23E-06	0.00002	2.34E-06	0.00001	5.70E-08	0.0000004
Lead	4.50E+01	9.73E-03	0.02	7.26E-03	0.02	1.64E-03	0.0036
Mercury - Elemental							
Mercury - Inorganic	1.30E-01	1.56E-04	0.12	1.21E-04	0.09	4.89E-05	0.04
Methyl Mercury		3.16E-06		2.45E-06		9.93E-07	
Nickel	3.80E+01	9.57E-05	0.0003	7.18E-05	0.0002	1.89E-05	0.00005
Phosphorous	9.00E-01	6.78E-05	0.01	4.91E-05	0.01	1.19E-06	0.0001
Silver	2.70E-01	4.83E-06	0.002	3.62E-06	0.001	9.51E-07	0.0004
Vanadium	7.70E+01	3.30E-05	0.00004	2.39E-05	0.00003	5.82E-07	0.000001
Zinc	1.20E+02	2.31E-03	0.002	1.73E-03	0.001	4.23E-04	0.0004

The results of loading of CoPCs to soil over the 35 year life of the EFW facility in all cases resulted in soil loadings of less than 1% of natural background concentrations.

4.2.3 Surface Water



Concentrations of CoPCs in drinking water are a function of many factors influencing the watershed, including direct deposition, runoff from pervious and impervious surfaces, soil erosion, direct diffusion into surface water, and internal transformation of compounds chemically or biologically.

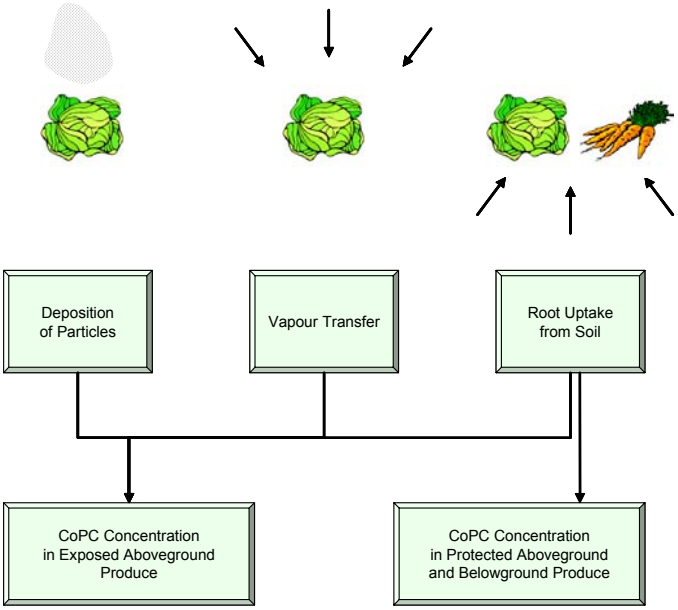
The total concentration of each CoPC is partitioned between the sediment and the water column. For calculation of CoPC concentrations in fish and in drinking water, US EPA (2005) recommended the use of the dissolved water concentration, minus any suspended particulates. For drinking water ingestion, this assumes that the water is filtered prior to consumption. As this is often not the case in many rural areas, we have used the total water column CoPC concentration, assuming no filtration of particulate phase CoPC. Receptors may be exposed to CoPC in water by using a nearby surface waterbody as a drinking water source.

To complete the modelling of the effects of airborne deposition to a watershed and water bodies, information on climate factors (e.g,, precipitation) and hydrology (e.g., river flow rates) were obtained from Environment Canada (Climate Normals – Oshawa Station). A hypothetical one-square-kilometre lake was assumed as the potential surface water source. The volumetric flow rate through the lake was calculated based on the size of the watershed and the average annual surface runoff in the region.

This is a very conservative exposure pathway that is being included in the generic risk assessment. Many residents of the Durham and York Regions are on supplied, treated municipal water sources that are not located in close proximity to any of the short listed sites. Shallow wells are also a source of drinking water in the Regions, however, this surface water source would be estimated to have a much higher contaminant load than if a groundwater impacted situation was considered.

4.2.4 Backyard Garden

Indirect exposure resulting from ingestion of produce depends on the total concentration of CoPC in the leafy, fruit, and tuber portions of the plant. Because differences exist in contamination mechanisms, consideration of indirect produce exposure was separated into two broad categories – aboveground and below ground produce (e.g., potatoes, carrots, beets). In addition, aboveground produce was further subdivided into exposed (lettuce, tomatoes, sprouts, beans) and protected (peas, corn, squash) categories.

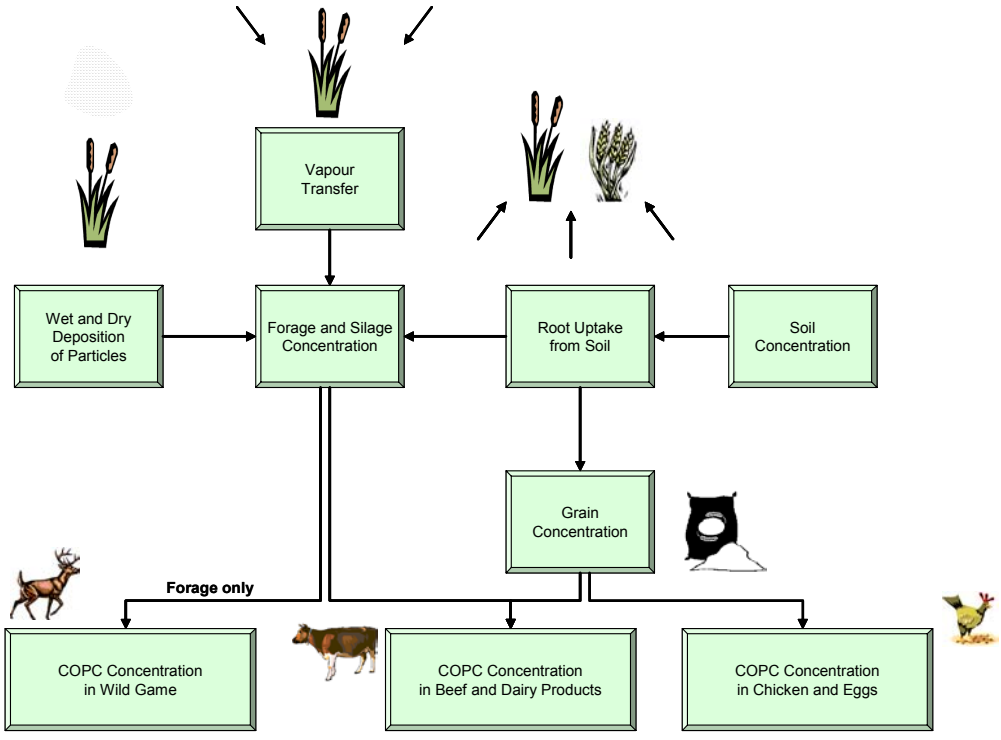


Aboveground exposed produce was assumed to become contaminated through three possible mechanisms: direct deposition of particles, vapour transfer, and root uptake; while aboveground protected produce and belowground produce were assumed to become contaminated through root uptake alone.

It is assumed in the risk assessment that backyard garden produce grown in the summer is preserved or frozen and is consumed year-round, though it is also recognized that, normally, an individual's entire intake of produce does come from their own garden.

4.2.5 Agriculture and Country Foods

For the purposes of this assessment, agriculture includes produce, beef, pork, poultry, eggs, and milk, while country foods include wild game and fish.



In the agricultural food chain, cattle, pigs, and chicken are assumed to be exposed to CoPC through impacted feed products (forage, silage, and grain) and through incidental ingestion of impacted soil. Cattle are assumed to spend six months per year in pasture and the other six months in the barn. During the summer months, the cattle consume forage that is assumed to

be impacted by wet and dry particle deposition, vapour transfer, and root uptake of CoPC. During the winter months, the cattle consume silage and grain that has been impacted by CoPC uptake prior to harvest. Chickens are assumed to be fed only grain.

Wild game are assumed to forage and consume incidental CoPC-affected soil in the vicinity of the facility. The wild game is assumed to spend its entire lifetime in the vicinity of the facility and not range into other regions that would be subject to less deposition, resulting in a conservative overestimation of wild game tissue concentrations for those animals with a large home range.

It is conservatively assumed that all CoPC are 100% bioavailable to cattle, pigs, and chicken. In addition, it is assumed that neither cattle nor chickens are able to metabolise any of their CoPC intake. Both of these assumptions will tend to overestimate the uptake of CoPC through the agricultural food chain, as there is no mechanism to offset the amount of bioaccumulation suggested by the biotransfer factors.

For modelling purposes, primary literature uptake factors for predicting animal tissue concentrations are available for beef. In accordance with US EPA (2005) guidance, to predict the uptake of CoPC into wild game and pork, the beef uptake factor is adjusted based on the relative lipid content of the game animal or pig. Whole body lipid contents for representative game species were obtained from Stephenson (2003), Wirsing et al. (2002), Stephenson et al. (1999), and Knott et al. (2005).

Fish in the one square kilometer lake are assumed to be exposed to CoPCs by a variety of mechanisms, including direct deposition to the water body, surface runoff, and soil erosion into the water body. It is assumed that all locally caught fish consumed by area residents comes from the lake within the deposition area being assessed. This is again a theoretical lake in the generic risk assessment and is likely a very conservative watershed for chemical loading that was assessed.

A primary factor to be considered when addressing the consumption of aquatic life as an exposure pathway is the propensity of CoPC to bioaccumulate or biomagnify (i.e., when chemicals accumulate in body tissue and biomagnify in the food chain). These factors can elevate concentrations of substances in aquatic life, resulting in exposures to top consumers, such as residents who fish from local water bodies. Bioconcentration/bioaccumulation factors (BCF/ BAFfish) for fish represent the ratio of the CoPC concentration in fish to the CoPC concentration in the water column/body where the fish is exposed.

4.2.6 Breast Milk

The potential for CoPC to accumulate in breast milk, and be transferred to infants, was evaluated as part of this HHRA. This pathway has been evaluated for all receptor types, with the exception of the commercial receptor, and for all organic CoPCs (i.e., excluding metals). Unlike the organics, metals do not bind to fat and so do not usually accumulate to higher concentrations in breast milk than in blood (Golding, 1997). As a result, infants are likely to be exposed to higher levels before birth and as a toddler than during breast-feeding.

Of possible particular concern are lead and mercury; however, much of the lead in breast milk does not come from the mothers' exposures during lactation. Instead, it comes from lead stored in the bones. Women can significantly reduce their child's exposure to lead by getting adequate calcium during pregnancy and lactation. Breast milk levels of mercury are usually lower than

levels of lead. Mercury does not accumulate in breast milk; in fact, the levels in the mother's blood are generally about three times higher than the levels in milk (Oskarsson, 1995). Therefore, prenatal exposure is generally more important than lactational exposure to mercury. Two major forms of mercury can enter breast milk. The most hazardous, methylmercury, does not enter breast milk at high rates because it is attached to red blood cells, but what little does get into breast milk is easily absorbed in the intestine of a nursing infant. The second form, inorganic mercury, enters breast milk easily but is not well absorbed in the infant's gastrointestinal system (Oskarsson, 1996).

The infant is assumed to be exclusively breast fed for six months, which is consistent with Health Canada's (2004a) definition of an infant and both Health Canada and the World Health Organization's current recommendations (Health Canada, 2004c, WHO, 2001). Although it is recognized that breast feeding practices vary widely, Ryan (1997) has shown that approximately 20% of US mothers are still breastfeeding their infants (in any amount) at six months of age. In addition, Health Canada (2004c) recommends that infants should be introduced to nutrient-rich, solid foods with particular attention to iron at six months of age.

The primary factor in the transfer to the infant is the mother's total uptake of the CoPC over the 35-year operating life of the facility. For dioxins and furans and PCBs, specifically, the concentration in breast milk is also a function of the maternal fat content, and the percentage of dioxin (or dioxin-like PCBs) that is stored in fat. During the first six months of breast feeding greater than 60% of a mothers fat-sequesters organic load is likely transferred to the infant. So it is during this lifestage that breast fed would be at the greatest risk of exposure to these contaminatns. Equations were adopted from McKone (1993) and US EPA (2005).

5.0 HUMAN HEALTH RISK ASSESSMENT

5.1 Problem Formulation

Problem formulation is the first step in the risk assessment process. Information is gathered on the proposed operation and its potential interactions with the environment to provide focus for the subsequent phases of the risk assessment. Key factors that are evaluated include:

- potential emissions from the proposed facility;
- screening of CoPC to focus on those chemicals that are most likely to contribute the greatest risk;
- screening and assessment of potential exposure pathways;
- characterization of potential receptors who may be exposed to emissions; and
- development of a conceptual model that describes the potential interactions between the operation and the surrounding communities and environment.

The primary objective of the human health risk assessment (HHRA) was to evaluate the likelihood of adverse health effects due to the proposed EFW facility. The focus of the assessment was the effects of emissions to air and water in the region, including potential traditional use of the land by First Nations and Métis peoples. Each step in the problem formulation leading to the conceptual model is described in the following sections.

5.1.1 People Evaluated in the Risk Assessment

Given that this is a generic risk assessment, receptor locations were identified at which to quantitatively predict the potential effects of project activities. In general, the overall annual average MPOI ground-level air concentrations (highest concentration) and corresponding deposition rates predicted by the dispersion modelling were used in determining receptor locations.

The receptor locations at which air concentrations and deposition rates were specifically identified are shown in Table 5-1, using a Cartesian grid system with (x,y) coordinates, where the location of the stack was set as the origin (0,0) of the coordinate system. Distances from the stack of the MPOI are shown in Table 3-1.

Based on the above, four receptor populations were identified for the multi-media risk assessment, as presented in Table 5-1. Each of the receptors is assumed to live or work at the MPOI.

Table 5-1 Receptor Locations

Receptor	Assumptions
1. Durham-York Resident	An individual living year-round in Durham-York who grows a vegetable garden and obtains some of their fish from local lake.
2. Durham-York Subsistence Farmer	An individual living year-round in Durham-York who is assumed to fish, to consume agricultural products (i.e., beef, milk, pork, chicken, and eggs), and grow a vegetable garden on their own property. The Subsistence Farmer is assumed to spend more time outdoors than the local resident and harvest 100% of their food (e.g., meat, fish, poultry, fruit, vegetables) from the local area.
3. Durham-York First Nations and Métis	A First Nations and Métis person who lives in Durham-York, grows a vegetable garden, and hunts and fishes. The First Nations and Métis people are assumed to spend more time on the land, more time outdoors, and harvest 100% of their country foods (e.g., game, fish) from the local area. In addition, their consumption rates of fish are considerably greater than non-native receptors.
4. Durham-York Commercial Worker and Commercial Day-Care	The Durham-York worker is assumed to spend ten hours per day at a commercial building (e.g., place of work) in Durham-York. The worker is different from the Resident in that it is assumed they do not live in the vicinity of the EFW facility. In addition, toddlers were assumed to be at a commercial day-care ten hours per day.

It is important that conservative assumptions are made about the potential human receptors. In accordance with Health Canada guidance, carcinogenic and non-carcinogenic CoPC are evaluated differently, as follows:

- Non-carcinogenic CoPC are assumed to act via a threshold mechanism and exposures are assessed within specific life stages. Generally, the toddler life stage, defined as 6 months to 4 years, is considered the most sensitive life stage based on receptor characteristics (e.g., lower body weights) combined with behavioural patterns (e.g., higher soil ingestion rates).
- Carcinogenic CoPC are assumed to act via a non-threshold mechanism and exposures are assessed over a lifetime. Health Canada recommends that a full lifetime of exposure be adopted as the most sensitive approach, based on combining exposures from five individual life stages:
    - Infant: 0 to 6 months
    - Toddler: 6 months – 4 years
    - Child: 5 years – 11 years
    - Teen: 12 years – 19 years
    - Adults: 20 years – 75 years

This combination of multiple life stages is referred to as a "composite" receptor.

Therefore the potential human receptors, or people who may be most affected by the potential emissions are listed in Table 5-2.

Table 5-2 Human Receptors

Receptor	Non-Carcinogenic CoPC	Carcinogenic CoPC
Durham-York Resident	Infant, Toddler	Composite
Durham-York Subsistence Farmer	Infant, Toddler	Composite
Durham-York First Nations and Métis	Infant, Toddler	Composite
Durham-York Commercial Worker and Commercial Day-Care	Toddler	Adult

These assumptions regarding receptors are the most protective approaches for the intended land uses. Important characteristics of the receptors (including body weight, soil ingestion rate, etc.) considered in the analysis are presented in **Appendix A**.

5.1.2 Chemicals of Potential Concern (CoPC)

The most likely route for CoPC release to the environment from the EFW facility is via airborne dispersion of particulates and vapours. Based on a review of similar operations, a comprehensive list of CoPC was determined that conservatively reflects the greatest potential health effects of the emissions, as discussed in Section 2. The multi-media risk assessment was completed using the CoPC in Table 5-3. The combustion gases are only considered for inhalation pathway.

Table 5-3 List of Chemicals of Potential Concern Evaluated in the Human Health Risk Assessment

Metals	Chlorinated Monocyclic Aromatics	Chlorinated Polycyclic Aromatics	Polycyclic Aromatic Hydrocarbons	Volatile Organic Compounds
Antimony Arsenic✓ Barium Beryllium Boron Cadmium✓+ Chromium ✓ Cobalt Lead✓+ Mercury✓+ Nickel Phosphorus Silver Vanadium Zinc	1,2-Dichlorobenzene 1,2,4-Trichlorobenzene 1,2,4,5-Tetrachlorobenzene Pentachlorobenzene Hexachlorobenzene 2,4-Dichlorophenol 2,4,6-Trichlorophenol 2,3,4,6-Tetrachlorophenol Pentachlorophenol	PCBs 2,3,7,8-TCDD TEQ✓+	*Benzo(a)pyrene group* Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(g,h,i)perylene Benzo(k)fluoranthene Chrysene Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Anthracene Napthalene Phenanthrene	Benzene✓ Chloroform Dichloromethane Formaldehyde Tetrachloroethylene Vinyl chloride✓

Notes:

Chemical list derived from Cantox Report for Human Health Risk Assessment for the Proposed Expansion of the KMS Peel, Inc. Brampton, Energy-From-Waste Facility (2000)

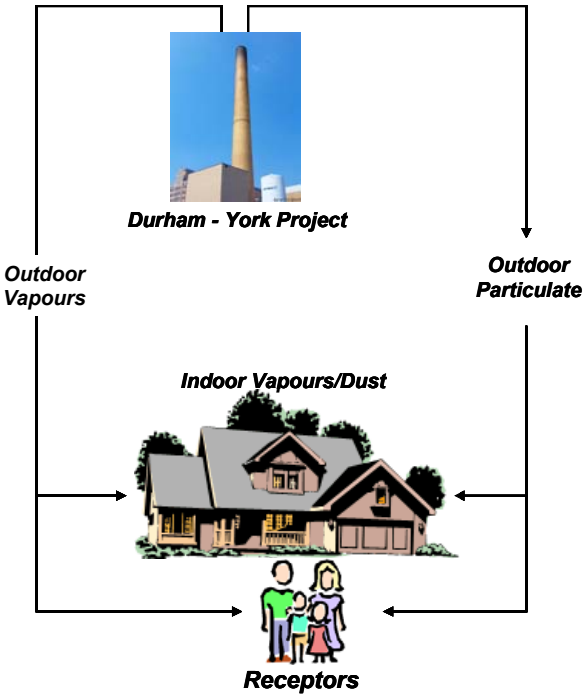
✓ chemicals also reviewed by MOE in Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration (1999)

\+ chemical also included in GUIDELINE A-7 Combustion and Air Pollution Requirements for New Municipal Waste Incinerators (MOE 2004)

5.1.3 Exposure Pathways

There are many mechanisms by which human receptors could be affected by project emissions. The purpose of this section is to identify the exposure pathways that will be evaluated in the risk assessment to estimate the type and magnitude of human exposure to CoPC emissions from the facility.

5.1.3.1 Vapours and Particulate Emissions



The only direct exposure pathway to the project emissions is via vapour or particulate inhalation in ambient air.

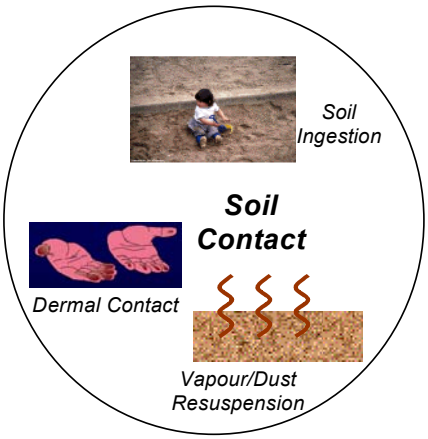
CoPC concentrations in air are calculated by summing the vapour phase and particle phase (particle and particle-bound) air concentrations of CoPC. Air concentrations used in the evaluation of chronic health risks were calculated using annual average values; whereas air concentrations used in the evaluation of acute health risks were calculated using 1-hour or 24-hour values.

As indicated, receptors can be directly exposed to vapours and particulates both outside in the ambient air and within their homes.

All CoPC are assessed for this exposure pathway except for methyl mercury. Methyl mercury is assumed not to exist in the vapour phase. This is the only exposure pathway for which elemental mercury (Hg^0) is assessed.

Inhalation rates for all human receptors were adopted from Health Canada (2004a).

5.1.3.2 Soil Contact



Surface soil surrounding the facility will be subject to particulate deposition and may accumulate levels of various CoPC over the operating life of the facility (35 years).

CoPC concentrations in soil are calculated by summing the vapour phase and particle phase deposition of CoPC to the soil. Both wet and dry deposition of particles and vapours are considered. In addition, CoPC loss mechanisms such as leaching, erosion, run-off, degradation, and volatilization are incorporated.

In accordance with US EPA guidance, the accumulation of CoPC in soil is averaged over the operating lifetime of the facility for carcinogenic chemicals. Potential risks from exposure to carcinogenic chemicals are averaged over a lifetime. For non-carcinogenic chemicals, where potential risks are evaluated within specific exposure durations

(e.g., toddler), the highest annual average soil concentration occurring during the operating life of the facility is used.

As indicated, direct exposure to contaminants in soil can be via three potential exposure mechanisms. Young children can incidentally ingest soil. Soil can become adhered to an individual's hands (e.g., during gardening) and chemicals can be absorbed through the skin. Particulate matter or dust and vapours can be re-suspended from the surface soil and inhaled by a receptor.

All CoPC are assessed for this exposure pathway except for elemental mercury. Elemental mercury is assumed to only exist in the vapour phase and is not assessed for soil contact.

Exposed skin surface areas were adopted from Health Canada (2004a) and Richardson (1997). Soil ingestion rates for all receptors were taken from Health Canada (2004a). Dust ingestion rates were calculated based on the methodology presented in *Appendix A of Appendix B.5 of the Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for Use at Contaminated Sites in Ontario* (MOE, 1996).

5.1.3.3 Drinking Water

US EPA (2005) does not include groundwater as an exposure pathway for assessment of airborne emissions as the shallow surface soil deposition is unlikely to affect deeper groundwater resources. It was conservatively assumed that all receptors obtain their drinking water from a surface water body in the vicinity of the facility. This water body would be subject to vapour and particulate emissions that deposit onto the water body in the facility area and CoPC may become dissolved in the water column.

Again this estimation of potential exposure would overestimate any potential risk to residents in the Regions who either consume treated municipal water or well water.

All CoPC are assessed for this exposure pathway except for elemental mercury. Elemental mercury is assumed to only exist in the vapour phase and is not assessed for drinking water exposure.

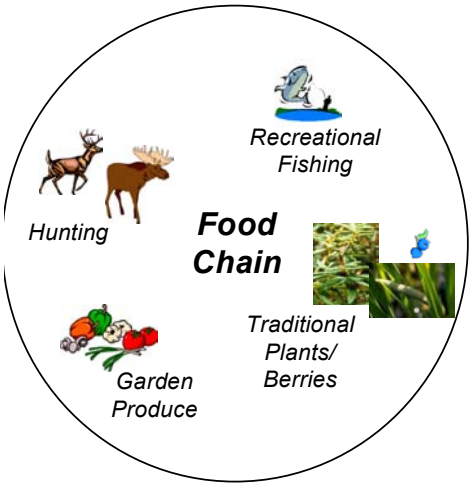
Drinking water ingestion rates for all receptors were taken from Health Canada (2004a).

5.1.3.4 Food Chain Uptakes

Particulate deposition and vapour transfer of CoPC may occur in the surrounding region and result in CoPC concentration or accumulation in the food chain.

Garden Produce

We have assumed that all local residents (including the Subsistence Farmer and First Nations and Métis) will grow vegetable gardens. Indirect exposure resulting from ingestion of produce depends on the total concentration of CoPC in the leafy, fruit, and tuber portions of the plant. Because of general differences in uptake mechanisms, garden produce is divided into four broad categories:



- Exposed aboveground vegetables (e.g., lettuce, sprouts);
- Protected aboveground vegetables (e.g., squash, beans, peas);
- Below ground vegetables (e.g., potatoes, carrots); and
- Exposed aboveground fruits (e.g., strawberries, blueberries).

All CoPC are assessed for this exposure pathway except for elemental mercury. Elemental mercury is assumed to only exist in the vapour phase and is not assessed for uptake through the food chain.

Garden produce ingestion rates for all four categories were taken from the Exposure Factors Handbook (EFH) (US EPA, 1997). When using intakes from the EFH, the mean values for sexes combined were used for each age group. Where age groupings were different than those assumed in this risk assessment, the highest reported intake for any of the age groups reported in the EFH falling within our assumed age categories was used.

The portion of a person's produce that comes from their garden was based on the EFH (US EPA, 1997). The US EPA estimates of home-grown produce consumption as a portion of total produce consumption are applicable to the United States and likely provide an overestimation of home-grown produce consumption at northern latitudes that have much shorter growing seasons.

At a site-specific level, it may be possible to assess the risks to the First Nations and Métis receptors associated with consuming traditional plants; however, for the purposes of this generic risk assessment, and in the absence of site-specific data on the use of traditional plants in the area, we have assumed that the First Nations and Métis population grow backyard gardens. It is likely that the ingestion rates associated with backyard produce consumption would overestimate the ingestion rates associated with traditional plants and berries.

Agriculture

We have assumed that the subsistence farmer receptor obtains all of their beef, pork, poultry, eggs, and milk from their farm (and does not supplement).

In the agricultural food chain, cattle, pigs, and chicken are assumed to be exposed to CoPC through impacted feed products (forage, silage, and grain) and through incidental ingestion of impacted soil. All CoPC are assessed through this pathway except for elemental mercury.

Agricultural ingestion rates for the subsistence farmer receptors were adopted from the EFH (US EPA, 1997). Mean per capita rates were used.

Hunting and Fishing

We have assumed that local fishers and First Nations and Métis people use the land in the area of the facility. It is possible that First Nations and Métis people, in particular, consume a large amount of wild game and fish from the local area. It is also assumed that the Durham-York Resident and Subsistence Farmer obtain all of their fish locally.

Uptake into wild game is assumed to occur through consumption of forage and incidental ingestion of soil. Bioconcentration or bioaccumulation of CoPC into fish is generally related to the CoPC loading to the water body. This can occur by a variety of mechanisms including direct deposition, vapour transfer, surface run-off, and erosion.

All CoPC are assessed through this pathway except for elemental mercury. It should also be noted that all mercury in fish is assumed to be converted to the methylated form (MHg) in the fish tissue.

Fish ingestion rates for the local residents and subsistence farmers were taken from the EFH (US EPA, 1997). Although Richardson (1997) (as cited in Health Canada, 2004a) provides fish consumption rates for non-native populations, these rates include a wide variety of fish products, including marine fish (cod, haddock), canned salmon, tuna, and sardines, freshwater fish, and shrimp (fresh, frozen, and canned). As the intention of this assessment is to evaluate the risks arising from the consumption of fish caught within the modelled watershed, these values were not considered appropriate. Mean fish intake rates for individuals who eat fish and reside in households with recreation fish consumption from the EFH were used. Fish ingestion rates for the First Nations and Métis receptors were taken from Health Canada (2004a), but it should be noted that these are not specific to Ontario First Nations.

5.1.3.5 Breast Milk

We have assumed that the infant (as defined by Health Canada (2004a)) is exclusively breast fed (meaning their intake of all other foods and water is set to zero) from age zero to six months.

Maternal body burden is the most important factor in the determination of transfer to the infant. Researchers have estimated that maternal body burden (specifically PCB/dioxin) decreases as much as 20% to 70% during 6 months of exclusive breastfeeding (Kreuzer et. al (1997) and Rogan et. al (1986)). Breast milk consumption rates for the infant were taken from Richardson (1997) for an exclusively breast fed infant.

5.1.4 Conceptual Model

Table 5-4 summarizes the exposure scenarios that are included in the site conceptual model and Figure 5-1 illustrates the model. As indicated, receptors are assumed to be exposed to the CoPC via five main exposure scenarios:

- vapour inhalation;
- particulate inhalation;
- soil contact;
- drinking water; and
- food chain uptakes.

Table 5-4 Summary of Exposure Pathways

	Receptors			
Exposure Pathways	**Durham – York Resident [1]**	**Durham – York Subsistence Farmer [2]**	**Durham – York First Nations/ Métis [3]**	**Durham – York Worker[4]**
Direct inhalation	✓	✓	✓	✓
Soil contact	✓	✓	✓	✓
Drinking water	✓	✓	✓	✓
Garden produce	✓	✓	✓	
Fish	✓	✓	✓	
Wild game			✓	
Agriculture (meat, poultry)		✓		

5) Resident includes an adult, toddler, and nursing infant.

6) Subsistence Farmer includes an adult, toddler, and nursing infant.

7) First Nations and Métis includes an adult, toddler, and nursing infant.

8) Commercial includes an adult worker and a toddler at a daycare facility

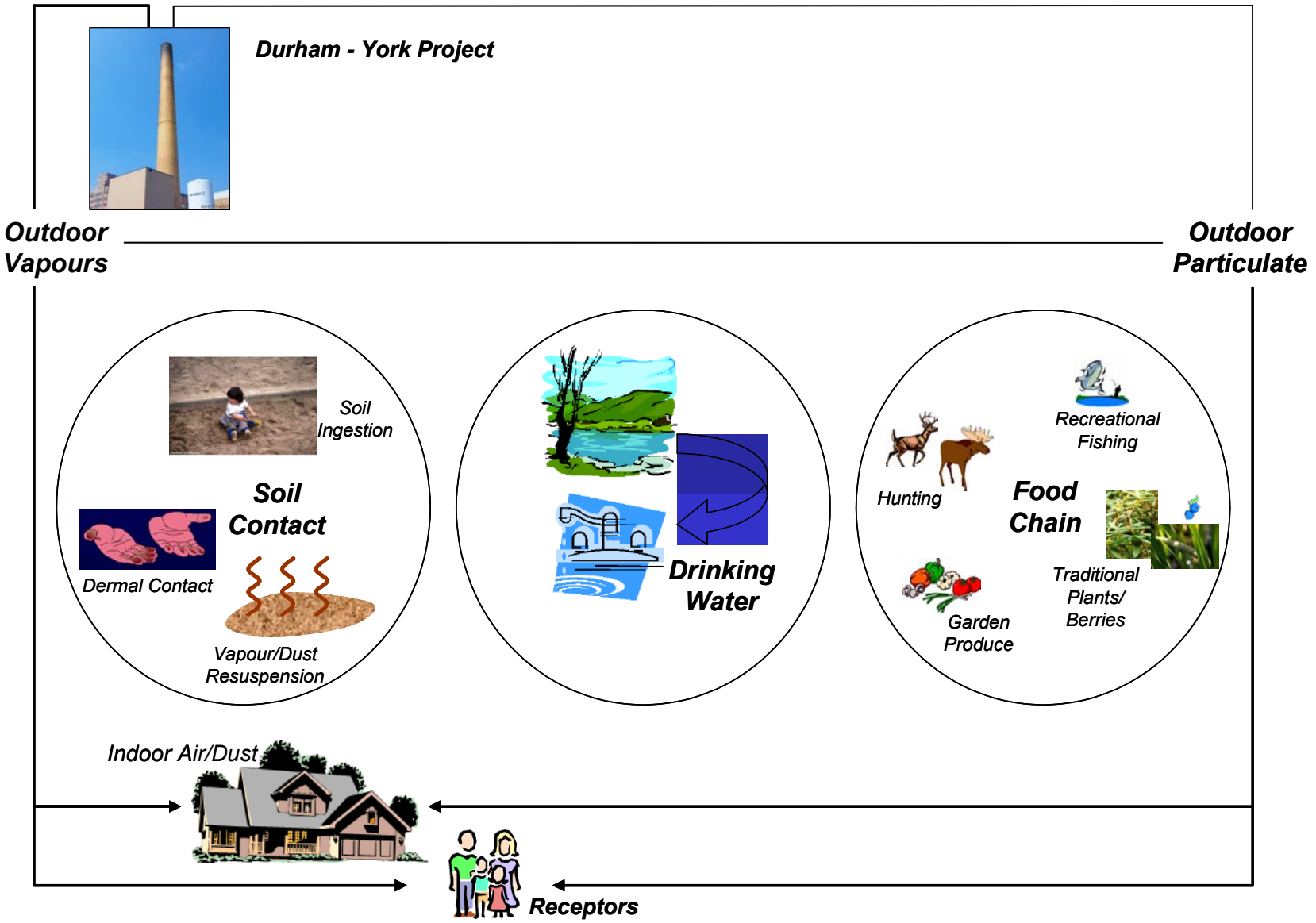


Figure 5-1 Site Human Health Conceptual Model

5.2 Toxicity Assessment

In this stage of the risk assessment, literature on the toxic potential of each CoPC was reviewed and toxicity reference values (TRVs) were selected for use in the risk characterization.

Health effects caused by the action of a chemical on the human body can vary depending on dose, length of exposure, and route of exposure. One of the usual methods of evaluating severity of health effects of a chemical is to expose test subjects to different doses of the chemical while keeping length of exposure and route of exposure constant. After exposure, the test subjects are compared to controls, and any adverse health effects at each dose level in the test population are noted. The results of such an experiment constitute a dose-response relationship.

Two distinct patterns of dose-response relationships have been observed: threshold behaviour and non-threshold behaviour. In the first pattern, threshold behaviour, a specific dose level can be identified, at which no adverse effects are observed. This dose, known as a No Observed Adverse Effects Level (NOAEL), adjusted by uncertainty factors, serves as the basis for many TRVs. Alternatively, if a NOAEL cannot be identified, a lowest observed adverse effects level (LOAEL), being the minimum dose, at which (usually minor) adverse effects are observed, may be used to derive a TRV instead; the application of an extra uncertainty factor to a LOAEL is warranted when deriving a TRV, since the “safe” dose level below that LOAEL may not have been identified.

Many dose-response relationships, however, do not show threshold behaviour, and no NOAEL or LOAEL can be clearly identified. Such cases arise when studying carcinogenic effects of chemicals, and for these effects, it is not practical to propose a TRV based on the NOAEL/LOAEL approach, since no dose can be designated as having zero risk. For chemicals that induce cancer, a slope factor or unit risk factor is used as a TRV; usually based on statistical measures of incidence of or mortality due to cancer in cohorts of individuals exposed to the chemicals in question.

5.2.1 Selection of TRVs

Toxicological information for both types of dose-response behaviours from the following sources was reviewed:

- Health Canada;
- United States Environmental Protection Agency (US EPA) Integrated Risk Information System (IRIS);
- International Agency for Research on Cancer (IARC);
- World Health Organization (WHO)
- Agency for Toxic Substances and Disease Registry (ATSDR);
- Canadian Council of Ministers of the Environment (CCME);
- Ontario Ministry of the Environment (MOE).

Given the number of credible government agencies that have developed TRVs it is a difficult task to decide as to the selection of the most appropriate TRV. In this study preference was first given to IRIS and Health Canada values, whereby the date of the review and validity of the studies were used for selection of TRVs. In the event that IRIS or Health Canada values were

not available, or more current TRVs had been established by reputable agencies based on sound toxicological studies they were selected for use.

A summary of the carcinogenic and non-carcinogenic TRV values used in the multi-media risk assessment is presented in Tables 5-5 and 5-6, respectively. In addition, the target organ and toxicological endpoint are provided.

It should be noted that the TRV for benzo(a)pyrene presented in Table 5-6 was used to assess the risks to human health for all carcinogenic PAHs, as a benzo(a)pyrene equivalent was calculated (further discussion on this point can be found in Section 5.2.3).

In addition to the TRVs, relative oral, inhalation, and dermal bioavailability values were also compiled, primarily from the sources listed above. Bioavailability refers to the amount of a chemical that reaches the bloodstream, once it has entered the body through a specific route. Oral bioavailability, then, refers to the fraction of a chemical that reaches the bloodstream after ingestion and its partial passage through the gastrointestinal tract. Similarly, inhalation bioavailability refers to the fraction of inhaled chemical that reaches the bloodstream after absorption in the lungs, and dermal bioavailability refers to the fraction of chemical that reaches the bloodstream after being applied to the skin. Bioavailability factors used in the risk assessment are summarized in Section 5.2.2.

Sections 5.2.3 and 5.2.4 outline the assessment of chemical mixtures in this risk assessment, and the approach to additive effects of the CoPC, respectively.

Table 5-5 Summary of Carcinogenic Toxicity Reference Values Selected for Use in the HHRA

Chemical	CAS No	Cancer Slope Factors and Unit Risk Factors						
		Oral (mg/kg-day)-1 CSFos	Oral Organ/Endpoint	Oral Reference	Inhalation URF (mg/m3)-1 URFi	Inhalation CSF (mg/kg-day)-1 CSFi	Inhalation Organ/Endpoint	Inhalation Reference
BTEX								
Benzene	71432	5.50E-02	Leukemia	IRIS 2000	3.30E-03		Leukemia	Health Canada, 2004
PAHs								
Anthracene	120127							
Benzo(a)pyrene	50328	4.60E-01	tumour - forestomach, squamous cell papillomas and	WHO 1998	8.70E+01		Lung cancer	WHO 2000
Naphthalene	91203				--			--
Phenanthrene	85018				--			--
PCBs								
Aroclor 1254 (Total PCBs)	11097691							
Dioxins and Furans								
2,3,7,8-TCDD Equivalent	1746016					--		
VOCs								
Chloroform	67663				5.00E-03		Kidney/tubular adenoma	MOE AAQC 2001
Dichloromethane	75092	7.50E-03	Liver/adenomas,carcinomas	IRIS 1995	4.70E-04		Liver/adenomas,carcinomas	IRIS 1995
Formaldehyde	50-00-0	--		--	5.26E-03		nasopharyngeal cancer (NPC) and sinonasal cancer (SNC)	CEPA, 2001
Tetrachloroethylene	127184	--		--	4.30E-04		tumours organ not identified	MOE AAQC 2005
Vinyl Chloride	75-01-4	1.40E+00	Liver/angiosarcoma, carcinoma, neoplastic nodules	IRIS 2000	5.00E-03		liver cancer	MOE AAQC 2005
Chlorinated Monocyclic Aromatics								
1,2-Dichlorobenzene	95-50-1					--		
1,2,4-Trichlorobenzene	120-82-1					--		
1,2,4,5-Tetrachlorobenzene						--		
Pentachlorobenzene	608-93-5					--		
Hexachlorobenzene	118-74-1	8.30E-01	Liver/ hepatic neoplastic nodules; Parathyroid adenomas	Health Canada, 2004	4.60E-01		Liver/ hepatocellular carcinoma	IRIS 1996
2,4,-Dichlorophenol	120-83-2					--		
2,4,6-Trichlorophenol	88-06-2	1.10E-02	Leukemia	IRIS 1994	3.10E-03		Leukemia	IRIS 1994
2,3,4,6-Tetrachlorophenol	58-90-2					--		
Pentachlorophenol	87865	1.20E-01	Liver, Adrenal gland, Vascular/ tumour	IRIS 1993		--		
Inorganics								
Antimony	7440360					--		
Arsenic	7440382	1.50E+00	Skin cancer	IRIS 1998	4.30E+00		Lung cancer	IRIS 1997
Barium	7440393	--		--	--	--		--
Beryllium	7440417				2.40E+00		Lung cancer	IRIS 1998
Boron	7440428					--		
Cadmium	7440439	--		--	9.80E+00		Lung cancer	Health Canada, 2004
Chromium Total	16065831				1.09E+01	4.76E+01	Lung cancer	Health Canada, 2004
Cobalt	7440484					--		
Lead	7439921	--		--	--	--		--
Mercury - Elemental	7439976	--		--	--	--		--
Mercury - Inorganic	7487947	--		--	--	--		--
Methyl Mercury	22967926	--		--	--	--		--
Nickel	7440020	--		--				
Phosphorous	7723140					--		-
Silver	7440224					--		
Vanadium	7440622	--		--	--	--		--
Zinc	7440666	--		--	--	--		--

References:

Health Canada, 2004 - Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs)
IRIS - US EPA Integrated Risk Information System (IRIS) Database for Risk Assessment (current as of September 1, 2006)
CEPA, 2001 - Priority Substances List Assessment Report - Formaldehyde. Envrionment Canada & Health Canada
MOE (Ontario Ministry of the Environment). 2005. Summary of Point of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria.

Table 5-6 Summary of Non-Carcinogenic Toxicity Reference Values Selected for Use in the HHRA

		Chronic Reference Doses and Reference Concentrations						
		Oral - Soil	Oral	Oral	Inhalation RfC	Inhalation RfD	Inhalation	Inhalation
		(mg/kg-day)	Study Organ/Endpoint (guideline basis)	Reference	(mg/m3)	(mg/kg-day)	Study Organ/Endpoint (guideline basis)	Reference
Chemical	CAS No	RfDcos			RfCci	RfDci		
BTEX								
Benzene	71432	4.00E-03	Blood/decr. lymphocyte count (BMDL)	IRIS, 2003 (May 07)	3.00E-02		blood; decr. lymphocyte count (BMDL)	IRIS 2003
PAHs								
Anthracene	120127	3.00E-01	NOEL	IRIS, 1993 (Nov. 2006)		3.00E-01	NOEL	Set equal to oral RfD as no inhalation RfD found.
Benzo(a)pyrene	50328				--	--		Carcinogenic by inhalation
Naphthalene	91203	2.00E-02	decr. body weight (NOAEL)	IRIS, 1998 (Nov. 2006)	3.00E-03		effects on nasal and respiratory epithelium (LOAEL)	IRIS, 1998 (Sept. 2006)
Phenanthrene	85018	4.00E-02	NA	RIVM, 2001	--	4.00E-02	NA	Set equal to oral RfD as no inhalation RfD found.
PCBs								
Aroclor 1254 (Total PCBs)	11097691	2.00E-02	NA	Health Canada, 2004		2.00E-02		Set equal to oral RfD as no inhalation RfD found.
Dioxins and Furans								
2,3,7,8-TCDD Equivalent	1746016	2.30E-09	Reproductive system effects (LOEL/NOEL)	Health Canada, 2004		2.30E-09	Reproductive system effects (LOEL/NOEL)	Set equal to oral RfD as no inhalation RfD found.
VOCs								
Chloroform	67663	1.00E-02	Liver/fatty cyst formation	IRIS, 2001 (Nov. 2006)	1.00E-03		AAQC based on carcinogenic effect	MOE AAQC 2001
Dichloromethane	75092	5.00E-02	Liver/ cellular proliferation, fatty change, both reversible (NOAEL)	Health Canada, 2004		--		Carcinogenic by inhalation
Formaldehyde	50-00-0	2.00E-01	histopathological changes G.I. tract, reduced weight (NOAEL)	IRIS, 1990 (Nov. 2006)	--	--		Carcinogenic by inhalation
Tetrachloroethylene	127184	1.40E-02	Various/ reduced wieght, altered kidney, liver (NOEL)	Health Canada, 2004	3.60E-01		Various/ reduced survival, hepatotoxicity, lung congestion, nephrotoxicity (LOAEL)	Health Canada, 2004
Vinyl Chloride	75-01-4	3.00E-03	Liver/cell polymorphism (NOAEL)	IRIS 2000	1.00E-01		Liver/cell polymorphism (NOAEL)	IRIS 2000
Chlorinated Monocyclic Aromatics								
1,2-Dichlorobenzene	95-50-1	4.30E-01	Kidney/tubular regeneration (NOEL)	Health Canada, 2004		4.30E-01		Set equal to oral RfD as no inhalation RfD found.
1,2,4-Trichlorobenzene	120-82-1	1.60E-03	Liver, Kidney/ weight increase (NOEL)	Health Canada, 2004	7.00E-03		Liver, Kidney/ weight increase, increased porphyrin excretion (NOEL)	Health Canada, 2004
1,2,4,5-Tetrachlorobenzene		2.10E-04	Liver, Kidney/ weight increase; Kidney, Thyroid/ histopathological effects (NOEL)	Health Canada, 2004		2.10E-04	Liver, Kidney/ weight increase; Kidney, Thyroid/ histopathological effects (NOEL)	Set equal to oral RfD as no inhalation RfD found.
Pentachlorobenzene	608-93-5	1.00E-03	Liver/ histological lesions (LOEL)	Health Canada, 2004		1.00E-03	Liver/ histological lesions (LOEL)	Set equal to oral RfD as no inhalation RfD found.
Hexachlorobenzene	118-74-1	5.00E-04	Liver, Heart/ weight increase; Liver, Kidney/ histopatholgical changes (NOEL)	Health Canada, 2004				Carcinogenic by inhalation
2,4,-Dichlorophenol	120-83-2	1.00E-01	NA	Health Canada, 2004		1.00E-01	NA	Set equal to oral RfD as no inhalation RfD found.
2,4,6-Trichlorophenol	88-06-2	3.00E-03	NA	RIVM 2001		--		Carcinogenic by inhalation
2,3,4,6-Tetrachlorophenol	58-90-2	1.00E-02	NA	Health Canada, 2004		1.00E-02	NA	Set equal to oral RfD as no inhalation RfD found.
Pentachlorophenol	87865	6.00E-03	NA	Health Canada, 2004		6.00E-03	NA	Set equal to oral RfD as no inhalation RfD found.
Inorganics								
Antimony	7440360	4.00E-04	Longevity, blood glucose, and cholesterol (LOAEL)	IRIS, 1991 (Nov. 2006)		4.00E-04	Longevity, blood glucose, and cholesterol (LOAEL)	Set equal to oral RfD as no inhalation RfD found.
Arsenic	7440382	3.00E-04	Hyperpigmentation, keratosis and possible vascular complications (NOAEL)	IRIS 1993	--	--		Carcinogenic by inhalation
Barium	7440393	2.00E-01	Kidney/ renal lesions	IRIS 2005	--	2.00E-01	Kidney/ renal lesions	Set equal to oral RfD as no inhalation RfD found.
Beryllium	7440417	2.00E-03	Small intestinal lesions (BMD10)	IRIS, 1998 (Nov. 2006)	2.00E-05		Beryllium sensitization and progression to chronic beryllium disease (LOAEL)	IRIS, 1998 (Nov. 2006)
Boron	7440428	2.00E-01	Developmental/decreased fetal weight (BMDL05)	IRIS 2004		2.00E-01	Developmental/decreased fetal weight (BMDL05)	Set equal to oral RfD as no inhalation RfD found.
Cadmium	7440439	8.00E-04	Kidney/renal cortex (Drinking water guideline)	Health Canada, 2004	--	--		Carcinogenic by inhalation
Chromium Total	16065831	1.00E-03	Drinking water guideline (NOAEL)	Heath Canada, 2004		--		Carcinogenic by inhalation
Cobalt	7440484	1.00E-02	Polycythemia (increased blood cells) (LOAEL)	ATSDR MRL (Dec. 2005	1.00E-04		Respiratory effects	ATSDR MRL (Dec. 2005)
Lead	7439921	1.85E-03	Neurological behaviour (LOAEL)	MOE 1994	--	1.85E-03	Neurological behaviour (LOAEL)	Set equal to oral RfD as no inhalation RfD found.
Mercury - Elemental	7439976	--		--	3.00E-04		Neurophysiological impairments (LOAEL)	IRIS, 1995 (Nov. 2006)
Mercury - Inorganic	7487947	3.00E-04	Autoimmune effects (LOAEL)	Health Canada, 2004	--	3.00E-04	Autoimmune effects (LOAEL)	Set equal to oral RfD as no inhalation RfD found.
Methyl Mercury	22967926	2.00E-04	Developmental effects	Health Canada 2005 / V	--	2.00E-04	Developmental effects	Set equal to oral RfD as no inhalation RfD found.
Nickel	7440020	2.00E-02	Decreased body and organ weights (NOAEL)	IRIS 1996 Ni salt	--	2.00E-02	Decreased body and organ weights (NOAEL)	Set equal to oral RfD as no inhalation RfD found.
Phosphorous	7723140	1.43E+04	Basd on recommended daily nutirent intake rate	Health Canada 1990		1.43E+04		Set equal to oral RfD as no inhalation RfD found.
Silver	7440224	5.00E-03	Skin/ argyria (LOAEL)	IRIS, 1996 (Nov. 2006)		5.00E-03	Skin/ argyria (LOAEL)	Set equal to oral RfD as no inhalation RfD found.
Vanadium	7440622	9.00E-03	Decrease in hair cystine content (NOAEL)	IRIS, 1996 (Nov. 2006)	--	9.00E-03	Decrease in hair cystine content (NOAEL)	Set equal to oral RfD as no inhalation RfD found.
Zinc	7440666	3.00E-01	Decreased blood enzyme levels (i.e., superoxide dismutase) (LOAEL)	IRIS, 2005 (Nov. 2006)	--	3.00E-01	Decreased blood enzyme levels (i.e., superoxide dismutase) (LOAEL)	Set equal to oral RfD as no inhalation RfD found.

References:

Health Canada, 2004 - Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs)
IRIS - US EPA Integrated Risk Information System (IRIS) Database for Risk Assessment (current as of September 1, 2006)
RIVM, 2001 - Report 711701 025. Re-evaluation of human-toxicological maximum permissible risk levels
ATSDR, 2005 - Minimal Risk Levels for Hazardous Substances (December 2005)
MOE (Ontario Ministry of the Environment). 2005. Summary of Point of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria.

5.2.2 Bioavailability

Bioavailability of a contaminant is defined as the fraction of an administered dose of contaminant that reaches the systemic circulation. The bioavailability will vary depending on the pathway of exposure (i.e., ingestion, inhalation, or dermal contact), the form of the contaminant (e.g., dissolved in water versus sorbed to fine soil), and the physiological characteristics of the receptor at the time of exposure (e.g., absorption may be higher if the receptor is malnourished). Bioaccessibility is the fraction of a contaminant in an environmental medium that is available for absorption based on laboratory extraction, but it is not necessarily absorbed.

The process of a contaminant entering the body can be described in two steps – contact with an outer boundary (exposure or intake), followed by actual entry into the bloodstream (uptake). Intake is typically defined as the process by which a contaminant crosses the outer surface of a receptor without passing an absorption barrier (such as through ingestion, inhalation, or dermal contact), while uptake is the process by which a contaminant crosses an absorption barrier (such as the lining of the gastrointestinal tract, the outer layer of skin, or the lining of the lungs) into the receptor.

As shown in Table 5-7, all oral and inhalation bioavailability factors have been conservatively set to 1.0, meaning each CoPC is 100% bioavailable via oral and inhaled routes of exposure. Further information on the sources of these relative bioavailability values can be found in the toxicological profiles in **Appendix B**.

Table 5-7 Relative Bioavailabilities Selected for Use in the Multi-media Risk Assessment

CoPC	Relative Oral Bioavailability		Relative Inhalation Bioavailability	Relative Dermal Bioavailability
	SOIL	WATER/ FOOD		
Benzene	1.0	1.0	1.0	0.08
Anthracene	1.0	1.0	1.0	0.29
Benzo(a)pyrene	1.0	1.0	1.0	0.2
Naphthalene	1.0	1.0	1.0	0.1
Phenanthrene	1.0	1.0	1.0	0.18
Aroclor 1254 (Total PCBs)	1.0	1.0	1.0	0.14
2,3,7,8-TCDD Equivalent	1.0	1.0	1.0	0.03
Chloroform	1.0	1.0	1.0	0.1
Dichloromethane	1.0	1.0	1.0	0.1
Formaldehyde	1.0	1.0	1.0	0.01
Tetrachloroethylene	1.0	1.0	1.0	0.1
Vinyl Chloride	1.0	1.0	1.0	0.16
1,2-Dichlorobenzene	1.0	1.0	1.0	0.1
1,2,4-Trichlorobenzene	1.0	1.0	1.0	0.08
1,2,4,5-Tetrachlorobenzene	1.0	1.0	1.0	0.01
Pentachlorobenzene	1.0	1.0	1.0	0.01
Hexachlorobenzene	1.0	1.0	1.0	0.13
2,4-Dichlorophenol	1.0	1.0	1.0	0.4
2,4,6-Trichlorophenol	1.0	1.0	1.0	0.26
2,3,4,6-Tetrachlorophenol	1.0	1.0	1.0	0.01
Pentachlorophenol	1.0	1.0	1.0	0.11
Antimony	1.0	1.0	1.0	0.29
Arsenic	1.0	1.0	1.0	0.03
Barium	1.0	1.0	1.0	0.1
Beryllium	1.0	1.0	1.0	0.03
Boron	1.0	1.0	1.0	0.1
Cadmium	1.0	1.0	1.0	0.14
Chromium III	1.0	1.0	1.0	0.04
Cobalt	1.0	1.0	1.0	0.1
Lead	1.0	1.0	1.0	0.006
Mercury - Elemental	1.0	1.0	1.0	0.001
Mercury - Inorganic	1.0	1.0	1.0	0.001
Methyl Mercury	1.0	1.0	1.0	0.2
Nickel	1.0	1.0	1.0	0.35
Phosphorous	1.0	1.0	1.0	0.1
Silver	1.0	1.0	1.0	0.25
Vanadium	1.0	1.0	1.0	0.1
Zinc	1.0	1.0	1.0	0.02

5.2.3 Chemical Mixtures

In order to properly assess health risks to the human receptors, certain groups of chemicals were assessed as mixtures. For the purposes of this assessment, the carcinogenic PAHs have been assessed as a mixture, the TRVs for which are based on those for benzo[a]pyrene, widely considered to be the most toxic and most carcinogenic of the PAHs. The modes of cancer induction of these PAHs are all similar; their carcinogenic potencies are; however, different. In this risk assessment, each of the carcinogenic PAHs has been assigned a toxic equivalency factor (TEF), relative to benzo(a)pyrene, to represent this differing potency. The TEFs were chosen based on the recommendations of the World Health Organization (WHO 1998), with benzo(a)pyrene being assigned a TEF of 1. These TEFs are summarized in **Appendix B**.

5.2.4 Additivity of Risks

In the assessment of toxic effects of a mixture, it is generally assumed that each component of the mixture causes the same type of adverse effects in a receptor, albeit perhaps at different potencies. It should be noted; however, that combined toxic effects may also be produced in a receptor due to exposure to interacting CoPC. Such combined effects may be additive, synergistic (greater than additive), or antagonistic (less than additive). These combined effects could arise because two or more CoPC target the same organs or tissues in the body, affect each others' bioavailabilities, or disturb biological processes in a similar manner. In order to assess these combined effects quantitatively, however, detailed studies of the interactions between CoPC are required, and little information is available in this regard.

Given the conservative nature of the generic risk assessment being undertaken, those chemicals that are reported to have the same toxicological endpoint or mode of action were summed to evaluate their additive potential risk.

5.3 Exposure Assessment

Exposure assessment involves estimating the amount of a CoPC a person may take into his or her body (i.e., a dose) through all applicable exposure pathways. For the purposes of this assessment, the dose of a CoPC depends on the concentrations in air, water, soil, agriculture, (e.g., poultry, cows, milk) fish, plants and wild game; the amount of time a person is in contact with these media; and the characteristics of the receptor (e.g., ingestion rate, inhalation rate, body weight, food preferences).

All input values, equations, and exposure assumptions are provided in **Appendices C, D, and E.**

5.3.1 Receptor Characteristics and Exposure Pathways

The contribution of a particular route of exposure (e.g., inhalation, ingestion) to total exposure is determined by CoPC fate and behaviour in the environment, as well as by the receptor characteristics relevant to each route of exposure (e.g., breathing rate, food consumption rate, area of skin exposed).

A toddler was used to characterize risks to receptors associated with threshold CoPC, and a composite receptor (consisting of infant, toddler, child, youth and adult life stages) was used to

assess risk to non-threshold CoPC. In addition, the infant life stage was used to evaluate the exposure to breast milk. Specific receptor characteristics and exposure variables used in the current assessment are summarized in **Appendix A**.

5.3.2 Predicting Human Intakes

Daily intakes from all sources are discussed by scenario in the following sections and presented for individual CoPCs. Ingestion rates and receptor characteristics (e.g., body weight) were obtained from a variety of sources, including Health Canada (2004a), the Exposure Factors Handbook (US EPA 1997) or from Richardson (1997) and are briefly discussed in Section 5.1.3. A receptor's total intake is illustrated in Figure 5-2.

Daily intakes are calculated in the form of chronic daily intakes (CDIs) (to assess non-carcinogenic endpoints) and lifetime average daily doses (LADDs) (to assess carcinogenic endpoints), using the equations presented below:

$$CDI_i = Intake_{nc} \times C_i$$

$$LADD_i = Intake_c \times C_i$$

Where:

CDI_i	chronic daily intake via pathway *i*	mg/kg bw-day
$LADD_i$	lifetime average daily dose via pathway *i*	mg/kg bw-day
$Intake_{nc}$	intake rate for medium *i* (e.g., game) (non-carcinogenic)	kg medium/kg bw-day
$Intake_c$	intake rate for medium *i* (e.g., game) (carcinogenic)	kg medium/kg bw-day
C_i	concentration of chemical in medium *i* (e.g., game)	mg CoPC/kg medium

Tables of all CDIs and LADDs by scenario and receptor for all CoPCs are presented in **Appendix F**.

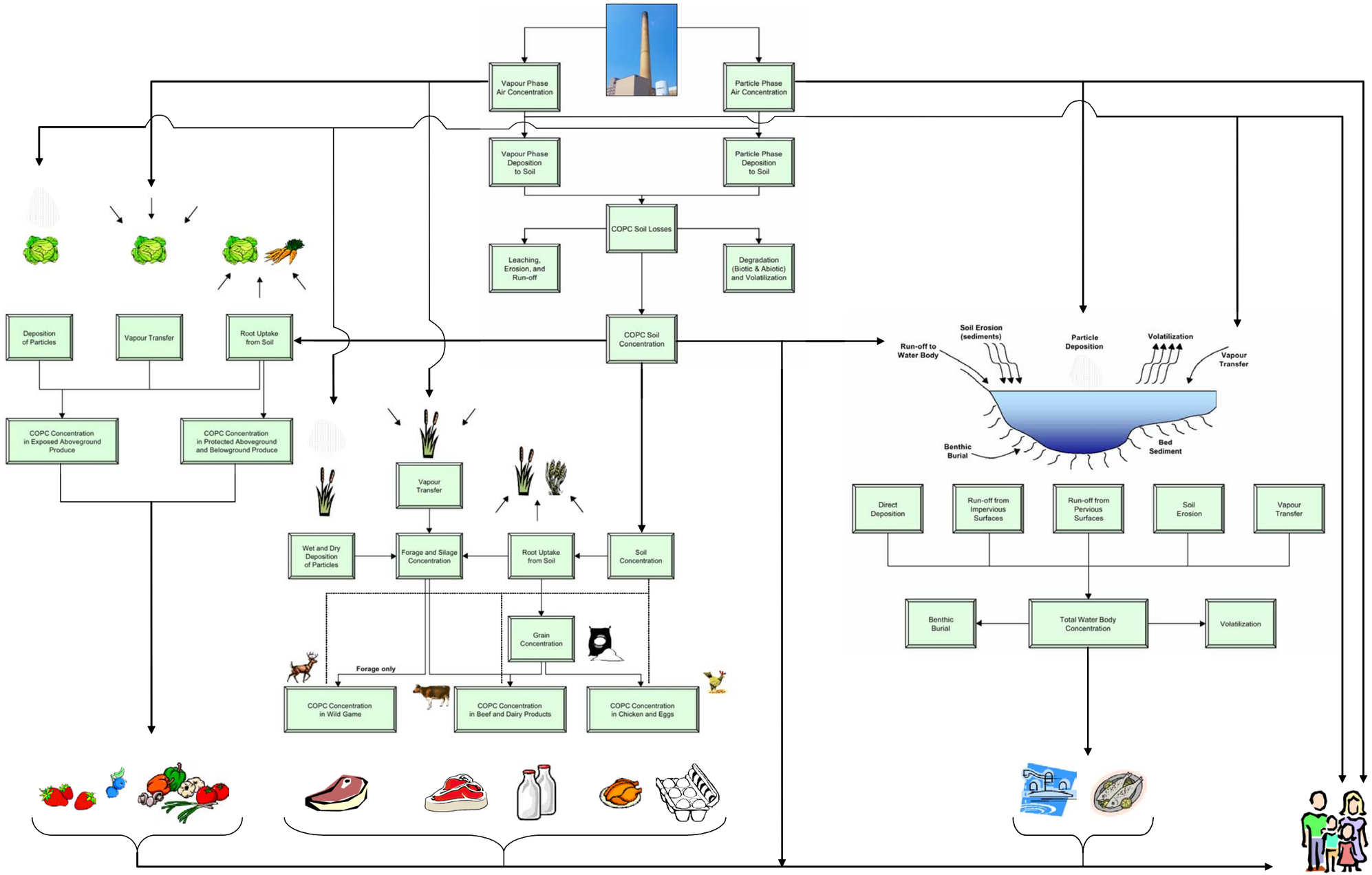


Figure 5-2 Exposure Model for Human Health Risk Assessment

5.4 Risk Characterization

The purpose of the risk characterization is to combine the information from the toxicity assessment (Section 5.2) and the results of the exposure assessment (Section 5.3) to estimate the potential risks to human health from the CoPC evaluated. This section briefly summarizes the general approach to the risk characterization for non-carcinogenic and carcinogenic CoPC, respectively.

5.4.1 Approach

Risk characterization is essentially a comparison of the predicted human intake of a CoPC to the toxicity reference value (TRV) for that CoPC. Evaluation of potential chronic and potential acute risks is completed separately; potential chronic health risks are evaluated using chronic daily intakes (CDIs) or lifetime average daily doses (LADDs), based on annual average air concentrations and depositions rates. Potential inhalation acute health risks are evaluated using short-term intakes, based on 1-hour and 24-hour air concentrations, compared with acute TRVs in Section 3.

The potential health effects associated with chemicals with non-carcinogenic endpoints are assessed differently than those for carcinogenic chemicals. Non-carcinogenic chemicals are generally considered to act through a threshold mechanism where it is assumed that there is a dose (or concentration) that does not produce any adverse effect. As the dose or concentration increases to the point where the body can no longer process or excrete the chemical, an adverse effect may occur. This point is termed the threshold and is different for every chemical.

For contaminants for which the critical effect is assumed to have no threshold (i.e., carcinogens), it is assumed that there is some probability of harm to human health at any level of exposure. There is a dose-response relationship that converts estimated daily intakes averaged over a lifetime of exposure directly to an incremental risk of an individual developing cancer.

Regulators in Ontario consider that a single increased case of cancer in an exposed population of 1,000,000 merits action. As such, a target risk (TR) of one in one million (E-06 or 10^{-6}) is used in the present analysis for carcinogenic effects.

5.4.2 Non-Carcinogens

The potential for adverse non-carcinogenic health effects for each CoPC is estimated by dividing the chronic daily intake (CDI) for each route of exposure (e.g., vapour and particulate inhalation, soil contact, drinking water, and food chain uptakes), by the reference dose (RfD) adjusted by a relative absorption factor for that RfD, as follows:

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

The computed ratios are termed the hazard quotients (HQs). The pathway-specific HQs for each CoPC are then summed to produce a total hazard quotient (HQ_{total}) for each CoPC in each of the four exposure scenarios:

If the HQ_{total} is less than 0.2, the intake of the CoPC by all routes of exposure does not exceed the tolerable intake and no adverse health effects are expected. HQ_{total} calculated in this risk assessment are benchmarked to 0.2 for the purposes of effects assessment. The use of a HQ benchmark of 0.2 is conservative as it allows 80% of the tolerable daily intake of a chemical to be received from other sources, including background.

5.4.3 Carcinogens

Carcinogenic risk characterization estimates the incremental probability that a person will develop cancer as a result of a lifetime of exposure to facility emissions. This incremental lifetime cancer risk (ILCR) is over and above the probability of developing cancer due to ambient exposures.

The likelihood of developing cancer is expressed as the product of the lifetime averaged daily dose (LADD) and the cancer slope factor (CSF) (adjusted by a relative absorption factor – assumed to be 1.0):

$$ILCR = LADD \text{ X } CSF$$

The product of the LADD and the CSF is unitless and provides an estimate of the potential carcinogenic risk associated with a CoPC. ILCRs are calculated for each CoPC for each exposure pathway. The pathway-specific ILCRs are then summed to produce a total ILCR for each CoPC by all exposure pathways in each of the four exposure scenarios.

If the $ILCR_{total}$ is less than the regulated acceptable limit of one in one million (E-06 or 10^{-6}) then it is concluded that there are no regulated unacceptable carcinogenic risks for that CoPC.

In all cases, HQ_{total} or $ILCR_{total}$ should be interpreted as conservative approximations. A HQ_{total} or $ILCR_{total}$ greater than 0.2 or 1E-06 does not necessarily imply that action is required to mitigate risks; rather, an exceedance is an indication that the data and assumptions used to estimate the risks should be more closely examined.

5.5 Effects Assessment

The assessments of potential chronic and acute risks were evaluated separately. Potential chronic health risks were evaluated using chronic daily intakes, based on annual average air concentrations and depositions rates, which were compared to TRVs. Potential acute health risks were evaluated using short-term daily intakes, based on 1-hour and 24-hour air concentrations, compared with ambient air quality criteria (AAQC).

A separate assessment was completed to assess the potential effects of changes in air quality on human health (Section 5.5.1). This was conducted in consideration of short-term exposure duration. To assess potential long-term changes to air, water, soil, and food resources and their effects on human health, a multi-media assessment was completed and is presented in Section 5.5.2. Long-term (chronic) inhalation exposure duration was assessed as part of the multi-media assessment.

5.5.1 Short Term (Acute) Inhalation Assessment

Short-term (i.e., acute) AAQC are founded on human health effects-based ambient air quality objectives for 1-hour and 24-hour averaging times. For the purposes of this screening of the modelled pollutants, only criteria based on human health effects were used. Criteria based on ecosystem health or nuisance factors, such as odour, were not considered. Acute effects are estimated by comparing the modelled air concentration to the AAQC (based on a short-term reference concentration or a minimum risk level (MRL)).

In general, the health-based AAQC were taken from the following regulatory agencies:

- Ontario Ministry of the Environment (MOE);
- Texas Commission on Environmental Quality (TCEQ);
- Alberta Ministry of the Environment (AENV); and
- World Health Organization.

For this assessment, preference was generally given to inhalation AAQC from MOE and TCEQ. Where toxicological information was available from both of these sources, the supporting documentation for each value was reviewed and selection of the final AAQC selection was based on the currency of the information and the level of protection.

For the purposes of deriving site-specific threshold levels, a chronic daily intake (CDI) is normally calculated for the exposed individual and compared to the AAQC. For the purposes of this screening, the modeled pollutant concentrations for each of the exposure durations (1-hour, 24-hour and annual) are conservatively considered the potential human inhalation exposure and will be employed rather than deriving a CDI.

As discussed in Section 5.4.2, the hazard quotient (HQ) represents the relationship between the magnitude of exposure to the contaminant from each route of exposure and the AAQC. The HQ indicates the probability of occurrence of adverse health effects. The benchmark HQ for acute inhaled exposures is 1.0. If the CDI/AAQC is greater than 1.0, then there may be potential for adverse health effects and further assessment would be required. Conversely, a HQ of less than 1.0 indicates that the intake of the pollutant from inhalation exposure does not exceed the tolerable intake and no adverse health effects are expected.

Hazard quotients were calculated for 1-hour and 24-hour exposure durations for Operating Scenario 1 (400,000 t/y). Hazard quotients were calculated for both the total modeled pollutant concentration in air, as well as for the total modeled pollutant concentration in air plus measured background air concentrations, where applicable. Please note that HQs were only calculated for those chemicals for which there were corresponding AAQC. For example, there was no 24-hour AAQC found for NO_x chemicals, therefore no HQ could be calculated for NO_x for 24-hour exposure.

5.5.1.1 Combustion Gases

Combustion gases [particulate matter, carbon monoxide, hydrogen chloride, hydrogen fluoride, nitrogen oxides (NO_x) and sulphur oxides (SO_x)] are contained within the MOE Guideline A-7 (*Combustion and Air Pollution Control Requirements for New Municipal Waste Incinerators*). In addition to the 1-hour and 24-hour average exposure pollutant concentrations, annual average

pollutant concentrations for the combustion gases were assessed, as chronic exposures are not assessed for combustion gases in the multi-media assessment. These are health based TRVs and reflect current toxicological standards. As shown in Table 3-5, ambient air quality concentrations (24-hour and annual average) are available for the region for $PM_{2.5}$, $NO_2$2, SO_2, and CO. These ambient concentrations have been added to the modelled 24-hour and annual average concentrations.

The results of the inhalation assessment for combustion gases are presented in Table 5-8.

As shown in Table 5-8, HQs for all combustion gases were less than 1.0 for the 1-hour, 24-hour, and annual average concentrations for Scenario 1. This indicates that the modelled 1-hour, 24-hour, and annual average concentrations of these pollutants do not pose an inhalation health risk to human receptors living at the MPOI, 24 hours per day. Because Scenario 1 represents the highest emission concentrations from the proposed facility, it follows that the combustion gases do not pose an inhalation health risk to human receptors living at the MPOI under any operating scenario.

Table 5-8 Combustion Gases Inhalation Assessment

Pollutant	AAQC 1-hour	AAQC 24-hour	AAQC Annual	Background 90th Percentile (24-hour equv.)	Background Annual	Operating Scenario 1 1-hour average		Operating Scenario 1 24-hour average				Operating Scenario 1 annual average			
						Total Pollutant Conc.	Total Pollutant HQ	Total Pollutant Conc.	Total Pollutant HQ	Total Pollutant Conc. + Bgd	Total + Bgd HQ	Total Pollutant Conc.	Total Pollutant HQ	Total Pollutant Conc. + Bgd	Total + Bgd HQ
Units	(µg/m^3)	(µg/m^3)	(µg/m^3)	(µg/m^3)	(µg/m^3)	(µg/m^3)		(µg/m^3)		(µg/m^3)		(µg/m^3)		(µg/m^3)	
Combustion Gases															
Total Particulate Matter PM	-- --	-- --	-- --			7.81E+00		2.27E+00		2.27E+00		2.35E-01		2.35E-01	
Particulate Matter PM10	-- --	50 [4]	20 [4]			7.81E+00		2.27E+00		2.27E+00		2.35E-01	0.012	2.35E-01	0.012
Particulate Matter PM2.5	-- --	25 [4]	10 [4]	20.0	8.1	6.92E+00		2.27E+00	0.0906	2.23E+01	0.89	2.35E-01	0.023	8.33E+00	0.833
Carbon Monoxide	15000 [1]	7000 [4]	-- --	721.7	469.7	1.61E+01	0.0011	3.03E+00	0.0004	7.25E+02	0.10	3.19E-01		4.70E+02	
Hydrogen Chloride	75 [1]	20 [3]	20 [2]			8.10E+00	0.1080	3.58E+00	0.1792	3.58E+00	0.18	3.70E-01	0.019	3.70E-01	0.019
Hydrogen Fluoride	4.9 [1]	-- --	-- --			7.23E-03	0.0015	3.20E-03		3.20E-03		3.30E-04		3.30E-04	
Nitrogen Oxides	200 [4]	200 [3]	40 [4]	45.0	22.0	6.38E+01	0.3192	2.76E+01	0.1380	7.26E+01	0.36	2.87E+00	0.072	2.48E+01	0.621
Sulphur Oxides [a]	450 [1]	20 [4]	-- --			1.68E+01	0.0373	7.46E+00	0.3728	7.46E+00	0.37	7.69E-01		7.69E-01	

NOTES:

[a] represented by sulphur dioxide

1. AENV - Alberta Environment
2. IRIS - US EPA Integrated Risk Information System
3. MOE - Ontario Ministry of the Environment
4. WHO - World Health Organization

Operating Scenario 1: Three (3) process units running 100% of the time

5.5.1.2 Acute Inhalation Risk for Chemicals of Potential Concern

As discussed above, all CoPC, other than combustion gases, including trace metals, chlorinated monocyclic aromatics, chlorinated polycyclic aromatics, PAHs, and VOCs, were assessed on a short-term (acute) basis only. As discussed in Section 3.1.4, there were no 1-hour or 24-hour average regional ambient monitoring data available, therefore the modelled results alone were compared to the AAQC.

The results of the 1-hour and 24-hour average inhalation assessment for CoPC other than combustion gases are presented in Tables 5-9 and 5-10, respectively.

As shown in Tables 5-9 and 5-10, HQs for all CoPC were less than 1.0 for the 1-hour and 24-hour average concentrations for Scenario 1. This indicates that the modelled 1-hour and 24-hour average concentrations of these pollutants do not pose a potential acute or short-term inhalation health risk to human receptors living at the MPOI, 24 hours per day. Given that Scenario 1 represents the highest emission concentrations from the proposed facility, it follows that the CoPC do not pose a potential acute or short-term inhalation health risk to human receptors living at the MPOI under any operating scenario.

Table 5-9 1-hour Acute Inhalation Assessment

Pollutant	AAQC 1-hour	Source Agency	Operating Scenario 1 1-hour average Total Pollutant Conc.	Total Pollutant HQ
Units	(µg/m^3)		(µg/m^3)	
Metals				
Antimony	5	TCEQ	8.22E-04	1.64E-04
Arsenic	5	TCEQ	1.81E-04	3.62E-05
Barium	5	TCEQ	1.42E-03	2.84E-04
Beryllium	0.02	TCEQ	1.92E-05	9.60E-04
Boron	100	TCEQ	4.59E-02	4.59E-04
Cadmium	0.1	TCEQ	4.20E-03	4.20E-02
Chromium (Total)	1	TCEQ	3.81E-04	3.81E-04
Cobalt	0.2	TCEQ	3.54E-05	1.77E-04
Lead	--	--	4.26E-02	
Mercury	0.25	TCEQ	6.00E-03	2.40E-02
Nickel	0.15	TCEQ	1.01E-03	6.72E-03
Phosphorus	1	TCEQ	6.93E-03	6.93E-03
Silver	0.1	TCEQ	1.41E-04	1.41E-03
Vanadium	--	--	2.01E-04	
Zinc	--	--	1.49E-02	
Chlorinated Monocyclic Aromatics				
1,2-Dichlorobenzene	30500	MOE	1.55E-05	5.07E-10
1,2,4-Trichlorobenzene	400	TCEQ	1.55E-05	3.86E-08
1,2,4,5-Tetrachlorobenzene	--	--	1.55E-05	
Pentachlorobenzene	1000	TCEQ	1.55E-05	1.55E-08
Hexachlorobenzene	0.25	TCEQ	1.55E-05	6.18E-05
2,4-Dichlorophenol	530	TCEQ	3.09E-05	5.83E-08
2,4,6-Trichlorophenol	--	--	1.55E-05	
2,3,4,6-Tetrachlorophenol	--	--	1.55E-05	
Pentachlorophenol	5	TCEQ	1.85E-05	3.70E-06
Chlorinated Polycyclic Aromatics				
PCB	0.1	TCEQ	2.17E-05	2.17E-04
2,3,7,8-TCDD TEQ	--	--	2.40E-08	
Polycyclic Aromatic Hydrocarbons				
Benzo(a)pyrene	3	TCEQ	8.04E-06	2.68E-06
Benzo(a)anthracene	--	--	8.04E-06	
Benzo(b)fluoranthene	0.5	TCEQ	8.04E-06	1.61E-05
Benzo(g,h,I)perylene	--	--	8.04E-06	
Benzo(k)fluoranthene	--	--	8.04E-06	
Chrysene	0.5	TCEQ	8.04E-06	1.61E-05
Dibenzo(a,h)anthracene	--	--	8.04E-06	
Indeno(1,2,3-c,d)pyrene	--	--	8.04E-06	
Anthracene	0.5	TCEQ	8.04E-06	1.61E-05
Naphthalene	--	--	4.92E-05	
Phenanthrene	0.5	TCEQ	2.46E-05	4.93E-05
Volatile Organic Compounds				
Benzene	30	AENV	1.08E-01	3.59E-03
Chloroform	100	TCEQ	1.53E-04	1.53E-06
Dichloromethane	260	TCEQ	5.27E-02	2.03E-04
Formaldehyde	65	AENV	1.42E-02	2.19E-04
Tetrachloroethylene	340	TCEQ	1.70E-03	5.00E-06
Vinyl Chloride	130	AENV	1.79E-04	1.37E-06

NOTES:
AENV - Alberta Environment
MOE - Ontario Ministry of the Environment
TCEQ - Texas Commision on Environmental Quality
WHO - World Health Organization
Operating Scenario 1: Three (3) process units running 100% of the time

Table 5-10 24-hour Short-term Inhalation Assessment

Pollutant	AAQC 24-hour	Source Agency	Operating Scenario 1	
			24-hour average	
			Total Pollutant Conc.	Total Pollutant HQ
Units	(µg/m^3)		(µg/m^3)	
Metals				
Antimony	25	MOE	3.65E-04	1.46E-05
Arsenic	0.3	MOE	8.04E-05	2.68E-04
Barium	10	MOE	6.31E-04	6.31E-05
Beryllium	0.01	MOE	8.52E-06	8.52E-04
Boron	--	--	2.04E-02	
Cadmium	2	MOE	1.86E-03	9.32E-04
Chromium (Total)	1.5	MOE	1.69E-04	1.13E-04
Cobalt	0.1	MOE	1.57E-05	1.57E-04
Lead	2	MOE	1.89E-02	9.45E-03
Mercury	2	MOE	2.66E-03	1.33E-03
Nickel	--	--	4.47E-04	
Phosphorus	--	--	3.07E-03	
Silver	--	--	6.26E-05	
Vanadium	2	MOE	8.92E-05	4.46E-05
Zinc	--	--	6.62E-03	
Chlorinated Monocyclic Aromatics				
1,2-Dichlorobenzene	--	--	6.84E-06	
1,2,4-Trichlorodibenzene	400	MOE	6.84E-06	1.71E-08
1,2,4,5-Tetrachlorobenzene	--	--	6.84E-06	
Pentachlorobenzene	--	--	6.84E-06	
Hexachlorobenzene	--	--	6.84E-06	
2,4-Dichlorophenol	--	--	1.37E-05	
2,4,6-Trichlorophenol	--	--	6.84E-06	
2,3,4,6-Tetrachlorophenol	--	--	6.84E-06	
Pentachlorophenol	20	MOE	8.18E-06	4.09E-07
Chlorinated Polycyclic Aromatics				
PCB	0.15	MOE	9.58E-06	6.39E-05
2,3,7,8-TCDD TEQ	--	--	1.06E-08	
Polycyclic Aromatic Hydrocarbons				
Benzo(a)pyrene	0.0011	MOE	3.56E-06	3.24E-03
Benzo(a)anthracene	--	--	3.56E-06	
Benzo(b)fluoranthene	--	--	3.56E-06	
Benzo(g,h,I)perylene	--	--	3.56E-06	
Benzo(k)fluoranthene	--	--	3.56E-06	
Chrysene	--	--	3.56E-06	
Dibenzo(a,h)anthracene	--	--	3.57E-06	
Indeno(1,2,3-c,d)pyrene	--	--	3.57E-06	
Anthracene	--	--	3.56E-06	
Naphthalene	22.5	MOE	2.18E-05	9.67E-07
Phenanthrene	--	--	1.09E-05	
Volatile Organic Compounds				
Benzene	--		9.85E-03	
Chloroform	1	MOE	6.77E-05	6.77E-05
Dichloromethane	3000	WHO	2.33E-02	7.77E-06
Formaldehyde	65	MOE	6.30E-03	9.70E-05
Tetrachloroethylene	250	WHO	7.53E-04	3.01E-06
Vinyl Chloride	1	MOE	7.90E-05	7.90E-05

NOTES:
MOE - Ontario Ministry of the Environment
WHO - World Health Organization
Operating Scenario 1: Three (3) process units running 100% of the time

5.5.2 Multi-Media Assessment

The multi-media risk assessment evaluated potential long-term effects on human health caused by CoPC in air or water that may accumulate in soil, plants, agriculture, wild game, or fish.

As previously discussed, for assessment of non-carcinogenic risks, HQs are presented for a toddler (6 months to 4 years of age) as this age group (toddler) is the most sensitive for non-carcinogenic risk assessment. HQs for the composite receptor are also presented for comparison purposes.

For carcinogenic risks, $ILCR_{total}$ are presented for a lifetime composite receptor, incorporating five life stages (infant, toddler, child, teen, and adult), with the exception of the commercial receptor, where $ILCR_{total}$ are presented for the adult, as discussed above. This is a very conservative, yet appropriate approach (exposure for an entire lifetime) to carcinogenic risk assessment.

Results of the risk characterization, expressed as HQ_{total} and $ILCR_{total}$, are discussed in the following sections and presented in **Appendix G** by scenario and receptor, for all CoPC. Summaries of the HQ_{total} and $ILCR_{total}$ by scenario are presented in Tables 5-11 through 5-13 followed by a brief discussion the exceedances by CoPC, for all receptors and all scenarios.

Table 5-11 Summary of HQ_{total} and $ILCR_{total}$ - Scenario 1: 3 Process Units

	Resident				Subsistence Farmer				First Nations				Commercial		
	Infant	Toddler	Composite		Infant	Toddler	Composite		Infant	Toddler	Composite		Toddler	Adult	
	Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	
Constituent	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	ILCR
BTEX															
Benzene	2.71E-05	8.21E-05	1.79E-05	2.49E-09	2.71E-05	8.68E-05	1.89E-05	2.73E-09	2.72E-05	1.09E-04	2.48E-05	4.02E-09	4.60E-05	2.22E-05	2.30E-09
PAHs															
Anthracene	3.26E-07	6.93E-07	1.38E-07	--	3.26E-07	6.94E-07	1.38E-07	--	3.26E-07	6.94E-07	1.38E-07	--	2.90E-07	1.15E-07	--
Benzo(a)pyrene	--	--	--	3.99E-08	--	--	--	5.24E-08	--	--	--	4.95E-08	--	--	2.73E-08
Naphthalene	8.72E-07	1.96E-06	3.93E-07	--	8.72E-07	2.07E-06	4.14E-07	--	8.72E-07	1.97E-06	3.94E-07	--	8.87E-07	3.57E-07	--
Phenanthrene	9.39E-09	5.52E-08	1.33E-08	--	9.62E-09	7.19E-08	1.61E-08	--	1.05E-08	1.07E-07	2.69E-08	--	4.42E-08	2.36E-08	--
PCBs															
Aroclor 1254 (Total PCBs)	6.43E-03	2.30E-04	6.06E-05	--	1.98E-02	7.14E-04	1.87E-04	--	1.48E-01	5.83E-03	1.53E-03	--	3.65E-06	1.37E-06	--
Dioxins and Furans															
2,3,7,8-TCDD Equivalent	1.43E-01	5.45E-03	1.37E-03	--	**8.61E-01**	6.28E-02	9.20E-03	--	**3.38E+00**	1.29E-01	3.47E-02	--	1.21E-03	4.50E-04	--
2,3,7,8-TCDD Equivalent - PEEL	1.80E-02	6.88E-04	1.73E-04	--	1.09E-01	7.92E-03	1.16E-03	--	**4.27E-01**	1.63E-02	4.38E-03	--	1.52E-04	5.68E-05	--
VOCs															
Chloroform	8.01E-06	1.77E-05	3.53E-06	1.75E-11	8.01E-06	1.78E-05	3.54E-06	1.75E-11	8.01E-06	1.78E-05	3.56E-06	1.75E-11	7.44E-06	2.97E-06	9.11E-12
Dichloromethane	1.52E-09	5.72E-06	1.53E-06	1.14E-09	1.54E-09	6.96E-06	1.79E-06	1.24E-09	1.55E-09	7.40E-06	1.97E-06	1.31E-09	5.52E-06	3.22E-06	1.05E-09
Formaldehyde	1.60E-09	1.16E-04	3.07E-05	1.72E-09	1.68E-09	1.84E-04	4.54E-05	1.72E-09	1.68E-09	1.67E-04	4.41E-05	1.72E-09	1.06E-04	6.21E-05	9.13E-10
Tetrachloroethylene	7.54E-09	1.19E-06	3.17E-07	1.68E-11	9.56E-09	2.33E-06	5.94E-07	1.68E-11	2.49E-08	1.09E-05	2.86E-06	1.68E-11	7.72E-07	4.51E-07	8.71E-12
Vinyl Chloride	9.37E-08	5.04E-07	1.21E-07	3.56E-10	9.37E-08	5.20E-07	1.24E-07	3.71E-10	9.37E-08	6.10E-07	1.49E-07	4.73E-10	3.79E-07	2.05E-07	4.59E-10
Chlorinated Monocyclic Aromatics															
1,2-Dichlorobenzene	4.24E-10	1.42E-09	3.12E-10	--	4.25E-10	2.39E-09	5.25E-10	--	4.30E-10	4.84E-09	1.21E-09	--	6.70E-10	3.18E-10	--
1,2,4-Trichlorobenzene	1.21E-07	6.82E-07	1.57E-07	--	1.31E-07	2.42E-06	5.45E-07	--	1.48E-07	4.60E-06	1.18E-06	--	2.16E-07	1.06E-07	--
1,2,4,5-Tetrachlorobenzene	2.56E-06	3.38E-05	8.81E-06	--	5.74E-06	1.00E-04	2.58E-05	--	3.52E-05	7.62E-04	2.00E-04	--	3.00E-06	1.55E-06	--
Pentachlorobenzene	9.33E-07	6.37E-06	1.65E-06	--	2.38E-06	1.95E-05	4.96E-06	--	1.59E-05	1.49E-04	3.89E-05	--	4.16E-07	1.83E-07	--
Hexachlorobenzene	3.92E-06	2.43E-05	6.43E-06	2.84E-09	1.18E-05	7.53E-05	1.98E-05	8.37E-09	8.72E-05	6.13E-04	1.61E-04	6.68E-08	3.54E-07	1.99E-07	1.67E-10
2,4-Dichlorophenol	4.12E-09	7.82E-07	2.07E-07	--	4.34E-09	1.23E-06	3.05E-07	--	4.92E-09	2.20E-06	5.79E-07	--	6.87E-07	4.00E-07	--
2,4,6-Trichlorophenol	3.31E-08	8.56E-06	2.23E-06	7.47E-11	7.86E-08	2.33E-05	5.54E-06	1.84E-10	2.00E-07	5.55E-05	1.45E-05	4.81E-10	5.49E-06	3.20E-06	6.67E-11
2,3,4,6-Tetrachlorophenol	1.50E-07	5.98E-06	1.59E-06	--	3.48E-07	1.53E-05	3.96E-06	--	2.19E-06	1.08E-04	2.83E-05	--	1.81E-06	1.05E-06	--
Pentachlorophenol	1.34E-05	1.35E-04	3.58E-05	2.58E-08	4.94E-05	6.94E-04	1.38E-04	9.94E-08	3.00E-04	3.32E-03	8.75E-04	6.30E-07	8.37E-06	4.89E-06	2.21E-09
Inorganics															
Antimony	2.50E-05	7.78E-05	1.65E-05	--	2.50E-05	1.28E-04	2.40E-05	--	2.50E-05	1.77E-04	4.29E-05	--	4.56E-05	2.06E-05	--
Arsenic	1.12E-07	9.08E-06	2.29E-06	1.89E-08	1.12E-07	2.80E-05	5.51E-06	2.04E-08	1.12E-07	9.43E-05	2.49E-05	2.91E-08	4.91E-06	2.76E-06	1.21E-08
Barium	8.67E-08	6.07E-07	1.47E-07	--	8.67E-08	1.37E-06	3.36E-07	--	8.67E-08	8.54E-06	2.22E-06	--	1.99E-07	9.27E-08	--
Beryllium	3.58E-09	3.10E-08	5.45E-09	1.06E-09	3.58E-09	1.72E-07	2.30E-08	1.06E-09	3.58E-09	1.37E-07	3.52E-08	1.06E-09	1.46E-08	5.24E-09	7.90E-10
Boron	2.70E-06	8.11E-06	1.64E-06	--	2.70E-06	1.76E-05	2.98E-06	--	2.70E-06	9.01E-06	1.91E-06	--	3.37E-06	1.49E-06	--
Cadmium	1.98E-06	1.93E-04	4.92E-05	9.46E-07	1.98E-06	6.07E-04	1.46E-04	9.46E-07	1.98E-06	3.78E-03	9.88E-04	9.46E-07	2.90E-05	1.55E-05	6.61E-07
Chromium (Total)	6.44E-08	4.12E-06	1.02E-06	9.92E-08	6.44E-08	1.96E-05	2.88E-06	9.92E-08	6.44E-08	1.90E-05	5.37E-06	9.92E-08	3.23E-06	1.83E-06	6.10E-08
Cobalt	1.86E-05	4.09E-05	8.14E-06	--	1.86E-05	4.14E-05	8.20E-06	--	1.86E-05	4.11E-05	8.22E-06	--	2.25E-05	8.94E-06	--
Lead	2.85E-04	6.95E-04	1.34E-04	--	2.85E-04	1.01E-03	1.76E-04	--	2.85E-04	7.17E-04	1.42E-04	--	5.01E-04	1.95E-04	--
Mercury - Elemental	2.10E-06	4.61E-06	9.18E-07	--	2.10E-06	4.61E-06	9.18E-07	--	2.10E-06	4.61E-06	9.18E-07	--	1.92E-06	7.62E-07	--
Mercury - Inorganic	1.14E-04	3.54E-04	6.92E-05	--	1.14E-04	1.40E-03	1.97E-04	--	1.14E-04	4.85E-04	1.13E-04	--	1.41E-04	5.77E-05	--
Methyl Mercury	6.99E-08	2.96E-02	7.84E-03	--	7.00E-08	9.12E-02	2.41E-02	--	7.01E-08	**7.58E-01**	1.99E-01	--	8.05E-07	3.14E-07	--
Nickel	6.36E-07	2.13E-06	4.62E-07	--	6.37E-07	5.03E-06	8.66E-07	--	6.37E-07	8.55E-06	2.21E-06	--	1.38E-06	6.20E-07	--
Phosphorous	5.70E-12	2.22E-11	4.63E-12	--	5.70E-12	2.69E-09	2.99E-10	--	5.70E-12	9.93E-10	3.31E-10	--	1.01E-11	4.89E-12	--
Silver	3.35E-07	1.44E-06	3.27E-07	--	3.35E-07	2.89E-06	5.69E-07	--	3.35E-07	7.22E-06	1.86E-06	--	7.59E-07	3.77E-07	--
Vanadium	2.75E-07	9.69E-07	2.12E-07	--	2.75E-07	1.51E-06	2.77E-07	--	2.75E-07	1.29E-06	3.08E-07	--	7.32E-07	3.46E-07	--
Zinc	6.06E-07	8.04E-06	2.01E-06	--	6.06E-07	2.36E-05	5.89E-06	--	6.06E-07	1.50E-04	3.93E-05	--	1.32E-06	5.94E-07	--

Table 5-12 Summary of HQ_{total} and $ILCR_{total}$ - Scenario 2: 2 Process Units

	Resident				Subsistence Farmer				First Nations				Commercial		
	Infant	Toddler	Composite		Infant	Toddler	Composite		Infant	Toddler	Composite		Toddler	Adult	
	Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	
Constituent	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	ILCR
BTEX															
Benzene	2.33E-05	7.00E-05	1.52E-05	2.11E-09	2.33E-05	7.39E-05	1.61E-05	2.30E-09	2.33E-05	9.21E-05	2.10E-05	3.39E-09	3.90E-05	1.87E-05	1.94E-09
PAHs															
Anthracene	2.77E-07	5.88E-07	1.17E-07	--	2.77E-07	5.89E-07	1.17E-07	--	2.77E-07	5.89E-07	1.17E-07	--	2.46E-07	9.79E-08	--
Benzo(a)pyrene	--	--	--	3.39E-08	--	--	--	4.45E-08	--	--	--	4.19E-08	--	--	2.29E-08
Naphthalene	7.40E-07	1.67E-06	3.33E-07	--	7.40E-07	1.75E-06	3.50E-07	--	7.40E-07	1.67E-06	3.34E-07	--	7.49E-07	3.02E-07	--
Phenanthrene	7.90E-09	4.54E-08	1.09E-08	--	8.08E-09	5.90E-08	1.32E-08	--	8.76E-09	8.75E-08	2.19E-08	--	3.61E-08	1.92E-08	--
PCBs															
Aroclor 1254 (Total PCBs)	5.22E-03	1.87E-04	4.92E-05	--	1.61E-02	5.81E-04	1.52E-04	--	1.20E-01	4.74E-03	1.24E-03	--	2.97E-06	1.12E-06	--
Dioxins and Furans															
2,3,7,8-TCDD Equivalent	1.16E-01	4.44E-03	1.12E-03	--	**7.16E-01**	5.26E-02	7.66E-03	--	**2.76E+00**	1.06E-01	2.84E-02	--	9.83E-04	3.67E-04	--
2,3,7,8-TCDD Equivalent - PEEL	1.47E-02	5.61E-04	1.41E-04	--	9.03E-02	6.65E-03	9.67E-04	--	**3.49E-01**	1.34E-02	3.58E-03	--	1.24E-04	4.63E-05	--
VOCs															
Chloroform	6.81E-06	1.51E-05	3.00E-06	1.49E-11	6.81E-06	1.51E-05	3.00E-06	1.49E-11	6.81E-06	1.51E-05	3.02E-06	1.49E-11	6.32E-06	2.52E-06	7.73E-12
Dichloromethane	1.29E-09	4.71E-06	1.26E-06	9.54E-10	1.30E-09	5.73E-06	1.48E-06	1.04E-09	1.31E-09	6.10E-06	1.62E-06	1.09E-09	4.55E-06	2.65E-06	8.72E-10
Formaldehyde	1.31E-09	9.59E-05	2.53E-05	1.46E-09	1.39E-09	1.52E-04	3.74E-05	1.46E-09	1.38E-09	1.38E-04	3.64E-05	1.46E-09	8.77E-05	5.12E-05	7.75E-10
Tetrachloroethylene	6.35E-09	9.82E-07	2.61E-07	1.42E-11	8.01E-09	1.92E-06	4.89E-07	1.42E-11	2.06E-08	8.99E-06	2.36E-06	1.42E-11	6.36E-07	3.71E-07	7.40E-12
Vinyl Chloride	7.96E-08	4.20E-07	1.00E-07	2.93E-10	7.96E-08	4.33E-07	1.04E-07	3.06E-10	7.96E-08	5.08E-07	1.23E-07	3.90E-10	3.14E-07	1.70E-07	3.78E-10
Chlorinated Monocyclic Aromatics															
1,2-Dichlorobenzene	3.60E-10	1.19E-09	2.60E-10	--	3.61E-10	1.97E-09	4.33E-10	--	3.65E-10	3.97E-09	9.89E-10	--	5.59E-10	2.64E-10	--
1,2,4-Trichlorobenzene	1.02E-07	5.63E-07	1.29E-07	--	1.11E-07	1.97E-06	4.45E-07	--	1.25E-07	3.75E-06	9.63E-07	--	1.79E-07	8.73E-08	--
1,2,4,5-Tetrachlorobenzene	2.11E-06	2.75E-05	7.17E-06	--	4.70E-06	8.14E-05	2.10E-05	--	2.86E-05	6.19E-04	1.62E-04	--	2.47E-06	1.27E-06	--
Pentachlorobenzene	7.66E-07	5.19E-06	1.35E-06	--	1.94E-06	1.58E-05	4.03E-06	--	1.29E-05	1.21E-04	3.16E-05	--	3.44E-07	1.51E-07	--
Hexachlorobenzene	3.19E-06	1.97E-05	5.22E-06	2.31E-09	9.62E-06	6.11E-05	1.60E-05	6.81E-09	7.08E-05	4.98E-04	1.30E-04	5.43E-08	2.87E-07	1.62E-07	1.39E-10
2,4-Dichlorophenol	3.48E-09	6.35E-07	1.68E-07	--	3.66E-09	9.99E-07	2.48E-07	--	4.13E-09	1.79E-06	4.70E-07	--	5.58E-07	3.25E-07	--
2,4,6-Trichlorophenol	2.69E-08	6.95E-06	1.81E-06	6.07E-11	6.39E-08	1.89E-05	4.50E-06	1.49E-10	1.63E-07	4.51E-05	1.18E-05	3.90E-10	4.46E-06	2.60E-06	5.42E-11
2,3,4,6-Tetrachlorophenol	1.23E-07	4.86E-06	1.29E-06	--	2.84E-07	1.24E-05	3.22E-06	--	1.78E-06	8.78E-05	2.30E-05	--	1.47E-06	8.55E-07	--
Pentachlorophenol	1.09E-05	1.10E-04	2.91E-05	2.10E-08	4.05E-05	5.75E-04	1.13E-04	8.17E-08	2.44E-04	2.70E-03	7.13E-04	5.13E-07	6.81E-06	3.97E-06	1.79E-09
Inorganics															
Antimony	2.12E-05	6.30E-05	1.33E-05	--	2.12E-05	9.96E-05	1.87E-05	--	2.12E-05	1.35E-04	3.24E-05	--	3.58E-05	1.60E-05	--
Arsenic	8.36E-08	6.74E-06	1.70E-06	1.60E-08	8.36E-08	2.06E-05	4.07E-06	1.70E-08	8.36E-08	7.01E-05	1.85E-05	2.35E-08	3.65E-06	2.06E-06	9.97E-09
Barium	7.33E-08	4.74E-07	1.14E-07	--	7.33E-08	1.04E-06	2.56E-07	--	7.33E-08	6.43E-06	1.67E-06	--	1.57E-07	7.26E-08	--
Beryllium	2.69E-09	2.29E-08	4.04E-09	9.00E-10	2.69E-09	1.26E-07	1.68E-08	9.00E-10	2.69E-09	1.02E-07	2.62E-08	9.00E-10	1.09E-08	3.93E-09	6.47E-10
Boron	2.29E-06	6.62E-06	1.34E-06	--	2.29E-06	1.35E-05	2.30E-06	--	2.29E-06	7.27E-06	1.53E-06	--	2.75E-06	1.20E-06	--
Cadmium	1.49E-06	1.45E-04	3.69E-05	8.04E-07	1.49E-06	4.54E-04	1.09E-04	8.04E-07	1.49E-06	2.84E-03	7.42E-04	8.04E-07	2.18E-05	1.16E-05	5.44E-07
Chromium (Total)	4.84E-08	3.08E-06	7.65E-07	8.39E-08	4.84E-08	1.43E-05	2.12E-06	8.39E-08	4.84E-08	1.41E-05	3.98E-06	8.39E-08	2.42E-06	1.37E-06	5.07E-08
Cobalt	1.58E-05	3.48E-05	6.92E-06	--	1.58E-05	3.51E-05	6.95E-06	--	1.58E-05	3.49E-05	6.97E-06	--	1.84E-05	7.32E-06	--
Lead	2.41E-04	5.79E-04	1.12E-04	--	2.41E-04	8.11E-04	1.43E-04	--	2.41E-04	5.95E-04	1.18E-04	--	3.99E-04	1.55E-04	--
Mercury - Elemental	1.78E-06	3.92E-06	7.79E-07	--	1.78E-06	3.92E-06	7.79E-07	--	1.78E-06	3.92E-06	7.79E-07	--	1.63E-06	6.47E-07	--
Mercury - Inorganic	9.70E-05	3.00E-04	5.86E-05	--	9.70E-05	1.16E-03	1.63E-04	--	9.70E-05	4.04E-04	9.37E-05	--	1.18E-04	4.84E-05	--
Methyl Mercury	5.42E-08	2.50E-02	6.62E-03	--	5.43E-08	7.70E-02	2.04E-02	--	5.43E-08	**6.40E-01**	1.68E-01	--	6.25E-07	2.43E-07	--
Nickel	5.36E-07	1.73E-06	3.72E-07	--	5.36E-07	3.85E-06	6.70E-07	--	5.36E-07	6.52E-06	1.68E-06	--	1.09E-06	4.87E-07	--
Phosphorous	4.84E-12	1.77E-11	3.67E-12	--	4.84E-12	1.95E-09	2.17E-10	--	4.84E-12	7.22E-10	2.41E-10	--	8.00E-12	3.80E-12	--
Silver	2.84E-07	1.15E-06	2.59E-07	--	2.84E-07	2.22E-06	4.39E-07	--	2.84E-07	5.49E-06	1.41E-06	--	6.00E-07	2.95E-07	--
Vanadium	2.32E-07	7.74E-07	1.68E-07	--	2.32E-07	1.17E-06	2.15E-07	--	2.32E-07	1.01E-06	2.38E-07	--	5.60E-07	2.63E-07	--
Zinc	5.12E-07	6.15E-06	1.53E-06	--	5.12E-07	1.78E-05	4.43E-06	--	5.12E-07	1.13E-04	2.94E-05	--	1.04E-06	4.66E-07	--

Table 5-13 Summary of HQ_{total} and $ILCR_{total}$ - Scenario 3: 1 Process Unit

	Resident				Subsistence Farmer				First Nations				Commercial		
	Infant	Toddler	Composite		Infant	Toddler	Composite		Infant	Toddler	Composite		Toddler	Adult	
	Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	Total (All Pathways)		Total (All Pathways)	Total (All Pathways)	
Constituent	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	Hazard Quotient	ILCR	Hazard Quotient	Hazard Quotient	ILCR
BTEX															
Benzene	2.03E-05	5.79E-05	1.24E-05	1.66E-09	2.03E-05	6.07E-05	1.31E-05	1.80E-09	2.03E-05	7.37E-05	1.66E-05	4.02E-09	3.11E-05	1.47E-05	1.46E-09
PAHs															
Anthracene	2.03E-07	4.32E-07	8.60E-08	--	2.03E-07	4.32E-07	8.61E-08	--	2.03E-07	4.32E-07	8.61E-08	--	1.81E-07	7.18E-08	--
Benzo(a)pyrene	--	--	--	2.49E-08	--	--	--	3.26E-08	--	--	--	4.95E-08	--	--	1.62E-08
Naphthalene	5.43E-07	1.22E-06	2.44E-07	--	5.43E-07	1.28E-06	2.56E-07	--	5.43E-07	1.22E-06	2.45E-07	--	5.46E-07	2.20E-07	--
Phenanthrene	5.73E-09	3.19E-08	7.64E-09	--	5.86E-09	4.13E-08	9.24E-09	--	6.32E-09	6.10E-08	1.53E-08	--	2.52E-08	1.34E-08	--
PCBs															
Aroclor 1254 (Total PCBs)	3.65E-03	1.30E-04	3.44E-05	--	1.13E-02	4.06E-04	1.06E-04	--	8.39E-02	3.31E-03	8.68E-04	--	2.09E-06	7.84E-07	--
Dioxins and Furans															
2,3,7,8-TCDD Equivalent	8.07E-02	3.08E-03	7.75E-04	--	**5.08E-01**	3.78E-02	5.45E-03	--	**1.92E+00**	7.35E-02	1.97E-02	--	6.81E-04	2.54E-04	--
2,3,7,8-TCDD Equivalent - PEEL	1.02E-02	3.89E-04	9.78E-05	--	6.41E-02	4.77E-03	6.88E-04	--	**2.43E-01**	9.28E-03	2.49E-03	--	8.60E-05	3.21E-05	--
VOCs															
Chloroform	5.00E-06	1.11E-05	2.20E-06	1.09E-11	5.00E-06	1.11E-05	2.20E-06	1.09E-11	5.00E-06	1.11E-05	2.22E-06	1.75E-11	4.63E-06	1.85E-06	5.68E-12
Dichloromethane	9.46E-10	3.31E-06	8.84E-07	6.86E-10	9.54E-10	4.03E-06	1.04E-06	7.44E-10	9.59E-10	4.29E-06	1.14E-06	1.31E-09	3.20E-06	1.87E-06	6.21E-10
Formaldehyde	9.26E-10	6.76E-05	1.79E-05	1.07E-09	9.77E-10	1.07E-04	2.64E-05	1.07E-09	9.74E-10	9.73E-05	2.56E-05	1.72E-09	6.18E-05	3.61E-05	5.68E-10
Tetrachloroethylene	4.60E-09	6.91E-07	1.84E-07	1.05E-11	5.77E-09	1.35E-06	3.44E-07	1.05E-11	1.46E-08	6.32E-06	1.66E-06	1.68E-11	4.48E-07	2.61E-07	5.43E-12
Vinyl Chloride	5.84E-08	3.01E-07	7.17E-08	2.07E-10	5.84E-08	3.10E-07	7.39E-08	2.16E-10	5.84E-08	3.63E-07	8.79E-08	4.73E-10	2.23E-07	1.20E-07	2.66E-10
Chlorinated Monocyclic Aromatics															
1,2-Dichlorobenzene	2.64E-10	8.57E-10	1.87E-10	--	2.65E-10	1.41E-09	3.08E-10	--	2.67E-10	2.80E-09	6.97E-10	--	4.02E-10	1.89E-10	--
1,2,4-Trichlorobenzene	7.50E-08	4.02E-07	9.18E-08	--	8.10E-08	1.39E-06	3.12E-07	--	9.08E-08	2.63E-06	6.75E-07	--	1.28E-07	6.23E-08	--
1,2,4,5-Tetrachlorobenzene	1.50E-06	1.93E-05	5.02E-06	--	3.31E-06	5.69E-05	1.47E-05	--	2.00E-05	4.33E-04	1.13E-04	--	1.75E-06	8.99E-07	--
Pentachlorobenzene	5.42E-07	3.64E-06	9.43E-07	--	1.37E-06	1.11E-05	2.82E-06	--	9.02E-06	8.45E-05	2.21E-05	--	2.46E-07	1.07E-07	--
Hexachlorobenzene	2.23E-06	1.38E-05	3.65E-06	1.62E-09	6.73E-06	4.27E-05	1.12E-05	4.76E-09	4.95E-05	3.48E-04	9.12E-05	6.68E-08	2.01E-07	1.13E-07	9.95E-11
2,4-Dichlorophenol	2.54E-09	4.44E-07	1.18E-07	--	2.66E-09	6.99E-07	1.73E-07	--	3.00E-09	1.25E-06	3.29E-07	--	3.91E-07	2.27E-07	--
2,4,6-Trichlorophenol	1.88E-08	4.86E-06	1.27E-06	4.25E-11	4.47E-08	1.32E-05	3.15E-06	1.04E-10	1.14E-07	3.15E-05	8.25E-06	4.81E-10	3.12E-06	1.82E-06	3.79E-11
2,3,4,6-Tetrachlorophenol	8.63E-08	3.40E-06	9.02E-07	--	1.99E-07	8.71E-06	2.25E-06	--	1.25E-06	6.14E-05	1.61E-05	--	1.03E-06	5.98E-07	--
Pentachlorophenol	7.64E-06	7.69E-05	2.04E-05	1.47E-08	2.86E-05	4.11E-04	8.04E-05	5.79E-08	1.71E-04	1.89E-03	4.99E-04	6.30E-07	4.77E-06	2.78E-06	1.26E-09
Inorganics															
Antimony	1.50E-05	3.35E-05	6.68E-06	--	1.50E-05	3.43E-05	6.80E-06	--	1.50E-05	3.52E-05	7.14E-06	--	1.42E-05	5.67E-06	--
Arsenic	1.72E-08	1.27E-06	3.31E-07	1.13E-08	1.72E-08	2.63E-06	6.47E-07	1.14E-08	1.72E-08	1.38E-05	3.61E-06	2.91E-08	7.48E-07	4.21E-07	6.22E-09
Barium	5.26E-08	1.96E-07	4.42E-08	--	5.26E-08	3.35E-07	8.02E-08	--	5.26E-08	1.76E-06	4.53E-07	--	7.18E-08	3.12E-08	--
Beryllium	7.07E-10	3.80E-09	7.06E-10	6.60E-10	7.07E-10	8.59E-09	1.50E-09	6.60E-10	7.07E-10	1.98E-08	4.93E-09	1.06E-09	2.87E-09	1.03E-09	3.90E-10
Boron	1.68E-06	3.73E-06	7.42E-07	--	1.68E-06	3.89E-06	7.65E-07	--	1.68E-06	3.74E-06	7.47E-07	--	1.55E-06	6.19E-07	--
Cadmium	3.91E-07	3.68E-05	9.49E-06	5.89E-07	3.91E-07	1.10E-04	2.75E-05	5.89E-07	3.91E-07	7.43E-04	1.94E-04	9.46E-07	5.71E-06	3.05E-06	3.39E-07
Chromium (Total)	1.27E-08	7.20E-07	1.87E-07	6.02E-08	1.27E-08	1.22E-06	2.68E-07	6.02E-08	1.27E-08	2.59E-06	6.85E-07	9.92E-08	6.36E-07	3.60E-07	3.31E-08
Cobalt	1.16E-05	2.55E-05	5.07E-06	--	1.16E-05	2.55E-05	5.07E-06	--	1.16E-05	2.55E-05	5.07E-06	--	1.07E-05	4.25E-06	--
Lead	1.71E-04	3.83E-04	7.57E-05	--	1.71E-04	4.02E-04	7.86E-05	--	1.71E-04	3.83E-04	7.59E-05	--	1.97E-04	7.74E-05	--
Mercury - Elemental	1.31E-06	2.88E-06	5.72E-07	--	1.31E-06	2.88E-06	5.72E-07	--	1.31E-06	2.88E-06	5.72E-07	--	1.20E-06	4.75E-07	--
Mercury - Inorganic	7.08E-05	2.16E-04	4.23E-05	--	7.08E-05	7.46E-04	1.07E-04	--	7.08E-05	2.69E-04	6.01E-05	--	8.05E-05	3.33E-05	--
Methyl Mercury	2.19E-08	1.78E-02	4.72E-03	--	2.20E-08	5.49E-02	1.45E-02	--	2.20E-08	**4.56E-01**	1.19E-01	--	2.53E-07	9.86E-08	--
Nickel	3.77E-07	9.62E-07	1.99E-07	--	3.77E-07	1.17E-06	2.39E-07	--	3.77E-07	2.07E-06	4.92E-07	--	5.02E-07	2.14E-07	--
Phosphorous	3.54E-12	7.96E-12	1.59E-12	--	3.54E-12	5.02E-11	6.25E-12	--	3.54E-12	2.26E-11	6.49E-12	--	3.33E-12	1.34E-12	--
Silver	2.07E-07	5.87E-07	1.25E-07	--	2.07E-07	7.51E-07	1.59E-07	--	2.07E-07	1.68E-06	4.13E-07	--	2.79E-07	1.26E-07	--
Vanadium	1.63E-07	3.66E-07	7.32E-08	--	1.63E-07	3.74E-07	7.41E-08	--	1.63E-07	3.71E-07	7.47E-08	--	1.58E-07	6.38E-08	--
Zinc	3.67E-07	2.03E-06	4.79E-07	--	3.67E-07	4.81E-06	1.18E-06	--	3.67E-07	2.81E-05	7.31E-06	--	4.77E-07	2.02E-07	--

Commercial Receptor

The Durham-York commercial receptor is assumed to live outside of the region but work full time in the vicinity of the facility. HQs and ILCRs for the Durham-York commercial receptor all met the appropriate benchmarks. This suggests that up to a 400,000 t/y EFW facility could be located within a commercial zone of land use without appreciable risk to receptors over its 35 year timeframe.

Durham-York Resident

The Durham-York resident is assumed to live full time in the region, have a backyard garden, and eat some locally caught fish (34% of fish intake). HQs and ILCRs for the Durham-York resident all met the appropriate benchmarks.

Durham-York Subsistence Farmer

The Durham-York subsistence farmer is assumed to live full time in the region and obtain 100% of their food (e.g., meat, fish, poultry, eggs, milk, produce) year-round from their farm. HQs and ILCRs for the Durham-York subsistence farmer all met the appropriate benchmarks, with the following exceptions:

- Scenario 1: Infant 2,3,7,8-TCDD Equivalent in breast milk (0.86)
- Scenario 2: Infant 2,3,7,8-TCDD Equivalent in breast milk (0.72)
- Scenario 3: Infant 2,3,7,8-TCDD Equivalent in breast milk (0.51)

Although the 2,3,7,8-TCDD TEQ modelled from the A-7 Guideline posed a potential risk for breast fed infants, a second scenario for TCDD TEQ was modelled as the actual current emission rate from the KMS Peel EFW facility. Under this scenario the HQ for breast fed infants dropped to a level below the acceptable benchmark. This suggests that subsistence farms could be located within the MPOI if dioxin and furan levels were reduced below the current MOE A-7 Guideline.

Durham-York First Nations and Métis

The Durham-York First Nations and Métis receptor is assumed to live full time in the region, have a backyard garden, and eat locally caught fish and wild game all at the MPOI (maximum ground level air concentration). HQs and ILCRs for the Durham-York First Nations and Métis receptor all met the appropriate benchmarks, with the following exceptions:

- Scenario 1: Infant 2,3,7,8-TCDD Equivalent in breast milk (3.38)
  Toddler Methyl Mercury in fish (0.76)
- Scenario 2: Infant 2,3,7,8-TCDD Equivalent in breast milk (2.76)
  Toddler Methyl Mercury in fish (0.64)
- Scenario 3: Infant 2,3,7,8-TCDD Equivalent in breast milk (1.92)
  Toddler Methyl Mercury in fish (0.46)

The First Nations receptor was included in the risk assessment to ascertain the potential risk to such a community living at the MPOI from an EFW facility; however, the closest First Nations Reserve to any of the five short listed sites under consideration is greater than 40 km away. Although the study suggests that it would not be appropriate to site such a facility nearby a First Nations community, it should be noted that the assumptions used in this risk assessment are very conservative, given its generic nature.

Similar to the findings for the subsistence farmer scenario, the risk of infants exposed to 2,3,7,8 - TCDD in breast milk is greatly reduced if the KMS Peel EFW facility current day emissions are modelled. However, concentrations were still elevated above the regulatory acceptable benchmark of 0.2.

Although methyl mercury concentrations in fish posed a potential risk to the toddler age group, this phenomenon has already been reported by Heath Canada (2007) for toddlers ingesting store bought fish. In fact, given the very conservative consumption rate of fish modelled (95 g/d), being higher than that modeled by Health Canada (10 g/d), and that all fish consumption would be from the theoretically modelled watershed and lake, exposure to methylmercury in fish is not expected to pose an undue risk to the toddler. That being said this is an area that requires further attention during the site-specific risk assessment.

5.6 Uncertainty Analysis for the Human Health Risk Assessment

Risk estimates normally include an element of uncertainty, and generally these uncertainties are addressed by incorporating conservative assumptions in the analysis. As a result, risk assessments tend to overstate the actual risk. Although many factors are considered in preparation of a risk analysis, analysis results are generally only sensitive to very few of these factors. The uncertainty analysis is included to demonstrate that assumptions used are conservative, or that the analysis result is not sensitive to the key assumptions.

A risk assessment containing a high degree of confidence will be based on:

- Conditions where the problem is defined with a high level of certainty based on data and physical observations;
- An acceptable and reasonable level of conservatism in assumptions which will ensure that risks are overstated; and
- An appreciation of the bounds and limitations of the final solution.

The exposure assessment performed as part of this study was based on:

- Available data to describe predicted facility emissions and current and reasonably foreseeable land use conditions;
- Sound conservative assumptions for certain parameters, as required; and
- Well-understood and generally accepted methods for risk prediction.

An evaluation of the major uncertainties and their potential effect on the findings is presented in the following sections.

5.6.1 Uncertainties in the Toxicity Assessment

5.6.1.1 Uncertainties in Toxicological Information

There is a very limited amount of toxicological information on the effects associated with human exposures to low levels of chemicals in the environment. What human information is available is generally based on epidemiological studies of occupationally exposed workers. These studies are generally limited in scope and provide results that may not be applicable to chronic or continuous exposures to low levels of chemicals. Because human toxicological information is limited, reference doses and cancer potency estimates for many compounds are based on the results of dose-response assessment studies using animals.

The use of experimental animal data to estimate potential biological effects in humans introduces uncertainties into the evaluation of potential human health effects. These estimations require that a number of assumptions be made:

- The toxicological effect reported in animals is relevant and could occur in humans.
- The assumption that extrapolation from high-dose studies to low-dose environmental exposures adequately represents the shape of the dose-response curve in the low-dose exposure range.
- Short-term exposures used in animal studies can be extrapolated to chronic or long-term exposures in humans.
- The uptake of a compound from a test vehicle (drinking water, food, etc) in animals will be the same as the uptake of the chemical from environmental media (soil, sediment, air-borne particulate matter) in humans.
- The pharmacokinetic processes that occur in the test animals also occur in humans.

There are clearly a number of uncertainties associated with extrapolating from experimental animal data to humans. In order to address these uncertainties, regulatory agencies, such as Health Canada and the US EPA incorporate a large number of conservative assumptions to try and account for the uncertainties associated with this process. The uncertainties are accounted for by the use of Uncertainty Factors that are used to lower the reference dose well below the level at which adverse health effects have been reported in the test species. Uncertainty factors are generally applied by factors of 10 and are used to account for the following types of uncertainties:

- Variation within the population (protection of sensitive members of the population).
- Differences between humans and the test species.
- Differences in using short or medium-term studies to estimate the health effects associated with long-term or chronic exposures.
- Limitations in available toxicological information.

The magnitude of the uncertainty factors applied by the various regulatory agencies provides an indication of the level of confidence that should be placed on the reference value. Uncertainty factors typically range between 100 and 10,000, although some can be lower than 10. The latter values are found for a few chemicals where sound and substantial human toxicological

information is available to enable the setting of toxicological end-point solely on the basis of human epidemiological information.

The application of uncertainty factors is intended to introduce a high degree of conservatism into the risk assessment process and to ensure, as far as possible, that limited exposures that exceed the reference concentrations will not result in adverse human health effects. Because risk assessments that use these regulatory limits incorporate the conservatism used in the development of the toxicological information, the results can generally be viewed as being extremely conservative.

5.6.1.2 Use of Surrogates

For the purposes of this assessment, PAHs and dioxins and furans were assessed using a toxic equivalency factor (TEF) surrogate approach.

Health Canada reports that it has recently commissioned a report to summarize the approach regarding assessment of carcinogenic and non-carcinogenic PAHs. This report is expected to provide a list of Potency Equivalent Factors/Toxic Equivalent Factors (PEFs/TEFs) utilizing the approach from the World Health Organization (WHO), which can be applied to various PAHs.

The surrogate approach for PAHs is based on the assessment of a whole mixture of PAH compounds assuming that any combination of PAH compounds is considered a dilution of a "surrogate" mixture of PAHs. The "surrogate" is generally considered to be a potent PAH-mixture with well defined chemistry and toxicology. The approach uses a single compound, benzo[a]pyrene, as the surrogate for the PAH fraction of other complex mixtures. Using this methodology, the risk from any PAH mixture of concern can be estimated as the product of the environmental levels of benzo[a]pyrene and the estimate of risk attributable to mixtures per unit amount of B[a]P. In general the approach does not predict the potency of an ambient complex mixture, only it's PAH component.

The relative potencies, or TEFs, can be used in one of two ways:

i. to modify the TRV for benzo(a)pyrene for each carcinogenic PAH; or

ii. to calculate benzo(a)pyrene equivalent concentrations for each of the PAHs and evaluate the total benzo(a)pyrene equivalent (B[a]PTEQ) concentration against the benzo(a)pyrene TRV.

The two approaches are mathematically equivalent however the second method is commonly used and consistent with existing approaches for the evaluation of mixtures of dioxin and furan. It was this second method that was adopted for use in this assessment.

For dioxins and furans, the emissions of all individual chemicals were summed to provide a total emission for the group. The chemical with the highest toxic potency (2,3,7,8-TCDD) was then chosen to represent the group and was compared to the sum of the individual emissions. This approach is inherently conservative and is likely to overestimate the risks. It also permits a surrogate evaluation of those chemicals for which no toxicological information was available.

5.6.2 Uncertainties in the Exposure Assessment

5.6.2.1 Estimation of Deposition Rates and Air Concentrations

CoPC concentrations in air and deposition rates were calculated based on theoretical maximum emission rates of vapours and particulates. The majority of the exhaust stack air emission estimates used in this study were based on pollutant emission concentration values obtained from annual stack testing of an existing similar thermal treatment facility in Ontario (the maximum emission concentrations from the 2003, 2004 and 2005 stack testing of the existing facility were used). The Guideline A-7 emission concentration limits were used as default exhaust stack air emission estimates for the eight (8) pollutants contained in the guideline to show that government regulations are stringent enough and do not pose risk. The air emission estimates from on-site traffic due to waste delivery and ash removal are based on US EPA MOBILE6.2 emission factors for heavy-duty diesel vehicles in the calendar year 2010. These maximum emissions are assumed to occur throughout the lifetime of the facility and therefore are likely to overestimate actual emissions.

Receptors were placed at the maximum points of impingement for their entire exposure duration (entire lifetime for carcinogenic exposures). Other receptor locations would experience lower air concentrations and deposition rates. In addition, receptors are unlikely to spend all (if any) of their time at the MPOI. These assumptions are likely to result in some overestimation of the potential risks.

5.6.2.2 Facility Location

The assessment of the proposed facility has been undertaken by assuming various 'site-specific' parameters, including the size of the watershed and the waterbody within it, the soil characteristics (e.g., density, permeability. erodibility), climate characteristics (e.g., precipitation, temperature), and waterbody characteristics (e.g., depth, sediment depth, flowrates). Wherever practical, region-specific values have been adopted, or conservative default assumptions have been made.

5.6.2.3 Background Concentrations

Background concentrations were not explicitly included within the fate and transport modelling, as it is expected that a site-specific background sampling program will be completed as part of the facility location selection process. A decision will be made as to the applicability of these results to the human health risk assessment and these findings may be incorporated into the assessment at that time.

5.6.2.4 Watershed Concentrations

Air concentrations and deposition rates for the hypothetical Lake watershed were set equal to the MPOI within the watershed. Average air concentrations and deposition rates over the entire watershed would be much lower than the MPOI values selected.

Estimating surface water and sediment CoPC concentrations involves numerous assumptions related to the fate and transport of the CoPC and physical processes such as surface run-off and soil erosion loads. US EPA guidance was followed and, wherever possible, local or regional information was used to define the watershed characteristics. This was inherently

difficult, due to the broadly defined region within which the facility may be constructed, and the fact that it was a hypothetical waterbody being assessed.

5.6.2.5 Food Chain Uptakes

Estimation of CoPC uptake through the food chain involves the use of assumptions regarding many factors, including root uptake factors, air to plant transfer factors, biotransfer and bioconcentration factors, and crop and soil ingestion rates. Typically, these assumptions are conservative and tend to overestimate, rather than underestimate, risks.

In addition, for the purposes of this risk assessment, we have assumed that each receptor obtains all their drinking water from the hypothetical lake at the MPOI within the watershed. It is assumed that this water source would not undergo any treatment or filtering prior to consumption. This is not likely within the Durham-York region but this assumption is employed as a conservative measure.

5.6.2.6 Receptor Characteristics

For each receptor scenario, all receptors were assumed to live at the MPOI location (land use specific or maximum) for the entire exposure duration. For carcinogenic chemicals, this equates to 24 hours/day, 365 days/year for 75 years. Throughout this exposure duration, dietary intakes are supplemented by produce and agricultural products grown or raised at the same location. These assumptions will combine to overestimate the potential risks, especially in the case of the Subsistence Farmer.

5.6.3 Uncertainties in the Risk Characterizations

5.6.3.1 Chemical Interactions

The risk assessment of contaminants is complicated by the reality that most toxicological studies are conducted on single chemicals, but exposures are rarely limited to single chemicals. Exposures generally involve more than one contaminant. Although chemicals in the environment are most often present in some sort of mixture, guidelines for protection of human health are almost exclusively based on exposure to single chemicals.

Chemicals in a mixture may interact in four general ways to elicit a response:

- **Non-interacting** – chemicals have no effect in combination with each other; the toxicity of the mixture is the same as the toxicity of the most toxic component of the mixture;
- **Additive** – chemicals have similar targets and modes of action but do not interact, the hazard for exposure to the mixture is simply the sum of hazards for the individual chemicals;
- **Synergistic** – there is a positive interaction among the chemicals such that the response is greater than would be expected if the chemicals acted independently; and
- **Antagonistic** – there is a negative interaction among the chemicals such that the response is less than would be expected if the chemicals acted independently.

For human health exposures, quantitative information on interactions among chemicals in mixtures is rarely available. In the absence of information on the mixture, risk is sometimes based on the addition of the risks of the individual mixture components, unless there is information indicating that the interaction is other than additive in nature. However, this practice is only appropriate if the CoPC in question have similar modes of action and similar toxic endpoints in the human body. There is uncertainty associated with any of the above approaches in that risk may be overestimated or underestimated.

In this risk assessment, the CoPC-specific HQs and ILCRs for a receptor have been summed within each exposure scenario if the target organ and toxicologic endpoint were the same (as shown in Tables 5-11 and 5-13).

The addivity of risks for threshold and non-threshold CoPCs are shown in Tables 5-14 and 5-15, respectively.

As shown in Table 5-14, there may be a risk to the First Nations toddler resulting from the potential additive risk of boron and methyl mercury. These two CoPC have both been shown to have developmental effects and/or decreased fetal weight. As previously discussed in Section 5.5.1, the methyl mercury on its own was predicted to pose a risk to First Nations toddlers, primarily due to the very conservative fish ingestion rates and the assumption that all the fish consumed by the toddler is caught from the one hypothetical lake.

As shown in Table 5-15, when all metal inhalation cancer risk endpoints (lung cancer) are summed the result is a Incremental Lifetime Cancer Risk of slightly in excess of 1E-06 at 1.08E-6. Given that the regulatory acceptable risk is set at 1E-06, this suggests that even if these compounds truly had additive impacts on the body, they would only be marginally above this level.

Women in Canada are estimated to have a lifetime probability of getting some form of cancer of approximately 38%, while for men it is 42% (Canadian Cancer Society, 2007). This represents a probability of 0.4 (4E-1). Given that the additive probability risk estimate for all lung cancer endpoints was 1.08E-06, the addition to the cancer background in Canada would reflect 0.400001. This is not meant to dismiss this estimated risk, rather to put it into context. This is also an area of concern that should be further evaluated in the site-specific risk assessment.

Table 5-14 Additivity of Risks – Threshold (Non-carcinogenic)

	Resident			Subsistence Farmer			First Nations			Commercial	
	Infant	Toddler	Composite	Infant	Toddler	Composite	Infant	Toddler	Composite	Toddler	Adult
Decreased body weight/organ weight *(Naphthalene, tetrachloroethylene, nickel)*	1.52E-06	5.29E-06	1.17E-06	1.52E-06	9.43E-06	1.87E-06	1.53E-06	2.14E-05	5.47E-06	3.04E-06	1.43E-06
Developmental effects *(Boron, methyl mercury)*	2.77E-06	2.96E-02	7.84E-03	2.77E-06	9.12E-02	2.41E-02	2.77E-06	**7.58E-01**	1.99E-01	4.17E-06	1.80E-06
Liver/kidney - weight increase *(1,2,4-Trichlorobenzene, 1,2,4,5-Tetrachlorobenzene, hexachlorobenzene)*	6.60E-06	5.88E-05	1.54E-05	1.77E-05	1.78E-04	4.61E-05	1.23E-04	1.38E-03	3.61E-04	3.57E-06	1.86E-06
Benchmark = 0.2 or 2E-01 **Bold** = Additive risks exceed the benchmark											

Table 5-15 Additivity of Risks – Non-Threshold (Carcinogenic)

	Resident	Subsistence Farmer	First Nations	Commercial
	Composite	Composite	Composite	Composite
Leukemia *(Benzene, 2,4,6-Trichlorophenol)*	2.57E-09	2.91E-09	4.50E-09	2.37E-09
Liver - angiosarcoma, carcinoma, neoplastic nodules *(Dichloromethane, vinyl chloride, hexachlorobenzene, pentachlorophenol*	3.01E-08	1.09E-07	6.99E-07	3.88E-09
Lung - cancer *(Arsenic, beryllium, cadmium, total chromium)*	**1.07E-06**	**1.07E-06**	**1.08E-06**	7.35E-07
Benchmark = 0.000001 or 1.00E-06 **Bold** = Additive risks exceed the benchmark				

5.6.3.2 Sensitive Populations

A susceptible population will exhibit a different or enhanced response to a CoPC than will most persons exposed to the same level of the contaminant in the environment. Reasons may include genetic makeup, age (e.g., children), health and nutritional status, and exposure to other toxic substances (such as cigarette smoke) (ATSDR, 2002). The non-cancer TRVs used in this risk assessment are estimates of a continuous exposure to the human population, including sensitive subgroups, that is likely to be without appreciable risk of adverse non-cancer effects during a lifetime. Toxicity doses and cancer slope factors used in the assessment have accounted for sensitive populations by applying uncertainty factors. Specifically, an uncertainty factor of 10 has typically been applied to account for intraspecies variations (i.e., susceptible populations).

Most air quality objectives are based on epidemiological studies of hospital reports (i.e., total population including sensitive subpopulations) while others are based on studies on asthmatics, which were generally considered to the subpopulation that is most susceptible to the respiratory effects of combustion gases and particles.

5.7 Summary of the Human Health Risk Assessment

It was determined that for the Commercial Worker / Day Care scenario and the Resident scenario that these receptors could in fact exist at the maximum point of impingement of emissions from the theoretical, generic EFW facility. This was the case for all three Operational Scenarios, which included a facility capable of processing up to 400,000 t/y of municipal solid waste.

For the First Nations scenario it was determined that methyl mercury modelled for fish consumption could potentially pose a potential risk if these receptors lived at the MPOI. However, given the conservative nature of the assumed fish consumption rates and the theoretical watershed modelled these issues could likely be further examined in a site-specific risk assessment.

In addition, for the subsistence Farm and First Nations receptor scenarios, it was conservatively estimated that if dioxins and furans were emitted at the A-7 Guideline value it could potentially result in an undue risk to infants being exclusively breast fed. When current-day emissions of dioxins and furans from the KMS Peel facility were modelled it was determined that the risk levels fell to below the applicable benchmark for the Subsistence Farm infant, but not however for the First Nations infant. This suggests that any EFW facility being considered for the Durham-York Regions should achieve dioxin and furan emission standards below the MOE A-7 Guideline and that particular attention needs to be paid to this issue.

6.0 ECOLOGICAL RISK ASSESSMENT

The ecological risk assessment (ERA) process evaluates the likelihood that adverse chronic ecological effects may occur, or are occurring, as a result of exposure to one or more stressors (USEPA 1992). For the purpose of this generic risk assessment, adverse ecological effects refer to toxicologically induced changes in wildlife health as a result of exposure to chemical stressors released into the environment from the EFW thermal treatment facility.

The ERA can be used to help classify potential ecological impacts of EFW facility activities by identifying contaminants of potential concern (CoPC), the likely pathways leading to wildlife exposure, and the possible population effects of such exposure. Considering this pro-active approach, results of the ERA will be used to determine if the proposed EFW facility is potentially environmentally acceptable. Furthermore, results of the ERA can be used to guide monitoring and mitigation programs, and guide the site-specific risk assessment priorities.

6.1 ERA Assessment Boundaries and Scenarios

This ERA evaluates the potential for adverse environmental effects that may occur as a result of exposure to Project emissions over the entire projected lifetime of the facility (35 years). Existing concentrations of chemicals in the environment were not evaluated; rather, this ERA focused on the additive risk (in addition to baseline) which may be introduced to the environment due to emissions from the EFW facility.

Three proposed Project operating scenarios were evaluated in this ERA, defined by the number of process units in operation:

Operating Scenario 1: 3 process units running at full capacity - 400,000 t/y

Operating Scenario 2: 2 process units running at full capacity - 266,666 t/y

Operating Scenario 3: 1 process units running at full capacity - 133,333 t/y

Potential environmental effects were evaluated at the maximum point of impingement (MPOI) surrounding the proposed facility. Based on results generated through air emission modeling, it was determined that this zone would most likely contain areas where environmental effects would be observed. The proposed location for this Project is situated in suburban and rural lands, and the vital components of these respective ecosystems are the focus of this ERA.

6.2 Ecological Risk Assessment Framework

The ERA process follows a widely recognized framework (Figure 6-1) that progresses from a qualitative initial phase (problem formulation), through exposure and toxicity (effects) analysis, and culminates in a quantitative risk characterization. The risk assessment methodology for this ERA is based on the following provincial and federal guidance documents:

- A Framework for Ecological Risk Assessment (General Guidance) (CCME 1996);
- Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration (MOE, 1999); and
- US EPA Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities (USEPA 1999).

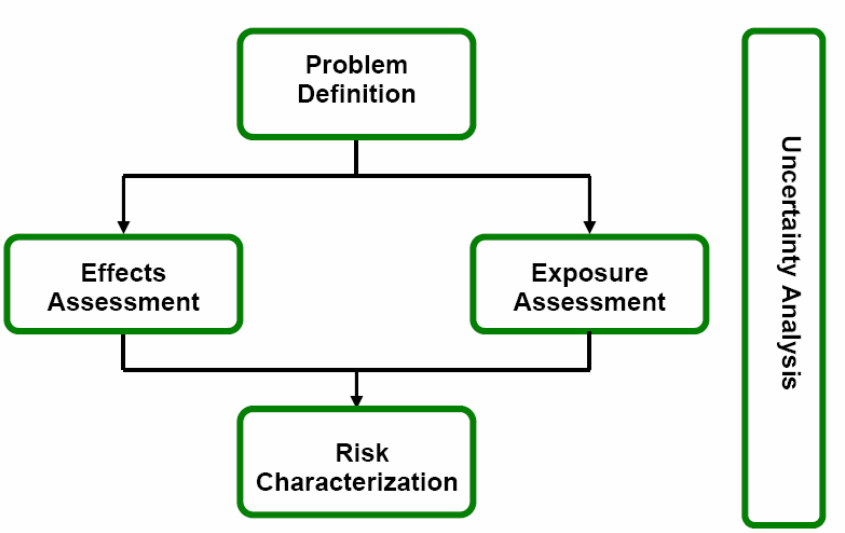


Figure 6-1 Ecological Risk Assessment Framework

6.3 Problem Formulation

The objective of the problem formulation stage of this ERA is to develop a focused understanding of how CoPCs will likely be released into the environment as a result of the Project and how they might affect the health of ecological receptors living at the MPOI. Problem formulation provides an early identification of key factors to be considered throughout the ERA, which in turn can be used to produce a more scientifically sound risk assessment (US EPA 1992). The end result of the Problem Formulation step is a conceptual model, a pictorial representation that outlines the direct and indirect sources of contaminants and the media and pathways of exposure for receptors.

Three issues require addressing in order to finalize a conceptual model for the Project:

- Identification of CoPCs: a list of chemicals expected to be emitted by the Project (and having the potential to cause adverse environmental effects) was compiled on

the basis of emissions by similar existing projects and recommendations from regulatory authorities;

- Ecological receptor Identification: a representative set of wildlife species (e.g., mammals, birds), or groups of similar species (e.g. terrestrial plant community) that may be exposed to stressors within the study area are selected; and
- Exposure pathway determination: the potential routes by which ecological receptors could be exposed to stressors in the study area are identified.

6.3.1 Chemicals of Potential Concern

CoPCs are compounds which are expected to be released from the Project and to have the potential to impose adverse effects on ecological health if released in sufficient quantity and/or to sensitive ecosystems. The CoPCs evaluated in this ERA were selected based on emissions data from similar facilities and guidance from the MOE (1999, 2004). Combustion gases were excluded as CoPCs since the inhalation pathway was not considered for this ERA. A list of the CoPCs evaluated in this ERA is presented in Table 6-1.

Table 6-1 List of Chemicals of Potential Concern Evaluated in the Ecological Risk Assessment

Metals	Chlorinated Monocyclic Aromatics	Chlorinated Polycyclic Aromatics	Polycyclic Aromatic Hydrocarbons	Volatile Organic Compounds
Antimony Arsenic✓ Barium Beryllium Boron Cadmium✓+ Chromium ✓ Cobalt Lead✓+ Mercury✓+ Nickel Silver Vanadium Zinc	1,2-Dichlorobenzene 1,2,4-Trichlorobenzene 1,2,4,5-Tetrachlorobenzene Pentachlorobenzene Hexachlorobenzene 2,4-Dichlorophenol 2,4,6-Trichlorophenol 2,3,4,6-Tetrachlorophenol Pentachlorophenol	PCBs 2,3,7,8-TCDD TEQ✓+ (dioxin/furan)	*Benzo(a)pyrene group* Benzo(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Benzo(g,h,i)perylene Benzo(k)fluoranthene Chrysene Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Anthracene Napthalene Phenanthrene	Benzene✓ Chloroform Dichloromethane Formaldehyde Tetrachloroethylene Vinyl chloride✓

Notes:

Chemical list derived from Cantox Report for Human Health Risk Assessment for the Proposed Expansion of the KMS Peel, Inc. Brampton, Energy-From-Waste Facility (2000)

✓ chemicals also reviewed by MOE in Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration (1999)

\+ chemical also included in GUIDELINE A-7 Combustion and Air Pollution Requirements for New Municipal Waste Incinerators (MOE 2004)

6.3.2 Identification of Valued Environmental Components

The objective of this step is to select a representative set of ecological receptors that may be exposed to CoPCs from the Project. Although the Project is situated in a partially developed suburban / rural area, there are numerous wildlife species which inhabit the surrounding lands and could potentially experience adverse health effects as a result of exposure to Project related CoPCs. An exhaustive assessment of all wildlife species is neither practical nor necessary for this ERA. Instead, this ERA evaluates the potential adverse effects of the Project imposed on a carefully selected subset of wildlife receptors living in or near the Project area.

Selection of these Valued Environmental Components (VECs) requires consideration:

- receptors that are indigenous at or near the site;
- any species of cultural or economic significance; and
- any species designated as threatened or endangered that lives in close proximity to the Project.

Selection of VECs must also ensure each applicable habitat and trophic level in the area is represented, and where possible, include species that are knowingly most sensitive to Project-related emissions. Therefore, analysis of the VECs in this ERA (outlined below) should provide an adequate representation of the potential for adverse health effects on all wildlife receptors in the Project area.

The following species were identified as VECs and evaluated in the ERA:

- Masked Shrew (*Sorex cinereus*);
- Meadow Vole (*Microtus pennsylvanicus*);
- Mink (*Mustela vison);*
- Common Muskrat (*Ondatra zibethicus*);
- Red Fox (*Vulpes vulpes*);
- American Robin (*Turdus migratorius*);
- Belted Kingfisher (*Ceryle alcyon);*
- Mallard (*Anas platyrhynchos*); and
- Red Tailed Hawk (*Buteo jamaicensis*).

For some VECs, it is more appropriate to assess potential risk at community level (i.e. all terrestrial plants living in the Project area), rather than individual species. The community-based VECs identified for this ERA are:

- fish;
- terrestrial plants;
- benthic invertebrates; and
- soil invertebrates.

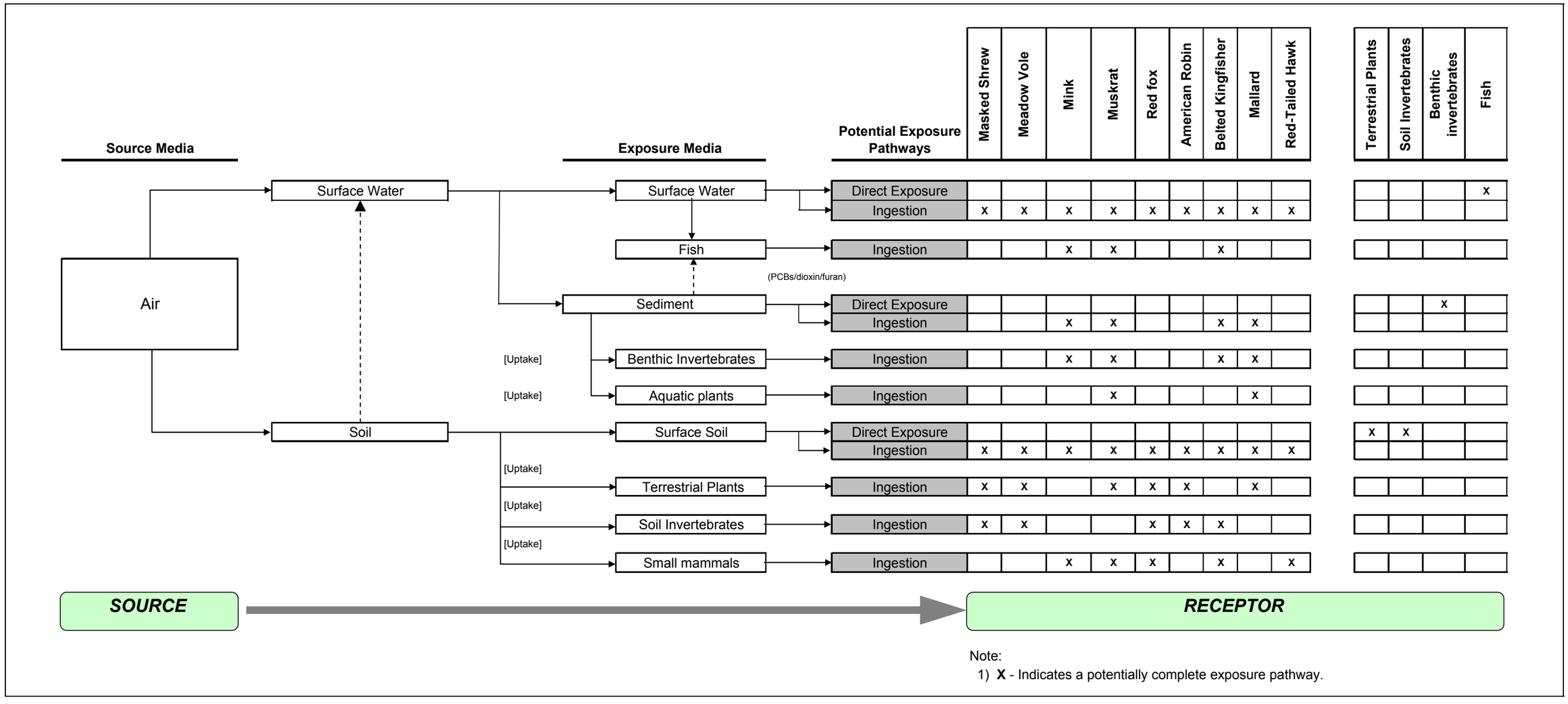
A description of the ecology and life history of each VEC is provided in **Appendix H**.

6.3.3 Conceptual Model

The conceptual model designed for this ERA is presented in Figure 6-2. The exposure pathways are designated by arrows leading from one compartment to another compartment and boxes with an “X” denote relevance to a particular receptor.

Mammalian, avian and aquatic VECs were assumed to spend 100% of their time in the Project area meaning that exposure CoPCs from emission sources will occur throughout the life of the receptor. It is possible that VECs may move in and out of the Project area, thereby reducing their exposure, but this conservative assumption is meant to be a protective measure.

Figure 6-2 Ecological Risk Assessment Conceptual Model



Note:
1) **X** - Indicates a potentially complete exposure pathway.

6.4 Exposure Assessment

6.4.1 Exposure Pathways

An exposure pathway identifies the potential routes of VEC exposure to CoPCs. In Table 6-3 a summary of potential exposure media for ecological receptors and a rationale for the inclusion or exclusion from this ERA, is presented. For terrestrial plants, soil invertebrates and terrestrial wildlife receptors, including mammals and birds, exposure to CoPCs may occur through the following routes:

- dermal contact with soils;
- inhalation;
- ingestion of soil and water (i.e., as a result of feeding or grooming); and
- ingestion of plants or prey species that have accumulated chemicals from the soil.

For aquatic organisms (e.g., fish and aquatic (benthic) invertebrates) exposure to CoPCs may occur through the following routes:

- ingestion of sediment (e.g., fish may ingest sediment contained within prey species);
- ingestion of aquatic prey;
- contact with sediment (i.e., in the case of aquatic invertebrates and aquatic plants);
- ingestion/contact with surface water.

Table 6-2 Rationale for Exposure Pathways Evaluated for Terrestrial Receptors

Exposure Pathway	Inclusion in ERA	Rationale
Soil (and Sediment) Ingestion	✓	During the operational phase of the Project, airborne emissions will deposit directly onto the soil. Wildlife species consume soil during foraging, preening and grooming. Therefore, this exposure pathway was evaluated in the ERA for receptors.
Ingestion of Terrestrial Vegetation, Soil Invertebrates and Small Mammal Prey	✓	During the operational phase of the Project, gaseous and fugitive dust emissions may deposit directly onto plant surfaces and soils. Chemicals may subsequently be taken up into plants that are food sources for wildlife. Consumption of plants could expose herbivorous wildlife to chemicals. Therefore, this exposure pathway was evaluated for herbivorous wildlife receptors. Carnivorous and omnivorous animals have the potential to be exposed to CoPCs via ingestion of prey that have themselves been exposed. For this reason, ingestion of prey was evaluated in the ERA for these receptors.
Dermal Contact	×	Although wildlife may be exposed by direct contact with surface water and soil, they would likely receive insignificant doses through this route relative to other routes, such as direct ingestion of water and soil. Therefore, the dermal exposure was not evaluated as a potential pathway of exposure in the ERA.
Inhalation	×	Wildlife may be exposed to CoPCs through inhalation of airborne emissions from the Project. Due to the conservative approach used in the HHRA for the evaluation of health risks from this pathway, inhalation was not explicitly considered in the ERA. Alternatively, it was concluded that if no unacceptable risks were estimated for human health, ecological receptors were assumed to be protected also.
Water Ingestion/Contact	✓	CoPCs may be present in the surface water as a result of deposition from air emissions and/or surface runoff. Drinking from these waterbodies may become a significant exposure pathway for wildlife.
Ingestion of Aquatic Invertebrates and Fish	✓	During the operational phase of the Project, CoPCs from the Project could enter surface waterbodies and be taken up by fish, invertebrates, and aquatic vegetation. Wildlife (e.g., Common Muskrat) may then ingest “contaminated” prey or plants and become subsequently exposed to CoPCs.

6.4.2 Exposure Point Concentration Determination

In order to evaluate the level of exposure to each of the VECs, it was necessary to first estimate the concentrations of each CoPC in various environmental media and biota (e.g. fish tissue). Using modeled air emission and deposition rates for each CoPC, environmental fate modeling was used to estimate exposure point concentrations (EPCs) in various environmental media (water, soil, and sediment). Exposure point concentrations used in the ERA were generated by the fate and transport model as described in Section 4. Where needed, additional ERA specific EPCs for each CoPC in various biota could then be calculated through the utilization of compound-specific uptake factors (UP), which describe the relationship between a specified chemical in a given media to various types of biota (e.g., the uptake of benzene in soil by terrestrial plants).

The generalized equation used to calculate a CoPC concentration in receptors, (such as soil invertebrates) from a soil concentration is as follows:

$$EPC_i = EPC_{soil} * UP_i$$

where:

EPC_i	exposure point concentration in biological compartment *i* (mg/kg wet weight);
EPC_{soil}	exposure point concentration in soil (mg/kg dry weight); and
UP_i	Uptake Factor from soil to target biotic tissue *i* (dimensionless).

An analogous equation is used to calculate EPCs (on a mg/kg wet tissue basis) using water (mg/L) or sediment (mg/kg dry sediment) EPC calculations. For this ERA, EPCs were calculated for soil, water, sediment, terrestrial plants, and fish following the methods described in Section 4. EPCs for aquatic plants, terrestrial and aquatic invertebrates, and small mammals (prey species) are specific to the ERA, and a description of the uptake factors involved in their calculations are provided in the ERA **Appendix H**.

6.4.3 Calculation of the Ecological Average Daily Dose

In order to conduct a risk assessment, it is necessary to estimate, the amount of a CoPC a VEC might be exposed to on a mg/kg/day basis (referred to as the average daily dose, or ADD). For each VEC, the ADD was calculated for all CoPC by considering the intake from each applicable exposure pathway. The generalized form for ADD is as follows:

$$ADD_j = IF_j \cdot AF_j \cdot EPC_j$$

For exposure pathway 'j', where:

IF_j	Intake Factor (kg contaminated media / kg body weight · day),
AF_j	Absorption Factor (default value of 1), and
EPC_j	Exposure Point Concentration (EPC; mg contaminant / kg media).

The Intake Factor is not specific to each CoPC, but is dependent on exposure media. It is calculated for each exposure pathway using the media-specific ingestion rate (IR), the fraction of the total

ingestion rate from the site (f_{site}, assumed to equal 100% for this ERA) and the receptor's body weight (BW) as follows:

$$IF_j = (IR_j \cdot f_{site}) / BW$$

For details related to the body weight, diet composition (plant, insect, prey), water, and soil ingestion rates for each of the receptors evaluated in the ERA, refer to **Appendix H**.

6.5 Toxicity Assessment

The objective of the toxicity assessment is to identify the potential adverse health effects associated with each CoPC as a consequence of chronic oral exposure. Using this knowledge, a Toxicity Reference Value (TRV) is generated, which defines the chronic daily dose of a CoPC below which unacceptable adverse effects are not expected to occur. TRVs are specific to each CoPC and ecological receptor evaluated in the ERA.

TRVs used in this risk assessment were determined from studies where endpoints were derived from the administered dose, rather than the absorbed dose (i.e., absorbed / retained concentration of contaminant in the organ or body). This is a conservative approach because compounds are often administered in a more available form than would be found in the environment.

6.5.1 Toxicity Reference Values

TRVs are generally based on dose-response studies conducted with laboratory animals (e.g., mammals: mice, rats, rabbits; birds: quail, chicken, ducks) where the lowest observed adverse effects levels (LOAELs) are quantified (or no observed adverse effects level (NOAEL), when LOAELs are not available). Generally, LOAELs are based on long-term growth or survival, or sub-lethal reproductive effects determined from chronic exposure studies. As such, these endpoints are relevant to the maintenance of local or regional wildlife populations. TRVs were established using toxicological data from a variety of sources, including but not restricted to:

- Oak Ridge National Laboratory Toxicity Benchmarks for Wildlife
- U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- *Canadian Environmental Protection Act*, Priority Substance List Assessment Reports
- Primary Scientific Literature

TRVs and references for each established LOAEL (or NOAEL) for laboratory mammals and birds are provided in **Appendix H**.

6.5.2 Body Weight Scaling

As mentioned above, toxicological testing is generally only conducted on a select few mammalian and avian laboratory species. To represent mammalian toxicity, the mouse or rat is the preferred test species, while chickens, mallards and northern bobwhite quail are the typical species for avian testing. Since toxicity may not scale with body weight at a 1:1 ratio, scaling factors were applied to adjust TRVs for the body weights of chosen VECs.

Mammalian Body Weight Scaling

If toxicity data for the specific representative mammalian receptors were not available, a body-size scaling factor (Sample and Arenal, 1999) was used for extrapolation of available data between species.

The body-size scaling factor is calculated as:

$$\text{Mammal Body Weight SF} = (BWt / BWr)^{0.06}$$

where:

SF	scaling factor
BWt	mean body weight for test species
BWr	mean body weight for receptor species

Avian Body Weight Scaling

If toxicity data for the specific representative avian receptors was not available, a body-size scaling factor (Sample and Arenal 1999) was used for extrapolation of available data between species.

The body-size scaling factor is calculated as:

$$\text{Bird Body Weight SF} = (BWt / BWr)^{-0.20}$$

where:

SF	scaling factor
BWt	mean body weight for test species
BWr	mean body weight for receptor species

6.5.3 Oral TRVs for Valued Environmental Components

For CoPCs where LOAELs or NOAELs were not available, sub-chronic or acute toxicity measures such as median lethal dose (LD_{50}) were obtained and modified using Uncertainty Factors (UFs) to convert these less than chronic values to surrogate chronic values (UFs described below). It should be noted that TRVs derived from laboratory mammals and birds are generally considered protective of wildlife mammalian and avian species, respectively.

In many cases for avian (bird) species, specific toxicity reference values for avian species were not available. Therefore, potential risk from exposure to these chemicals of potential concern can only be evaluated qualitatively, meaning that if there is no risk to mammals from these chemicals in the absence of bird toxicology one must assume this would be protective of birds in the area.

In human toxicology it has always been standard practice to take toxicity data from laboratory animals (such as mice) and to apply various levels of UF in order to make the data safely applicable to humans. These safety factors have typically been factors of 10 to go from human to animal, with additional factors of 10 applied depending upon what other necessary assumptions are made (usually five more levels of uncertainty). Therefore, uncertainty factors in human toxicology can range up to 10^5. Recent documentation from the USEPA (2002) recognizes that uncertainty factors can "build" upon each other

to unreasonable proportions, and suggests that uncertainty values should range between 1 and 10, but prefers values of 1, 3 or 10 for ERAs.

In this ERA where required UFs were implemented including:

- a division by 5 to convert an acute or subchronic dose to chronic dose;
- a division by 6 to convert an LD_{50} value to a LOAEL value

These UFs are cumulative, so an acute LD_{50} would be converted to a chronic LOAEL by dividing by 30, the product of the two UFs required for the endpoint conversion (e.g., 6 * 5).

6.5.3.1 Phytoxicity Assessment

Individual plant species were not evaluated in the ERA. Rather, toxicity assessments were based on phytotoxicity benchmarks that are considered protective of all plant life for each CoPC. In addition to the CoPCs identified for this ERA, the potential risk to terrestrial plants from exposure to sulphur oxides emitted from the facility were evaluated. Sulphur oxides are considered highly phytotoxic combustion gases, and international air quality guidelines for the protection of terrestrial vegetation have been established for them by the WHO (2000) (represented as sulphur dioxide). Phytotoxicity benchmarks used in this ERA are found in **Appendix H**.

6.5.3.2 Freshwater Receptor Toxicity Assessment

Individual freshwater species were not evaluated in the ERA. Rather, toxicity assessments were based on freshwater quality screening benchmarks that are considered protective of all aquatic life for each CoPC. **Appendix H** outlines the freshwater quality benchmarks used in this ERA.

6.5.3.3 Sediment Toxicity Assessment

Again individual freshwater sediment species were not evaluated in the ERA. Rather, toxicity assessments were based on freshwater sediment quality screening benchmarks that are considered protective of all aquatic sediment life for each CoPC. **Appendix H** outlines the freshwater sediment quality screening benchmarks used in this ERA. Individual PAHs were not assessed for sediment toxicity; rather the toxicity of the PAH mixture was assessed.

6.5.3.4 Soil Invertebrate Toxicity Assessment

Soil invertebrates were not evaluated for individual species in the ERA. Rather, toxicity assessments were based on soil screening benchmarks that are considered protective of all terrestrial invertebrate life for each CoPC. The soil screening benchmarks used in this ERA are provided in **Appendix H**.

6.6 Ecological Risk Characterization

Risk characterization evaluates the evidence linking site CoPCs with adverse ecological effects by combining information from the exposure and toxicity assessments. To characterize risk, all chemical and biological data relating to the site must be evaluated. In particular, fate and transport studies can provide evidence of links between site contaminants and observed or predicted environmental effects. Hazard Quotients (HQs) were calculated as the ratio of the predicted exposure to the toxicity reference value:

$$HQ = \frac{\text{average daily dose (ADD)}}{\text{toxicity reference value (TRV)}}$$

HQs were calculated for each receptor at the EFW facility for all three operating scenarios, taking into consideration all applicable exposure pathways. For example, the HQ for the meadow vole was calculated as the sum of HQs for each relevant exposure pathway:

$$HQ_{\text{meadow vole}} = HQ_{\text{vegetation ingestion}} + HQ_{\text{soil ingestion}} + HQ_{\text{invertebrate ingestion}} + HQ_{\text{water ingestion}}$$

A HQ less than 1.0 indicates that the exposure concentration is less than the threshold of toxicity and there is a low probability that adverse environmental effects might occur. However, given that background concentrations in environmental media were not taken into consideration a more conservative HQ benchmark of 0.2 was employed. Although this is rarely the case in ecological risk assessment, this more conservative benchmark was warranted given that cumulative effects were not assessed.

Regardless, it is likely that no adverse environmental effect would occur at HQs less than 0.2. Saying this, a HQ value of greater 0.2 does not automatically indicate that there is an unacceptable level of risk. In these cases, values greater than 0.2 indicate that there is a possibility that of adverse ecological effects could occur if background concentrations were taken into consideration and dictate a need for more careful review of both predicted exposure levels and exposure limit derivations. As a result, HQ values greater than 0.2 should be examined carefully, and further, more focused investigations may be required to reduce conservatism and provide a more realistic assessment of the actual level of risk. If it is ultimately determined that the HQ value is indeed greater than 1.0, then site management or mitigation activities may be appropriate in order to reduce risks to ecological receptors.

6.6.1 Additivity of Hazard Quotients

For certain chemical groupings, a more appropriate characterization of risk is achieved by summing the ecological HQs for each compound. This approach is warranted when evaluating a group of CoPCs that are known to cause effects on the same target organ, and by the same mode of action. For these reasons, ecological HQs were summed in this ERA for PAHs to derive a single conservative HQ index that is representative of that group. Risk from exposure to all other CoPCs were evaluated independently.

6.6.2 Ecological Assessment of Risk

HQ values for all VECs based on Scenario 1 (3 process units, 400,000 t/y) exposure conditions are presented in Table 6-3 and Table 6-4. The highest HQ for a terrestrial VEC was approx. 0.17, estimated for the Belted Kingfisher from exposure to methyl mercury. HQs for all remaining individual terrestrial and avian VECs were much lower than 0.2 (HQs did not exceed 0.08), indicating that the potential risk to these VECs exposed to CoPCs from the facility is not expected.

For community based VECs the highest HQ was 0.80 for aquatic organism exposure to dioxins (2,3,7,8 – TCDD equivalent). HQs for all other community based VECs were much lower than 0.2 (HQs did not exceed 0.05).

The EPCs for dioxins were calculated by assuming an emission rate equal to the MOE (2004) Guideline A-7 emission concentration limit. As a comparison, HQs were also generated using current emission rates from the KMS Peel EFW facility (provided in Section 5). KMS Peel emission rates for dioxins were considerably lower than the MOE (2004) guideline A-7 emission concentration limit. Consequently, the HQ for aquatic organism exposure to dioxins decreased to 0.1 (from approx. 0.8, for scenario 1 exposure), when using the KMS Peel emission rates.

For both instances in which an elevated HQ was determined, the primary source of exposure was from CoPC concentrations in water (indirectly, for the Belted Kingfisher as a result of fish consumption). Similar to the findings of the human health risk assessment, this may be a function of the theoretical watershed that was modeled as part of this ERA. Therefore these issues should be further considered in a site-specific risk assessment.

Scenario 1 is comprised of three process units in operation, and is therefore the scenario which predicts the highest potential exposure to ecological receptors. It follows then, that the absence of (unacceptable) risk predictions under scenario 1 conditions, is also an indication of no unacceptable risk under scenario 2 and 3 (2 and 1 process units, respectively).

A detailed summary of HQs for all VECs, and each operating scenario, is provided in **Appendix H**.

Table 6-3 Hazard Quotients for Terrestrial VECs from Scenario 1 Exposure Conditions

CoPC	Masked Shrew	Meadow Vole	Mink	Muskrat	Red Fox	American Robin	Belted Kingfisher	Mallard	Red-Tailed Hawk
BTEX									
Benzene	2.57E-09	2.01E-08	9.21E-09	6.50E-09	2.66E-09	--	--	--	--
PAHs									
Anthracene	9.11E-11	8.67E-11	1.59E-11	3.14E-10	1.36E-11	4.44E-08	9.82E-09	3.93E-08	7.61E-10
Benzo(a)anthracene	3.85E-10	3.94E-10	2.19E-09	2.69E-08	6.29E-11	--	--	--	--
Benzo(a)pyrene	5.79E-09	5.06E-09	3.85E-08	4.61E-07	1.82E-09	--	--	--	--
Benzo(b)fluoranthene	6.58E-10	3.56E-10	6.40E-09	5.86E-08	5.82E-11	--	--	--	--
Benzo(g,h,i)perylene	7.39E-06	1.44E-04	2.99E-05	9.98E-06	4.58E-05	--	--	--	--
Benzo(k)fluoranthene	5.89E-10	8.73E-10	4.49E-09	4.46E-08	1.30E-10	--	--	--	--
Chrysene	8.03E-10	5.40E-10	1.85E-09	2.45E-08	8.69E-11	--	--	--	--
Dibenz(a,h)anthracene	9.91E-09	1.40E-07	4.54E-08	1.44E-07	4.20E-08	--	--	--	--
Indeno(1,2,3-cd)pyrene	9.61E-11	1.94E-10	2.51E-10	5.96E-09	5.25E-11	--	--	--	--
Naphthalene[1]	2.40E-09	2.87E-08	7.45E-10	7.84E-09	3.24E-09	--	--	--	--
Phenanthrene[1]	3.17E-09	3.12E-09	2.17E-09	4.80E-08	5.58E-10	6.10E-08	4.57E-08	2.29E-07	1.29E-09
Total PAHs	7.41E-06	1.44E-04	3.00E-05	1.08E-05	4.58E-05	1.05E-07	5.55E-08	2.69E-07	2.05E-09
PCBs									
Aroclor 1254 (Total PCBs)	1.74E-06	9.58E-07	1.66E-04	3.55E-05	3.15E-07	1.05E-06	2.31E-04	9.84E-06	8.31E-08
Dioxins and Furans									
2,3,7,8-TCDD Equivalent	5.83E-04	5.24E-05	5.92E-04	2.37E-03	1.25E-04	6.26E-05	1.56E-04	1.56E-04	7.99E-06
2,3,7,8-TCDD Equivalent - Peel	5.19E-05	6.39E-06	7.25E-05	3.21E-04	1.27E-05	5.69E-06	1.96E-05	2.09E-05	8.26E-07
VOCs									
Chloroform	7.94E-11	6.23E-10	2.33E-10	5.86E-09	8.21E-11	--	--	--	--
Dichloromethane	5.76E-08	6.33E-07	4.64E-08	6.86E-08	6.90E-08	--	--	--	--
Formaldehyde	8.16E-06	8.97E-05	2.96E-06	7.14E-06	8.50E-06	6.29E-04	2.41E-05	5.85E-05	8.73E-07
Tetrachloroethylene	8.43E-09	1.12E-07	1.39E-07	2.60E-08	1.21E-08	--	--	--	--
Vinyl Chloride	2.48E-09	6.79E-09	4.84E-09	1.12E-07	1.85E-09	--	--	--	--
Chlorinated Monocyclic Aromatics									
1,2-Dichlorobenzene[1]	1.33E-10	2.01E-09	3.70E-10	1.64E-10	2.04E-10	--	--	--	--
1,2,4-Trichlorobenzene	4.01E-09	2.48E-08	2.54E-09	1.96E-09	2.70E-09	--	--	--	--
1,2,4,5-Tetrachlorobenzene	5.04E-08	1.62E-07	1.75E-06	1.94E-07	2.01E-08	--	--	--	--
Pentachlorobenzene	2.97E-07	4.24E-08	2.38E-06	9.00E-07	1.11E-08	--	--	--	--
Hexachlorobenzene	5.52E-07	3.00E-07	1.76E-05	3.35E-06	4.71E-08	1.40E-07	6.64E-06	2.45E-07	1.04E-09
2,4,-Dichlorophenol	3.93E-07	5.59E-06	9.25E-07	6.93E-07	5.83E-07	--	--	--	--
2,4,6-Trichlorophenol	5.12E-09	9.08E-08	1.37E-08	6.95E-09	9.53E-09	--	--	--	--
2,3,4,6-Tetrachlorophenol	8.39E-09	2.95E-08	8.84E-07	4.54E-08	5.45E-09	--	--	--	--
Pentachlorophenol	7.74E-02	7.89E-04	4.10E-03	7.75E-05	6.10E-03	2.08E-02	1.35E-03	2.15E-07	8.48E-04
Inorganics									
Antimony	1.73E-06	2.43E-06	8.28E-07	1.95E-07	2.76E-07	--	--	--	--
Arsenic	5.25E-06	4.33E-07	4.31E-07	4.08E-08	1.85E-07	7.11E-07	1.79E-07	9.89E-09	3.61E-09
Barium[1]	5.40E-07	9.30E-07	5.61E-06	2.29E-07	2.37E-07	1.13E-07	9.48E-07	6.02E-08	6.57E-09
Beryllium	3.96E-09	7.72E-09	1.78E-08	9.53E-10	1.84E-08	--	--	--	--
Boron	4.82E-07	2.87E-06	1.14E-07	2.32E-07	3.62E-07	8.71E-06	1.55E-07	8.12E-07	1.00E-07
Cadmium	1.23E-04	3.32E-06	7.56E-05	1.78E-06	8.15E-05	9.01E-05	2.86E-05	9.33E-07	3.98E-05
Total Chromium	1.53E-07	1.98E-07	9.94E-07	3.19E-08	1.08E-06	6.42E-07	5.22E-07	5.10E-08	9.74E-07
Cobalt	4.19E-09	6.89E-09	3.62E-10	4.50E-10	7.46E-10	4.11E-08	5.70E-09	3.56E-09	3.31E-10
Lead	1.82E-05	2.18E-06	8.37E-05	1.72E-06	9.37E-05	1.43E-04	1.69E-04	6.28E-06	4.57E-04
Inorganic Mercury[1]	2.06E-05	3.36E-05	4.53E-05	4.55E-04	4.31E-06	1.04E-04	1.50E-04	3.45E-04	8.72E-07
Methyl Mercury	7.91E-05	5.48E-05	1.57E-02	4.57E-04	8.39E-06	6.90E-04	1.68E-01	4.33E-05	5.18E-06
Nickel	1.75E-07	4.09E-08	8.15E-06	1.55E-07	9.06E-06	2.43E-07	2.46E-06	1.43E-08	6.86E-06
Silver	2.49E-07	1.30E-07	1.59E-07	1.42E-08	1.84E-08	--	--	--	--
Vanadium[2]	1.71E-07	2.92E-07	1.03E-06	1.90E-07	4.34E-08	1.48E-07	6.29E-07	2.57E-06	4.02E-09
Zinc	2.65E-03	2.84E-05	8.39E-03	1.54E-04	9.41E-03	1.03E-02	9.43E-03	1.46E-06	2.59E-02

[1] HQ was determined using NOAEL based TRV for mammalian VEC

[2] HQ was determined using NOAEL based TRV for avian VEC

Table 6-4 Hazard Quotients for Community-Based VECs from Scenario 1 Exposure Conditions

CoPC	Phytotoxicity	Soil Invertebrates	Aquatic Life	Benthic Invertebrates
BTEX				
Benzene	4.45E-08	4.45E-08	2.90E-06	3.58E-05
PAHs				
Anthracene	9.21E-08	9.21E-08	4.07E-04	not calculated for individual PAH compounds
Benzo(a)anthracene	4.41E-08	4.41E-08	1.68E-04	
Benzo(a)pyrene	6.40E-07	6.40E-07	1.30E-04	
Benzo(b)fluoranthene	2.60E-07	2.60E-07	1.28E-04	
Benzo(g,h,i)perylene	5.21E-09	5.21E-09	5.82E-05	
Benzo(k)fluoranthene	2.21E-07	2.21E-07	1.02E-04	
Chrysene	3.15E-07	3.15E-07	1.36E-04	
Dibenz(a,h)anthracene	3.17E-07	3.17E-07	1.64E-05	
Indeno(1,2,3-cd)pyrene	1.00E-08	1.00E-08	9.50E-07	
Naphthalene	2.47E-08	2.47E-08	1.58E-05	
Phenanthrene	8.58E-08	8.58E-08	9.11E-05	
Total PAHS	**2.02E-06**	**2.02E-06**	**1.25E-03**	**6.98E-05**
PCBs				
Aroclor 1254 (Total PCBs)	1.59E-06	1.27E-05	2.63E-04	9.03E-04
Dioxins and Furans				
2,3,7,8-TCDD Equivalent	4.76E-05	4.76E-05	**7.97E-01**	1.49E-05
2,3,7,8-TCDD Equivalent - Peel	6.01E-06	6.01E-06	1.01E-01	1.88E-06
VOCs				
Chloroform	2.67E-09	2.67E-09	1.37E-05	4.26E-07
Dichloromethane	3.15E-09	3.15E-09	7.74E-05	8.21E-06
Formaldehyde	NA	1.10E-03	7.09E-04	6.09E-04
Tetrachloroethylene	6.00E-08	6.00E-08	2.68E-06	5.94E-06
Vinyl Chloride	5.11E-08	5.11E-08	2.60E-08	7.41E-07
Chlorinated Monocyclic Aromatics				
1,2-Dichlorobenzene	8.90E-10	8.90E-10	4.74E-06	1.71E-07
1,2,4-Trichlorobenzene	2.52E-09	2.52E-09	1.81E-07	6.18E-08
1,2,4,5-Tetrachlorobenzene	NA	1.00E-07	3.79E-06	2.75E-12
Pentachlorobenzene	NA	1.31E-07	5.35E-07	6.48E-04
Hexachlorobenzene	2.88E-06	2.88E-06	1.54E-02	7.37E-04
2,4,-Dichlorophenol	5.34E-08	5.34E-08	9.40E-03	1.35E-04
2,4,6-Trichlorophenol	3.71E-08	3.71E-08	2.52E-05	3.32E-05
2,3,4,6-Tetrachlorophenol	NA	2.08E-06	4.92E-04	4.27E-05
Pentachlorophenol	1.80E-07	2.90E-08	2.76E-03	NA
Inorganics				
Antimony	5.77E-06	9.62E-07	2.08E-06	4.69E-07
Arsenic	6.24E-07	1.87E-07	2.59E-07	6.63E-08
Barium	3.72E-07	5.64E-07	2.06E-06	5.15E-08
Beryllium	9.56E-08	5.98E-08	1.27E-07	4.51E-07
Boron	7.74E-04	1.94E-05	2.81E-03	NA
Cadmium	1.29E-05	2.96E-06	1.36E-03	1.22E-05
Total Chromium	2.79E-08	2.09E-07	1.16E-06	1.81E-08
Cobalt	2.49E-07	9.80E-08	2.57E-06	2.70E-08
Lead	8.11E-05	5.72E-06	1.28E-04	1.39E-05
Inorganic Mercury	1.56E-05	1.56E-05	1.49E-04	1.22E-02
Methyl Mercury	4.65E-07	4.65E-07	1.89E-04	1.58E-06
Nickel	1.10E-06	8.26E-07	4.92E-06	2.72E-07
Silver	2.41E-07	2.41E-07	4.74E-04	1.10E-06
Vanadium	3.30E-07	1.65E-07	6.85E-06	2.06E-06
Zinc	4.61E-05	1.15E-05	3.12E-05	7.37E-07
Combustion Gases				
Sulphur oxides (as SO_2) - 24 Hour	3.45E-02	NA	NA	NA
Sulphur oxides (as SO_2) - Annual	1.60E-02	NA	NA	NA

NA = Not Available

6.7 ERA Uncertainty Analysis

Uncertainty is an inherent in many aspects of predicting health risks to ecological receptors. The level of uncertainty is dependent upon the availability and quality of information, as well as the variability associated with many of the processes and factors being considered. When conducting risk assessments, it is standard practice to implement conservative assumptions (i.e., to make assumptions that are inherently biased towards safety) when uncertainty is encountered. This strategy generally results in an overestimation of actual risk, which helps ensure that the overall ERA conclusions will be protective of the health of ecological receptors. The following sections outline the main sources of uncertainty in this ERA.

6.7.1 Selection of VECs and VEC Characterization

The VECs evaluated in this ERA were carefully selected to include receptors that could be reasonably expected to be present on the site, and could collectively provided a representation of vital components of the food web at the site (i.e., omnivore, herbivore). These VECs are also known to be reasonably or conservatively representative of other species that may be present on the site and exposed to CoPCs. The use of site-specific receptors decreases uncertainty since local species are considered.

The use of VECs is intended to limit the number of ecological receptors to a reasonable number. The VECs selected are considered to be consistently present in the study areas and to be highly exposed to the CoPCs present at the site via relevant exposure pathways. Therefore, it is reasonable to assume that conclusions that are reached in respect of VECs can be generalized to other biota that might use the site.

For each VEC, the estimated exposure to CoPCs was heavily dependent on attributes such as water ingestion rates, food ingestion rates, dietary composition, soil ingestion rates, etc. These attributes were characterized through extensive reviews of the available scientific literature. Where VEC-specific values were unavailable, body weight based estimation was often utilized (i.e., estimation of food requirements using Nagy's (1987) equations).

For this ERA it has been assumed that each receptor organism spends its entire life cycle at the site (exposed to the EPC for each CoPC). It is therefore likely that the level of exposure has been overestimated for some species, particularly for more migratory VECs (i.e. Red-Tailed Hawk).

Amphibian and reptile species are sensitive to environmental stressors. However, due to the lack of toxicity information for the chemicals and chemical groups evaluated in this ERA, amphibians could not be evaluated as a receptor in this risk assessment. For the purposes of this assessment, amphibians were assumed to be adequately protected since all other aquatic VECs were deemed protected. A similar conclusion is extended to reptiles, which are considered protected due to a lack of unacceptable risk determined for any terrestrial VECs.

6.7.2 Receptor-Specific Toxicity Data

For most CoPCs and VECs, toxicity data are available in some form. However, due to a lack of available toxicity information, it is important to note that toxicity values were not necessarily specific to the VEC species, or to a reproductive or population-level endpoint. As a result, there is uncertainty associated with extrapolations that were used to translate, for example, subchronic to chronic toxicity

endpoints. The toxicity data represent an organism that is expected to be sensitive to the CoPC. The conversion factors that are used are scientifically based, and are applied in a manner that is consistent with regulatory guidance.

The preferred measure of toxicity for TRVs in this ERA is the chronic LOAEL. For certain CoPCs the only chronic endpoints available were NOAELs. In this situation, the NOAEL was used as the TRV (without the application of uncertainty factors). The decision not to apply uncertainty factors to translate a NOAEL to a LOAEL is a conservative measure to avoid overestimating the LOAEL (thus, underestimating potential risks). For mammalian VECs, NOAEL based TRVs were used for the following CoPCs: naphthalene, phenanthrene, 1,2-dichlorobenzene, barium, and inorganic mercury. For avian VECs, a NOAEL based TRV was used for vanadium.

The magnitude of uncertainty associated with the extrapolation of mammalian toxicity data for the purpose of generating TRVs for avian VECs was deemed unacceptable for this ERA. Consequently, potential health risks to avian VECs could not be assessed for several CoPCs (due to a lack of avian toxicity data).

6.7.3 Data Limitations

Various models were used to simulate air emissions and environmental fate of CoPCs for the purpose of generating EPCs. These modeling exercises were carried out in a way that is expected to result in conservative estimates that are likely to overstate the actual level of risk. For example, ecological risk was evaluated by conservatively assuming that deposition within the entire Project area occurs at the maximum point of impingement (POI). It is important to recognize that the modeled air emissions data is also inherently conservative. Therefore, the forecasted risks to ecological receptors that result from these data are also highly conservative.

However, site specific background concentrations of CoPCs in environmental media were not included in this assessment. Therefore, results should be viewed with caution and lead to the importance of collection of statistically representable concentrations of CoPCs at the ultimately selected preferred site.

6.7.4 Selection of Chemicals of Potential Concern

This ERA attempts to evaluate the potential for adverse ecological risks from a proposed facility. Consequently, an emission inventory was not available. Identifying the chemicals of potential concern for this Project relied on Ontario MOE guidance for incinerator emissions and emission inventories available for similar facilities. Using these sources, the chemicals of potential concern were identified by selecting those chemicals that could have the potential to exert adverse effects on ecological receptors. Although uncertainty will exist until an actual emission inventory can be constructed, the methods used to identify chemicals of potential concern provide the best available alternative at this time.

6.7.5 Chemical Speciation

The fate, food chain interactions, and toxicity of a number of inorganic contaminants depend to a large extent upon their chemical form. As such, conservative assumptions about chemical form, bioavailability, and absorption over the gut were generally carried forward in the risk assessment, and

the potential for toxicity is likely to be overstated. For example, it has been assumed that 100% of each ingested CoPC is absorbed from ingested soil or food, and is available to the organism as a potentially toxic substance. This may be reasonable for some CoPCs, but will be highly conservative for others.

6.7.6 Environmental Fate and Transport

The environmental fate and transport of CoPCs is modeled following US EPA and similar fate and transport models. While the overall model structures are believed to be generally reliable, the quality of many of the key parameter values describing the environmental fate and partitioning of CoPCs is variable. For some CoPCs and/or environmental media, the environmental fate and transport parameters are uncertain, and in the face of this uncertainty, conservative assumptions have been implemented that may overstate the likely environmental concentrations and exposure of wildlife to these and other substances.

6.7.7 Food Chain Interactions

Very limited "real world" data exist that allow quantification of the true relationship between a chemical in an environmental medium and chemical transfer through the food chain. Only a few classes of chemicals appear to be magnified through the food chain. These substances include methyl mercury, PCBs, some chlorinated pesticides (such as DDT), and some PCDD/PCDF compounds. These substances all have a tendency to partition into fatty tissue rather than water. They are also resistant to natural degradation processes by metabolic enzymes. PAHs and petroleum hydrocarbons represent other classes of hydrophobic chemicals present in the environment. While these hydrocarbons are hydrophobic, they can be metabolized and/or excreted by some invertebrates and most vertebrates. For this reason, food chain magnification does not tend to occur with PAHs and petroleum hydrocarbons, although they can still be accumulated to some extent by many wildlife species. For other organic substances, the extent of food chain magnification is not well understood. Among the inorganic chemicals, some, such copper and zinc are subject to biological regulation. Others, such as thallium and mercury, appear to have high potential for bioaccumulation, and still others, such as methyl mercury, undergo biomagnification in the food chain. The extent of food chain magnification is a source of uncertainty that is generally treated in a conservative manner.

6.7.8 Inhalation Pathway

This ERA does not explicitly evaluate the potential risks to ecological receptors from exposure to CoPCs via the inhalation pathway. Given the nature of this project, it is assumed that terrestrial receptors would be exposed to certain CoPCs in this manner. However, the magnitude of this exposure is not expected to rival the contribution from other environmental media following deposition and environmental transport of CoPCs. Although inhalation TRVs are available for several CoPCs, there is insufficient toxicity data for others. For this ERA, the inhalation risk to ecological receptors was derived from results of the HHRA. Given the conservative approach used in the HHRA, it was concluded that a lack of unacceptable risk for the HHRA (from the inhalation pathway), would also denote that this pathway would not present an unacceptable risk to ecological receptors.

Given the uncertainty associated with the above assumption, inclusion of this pathway in future site-specific risk assessments is warranted.

6.8 ERA Conclusions

The purpose of this ERA was to evaluate the additional potential risk introduced to ecological receptors from airborne emissions released into the environment as a result of the activity associated with the proposed energy-from-waste thermal treatment facility. The three scenarios evaluated in this ERA consisted of either one, two, or three process units in operation. As expected, additional process units translate to increased exposure to ecological receptors. However, for each scenario the estimated risk was determined to be well below levels of concern (HQ = 0.2) for all VECs. Additionally, results of the evaluation of inhalation risks to humans (Section 5) indicate that inhalation exposure to ecological receptors is not expected to be a concern. Therefore the operation of the proposed Project is not expected to introduce an unacceptable risk to ecological receptors living near the facility.

The only exception was a potential risk was identified for aquatic receptors exposed to modeled concentrations of dioxin at the A-7 Guideline (HQ=0.8). However, when the current day KMS Peel facility dioxin emissions were modeled it resulted in an acceptable level of risk to these receptors of HQ=0.1. This suggests that emissions of dioxin from any proposed facility should achieve emissions concentrations of dioxin below the A-7 Guideline.

That being said the limitation on the ecological risk assessment is that it evaluates the potential risk to receptors exposed only to facility emissions and then fate and transport through the ecosystem. It does not account for potential cumulative effects on background concentrations that need to be quantitatively assessed in any site-specific risk assessment.

7.0 STUDY LIMITATIONS

The intent of this study was to evaluate the potential health and environmental impacts that could result from siting an EFW facility in the Durham and York Regions. This HHERA was conducted following best-practices that would be employed in any site-specific risk assessment. However, there are a number of limitations to conducting a human and ecological risk assessment feasibility study for a theoretical facility. This section attempts to address a number of the limitations that should be taken into consideration in the event that Durham and York Regions pursue a thermal treatment EFW facility as one option for dealing with their residual municipal solid waste.

The greatest source of uncertainty and the principal limitations for this study are two fold:

3. The final preferred site for the thermal treatment EFW facility has yet to be determined.
4. The final technology and vendor have not yet been selected.

The air modelling exercise and HHERA were conducted on a "Regional" specific basis given that the final site has yet to be selected. Both the air modelling and the risk assessments are sensitive to site specific parameters and features that could not be fully accounted for in this study. In addition, background concentrations of potential contaminants of concern have yet to be collected. Therefore, for the most part, this study focuses on the incremental potential risk that would be expected from emissions from such a facility. Detailed collection of baseline chemical concentrations in environmental media, at and surrounding, the preferred site will be critical in assessing the potential cumulative risk that could exist from siting of such a facility.

The human health receptor groups selected for risk analysis in this study are those typically modelled in facility emissions risk assessment. Each of these receptor groups was theoretically assumed to be located at the MPOI (highest ground level concentrations) for facility emissions. This is a very conservative feature of this generic risk assessment as it is unlikely that the Subsistence Farm and First Nations communities would be located near the MPOI once the preferred site is selected. During the site-specific risk assessment it will be critical to understand the current and potential future land use surrounding the proposed EFW facility property. This will allow for modelled facility emissions, deposition, fate and transport and ultimately potential risk to each of these receptor groups to be evaluated.

Given that the preferred thermal treatment EFW technology and vendor have yet to be selected it was not possible to obtain facility specific emission factors in this study. Therefore, emissions from the KMS Peel facility and the MOE A-7 Guidelines were modeled for their potential risk. Although pollution control technology has advance greatly over the past decade for thermal treatment facilities, it is critical that a detailed emissions inventory be sought from all potential vendors of technology. These detailed emissions inventories should be screened to ensure that the appropriate chemicals of potential concern (CoPCs) are evaluated in the site-specific risk assessment.

Section 8.0 details further considerations that are being undertaken for the site-specific risk assessment.

8.0 CONCLUSIONS AND RECOMMENDATIONS

A human health and ecological risk assessment was conducted to ascertain the potential risks posed by siting a municipal solid waste thermal treatment EFW facility in the Durham/York Regions. Given that the final site has yet to be established, nor has the preferred technology or vendor been selected, this study was based on a "Regional" specific, theoretical facility.

Three operating scenarios were modelled for their emissions to air:

- Scenario 1 – 3 process units, facility processing 400,000 t/y
- Scenario 2 – 2 process units, facility processing 266,666 t/y
- Scenario 3 – 1 process unit, facility processing 133,333 t/y

The resulting ground level 1-hour, 24-hour and annual average concentrations were predicted for the maximum point of impingement; as well as wet and dry deposition concentrations to soil and water. These deposition rates of inorganic and organic contaminants of potential concern were then modelled through environmental fate and transport algorithms to establish concentrations in a variety of environmental media (e.g. soil, water, sediment, vegetation, wildlife).

It was determined that concentrations of contaminants in air met the Ontario Ministry of the Environment Schedule 3 and ambient air quality criteria published under O.Reg. 419 at the MPOI. In addition, concentrations of contaminants were added to baseline data collected at three MOE ambient air quality stations in the Durham/York Region. Short-term and long-term air concentrations from the facility did not pose an undue risk to residents, even if they were living at the maximum zone of impact. However, it should be noted that smog formation was not considered in this assessment.

A quantitative human health risk assessment was undertaken to determine if contaminant loading under any of the three Operating Scenarios would pose a potential risk to the following receptors if they were living and/or working at the MPOI:

- Commercial Worker and Commercial Day-care Toddler
- Durham/York Resident
- Durham/York Subsistence Farmer
- Durham/York First Nations and Métis

It was determined that for the Commercial Worker / Day Care scenario and the Resident scenario that these receptors could in fact exist at the maximum point of impingement of emissions from the theoretical, generic EFW facility. This was the case for all three Operational Scenarios, which included a facility capable of processing up to 400,000 t/y of municipal solid waste.

For the First Nations scenario it was determined that methyl mercury modelled for fish consumption could potentially pose a potential risk if these receptors lived at the MPOI. However, given the conservative nature of the assumed fish consumption rates and the theoretical watershed modelled these issues require further examination in a site-specific risk assessment.

In addition, for the Subsistence Farm and First Nations receptor scenarios, it was conservatively estimated that if dioxins and furans were emitted at the A-7 Guideline value it could potentially result in an undue risk to infants being exclusively breast fed up until the age of 6 months. When current-day emissions of dioxins and furans from the KMS Peel facility were modelled it was determined that the

risk levels fell to below the applicable benchmark for the Subsistence Farm infant, but not for the First Nations infant. This suggests that any EFW facility being considered for the Durham-York Regions should achieve dioxin and furan emission standards below the MOE A-7 Guideline and that particular attention needs to be paid to this issue.

The ERA evaluated the additional potential risk introduced to ecological receptors from airborne emissions released into the environment as a result of the activity associated with the proposed energy-from-waste thermal treatment facility. As expected, additional process units translate to increased exposure to ecological receptors. However, for each scenario the estimated risk was determined to be well below levels of concern (HQ = 0.2) for all VECs.

The only exception was a potential risk was identified for aquatic receptors exposed to modeled concentrations of dioxin at the A-7 Guideline (HQ=0.8). However, when the current day KMS Peel facility dioxin emissions were modeled it resulted in an acceptable level of risk to these receptors of HQ=0.1. This suggests that emissions of dioxin from any proposed facility should achieve emissions concentrations of dioxin below the A-7 Guideline.

Additionally, results of the evaluation of inhalation risks to humans (chapter 5) indicate that inhalation exposure to ecological receptors is not expected to be a concern.

Therefore the operation of the proposed Project is not expected to introduce an unacceptable risk to ecological receptors living near the facility.

That being said the limitation on the ecological risk assessment is that it evaluates the potential risk to receptors exposed only to facility emissions and then fate and transport through the ecosystem. It does not account for potential cumulative effects on background concentrations that need to be quantitatively assessed in any site-specific risk assessment.

Overall, it was determined that a thermal treatment EFW facility could be sited in the Durham and York Regions.

9.0 NEXT STEPS

The findings of this report will be used to support siting criteria to establish the preferred site, identifying potential concern for vendors of specific technology and ultimately to support a site-specific risk assessment.

This study was conducted a feasibility assessment of siting such a facility from a human and ecological health perspective only. Given that the EA is currently in the process of selecting a preferred vendor and technology and finalizing the preferred site, there are a number of follow-up recommendations that should be undertaken.

9.1 Baseline Environmental Data Collection

This generic risk assessment did not account for existing baseline chemical concentrations in the environment. In any site-specific risk assessment this information will be critical to understand the potential cumulative impact that the EFW facility would have on health and the environment. At the time of preparation of this report, a baseline monitoring program for a suite of contaminants of potential concern had been initiated in Durham and York Regions. Once the preferred site has been selected there are plans to conduct an extensive baseline chemical analysis of soil, water, sediment and biota in the area.

This baseline data collection serves two purposes. The first is that it will allow for meaningful site-specific data to be included in the site-specific risk assessment. The second, but as important, feature of this program is that it would serve as the environmental baseline or benchmark of chemicals in the environment prior to construction and operation of an EFW facility. Thus during the life of the facility, a monitoring program could be developed to monitor any change in chemical concentrations as a result of operations.

9.2 Site Specific Risk Assessment and Air Dispersion Modelling

A detailed site specific human health and ecological risk assessment and air dispersion modelling project should be undertaken once a preferred site and vendor is selected. This detailed site specific HHERA should address the concerns raised in this generic risk assessment and should include, at a minimum, consideration of cumulative environmental effects.

Cumulative environmental effects could only be determined if a detailed baseline collection of contaminants in a variety of environmental media is conducted. In addition to existing baseline concentrations of chemicals in the environment, consideration should also be given to the potential cumulative impact that other proposed projects could have, in addition to the EFW facility. These are projects that have been publicly disclosed in the area of the preferred site.

In the event that the initial results of the site-specific risk assessment reveal an unacceptable risk to either health and the environment, this does not automatically suggest that the facility could not still be built. Rather, discussions between the risk assessment team and the pollution control engineers could take place to enhance the performance of the technology to reduce the emission of chemicals to the environment.

Ultimately, prior to regulatory approval of the project, it will need to be clearly demonstrated that on a site-specific basis the emissions from the facility would not pose an unacceptable regulatory risk to either humans or the environment.

10.0 REFERENCES

AENV (Alberta Ministry of the Environment). 2005. Alberta Ambient Air Quality Objectives. Science and Technology Branch, Environmental Sciences Division, Environmental Service. Edmonton, AB.

ATSDR. 2005. Minimal Risk Levels (MRLs) for Hazardous Substances. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (http://www.atsdr.cdc.gov/mrls.html#bookmark02)

Cantox, 2000. Human Health Risk Assessment for the Proposed Expansion of the KMS Peel , Inc. Brampton, Energy-From-Waste Facility. Cantox Environmental Inc., Mississauga, ON.

Canadian Cancer Society. 2007. Canadian Cancer Statistics 2007.

CEPA. 1999. Canadian Environmental Protection Act.

Canadian Environmental Protection Act (CEPA), Priority Substance List Assessment Reports. Available online at: http://www.ec.gc.ca/CEPARegistry/subs_list/Priority.cfm

CCME. 1996. A Framework for Ecological Risk Assessment: General Guidance. Canadian Council of Ministers of the Environment. Winnipeg, Manitoba. En 108- 4-10-1996E.

Golding J. 1997. Unnatural constituents of breast milk--medication, lifestyle, pollutants, viruses. Early Hum Dev 49:S29-S43 (1997).

Health Canada, 2004a. Federal Contaminated Site Risk Assessment in Canada. Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA).

Health Canada, 2004b. Federal Contaminated Site Risk Assessment in Canada. Part II: Health Canada Toxicological Reference Values (TRVs).

Health Canada, 2004c. Exclusive Breastfeeding Duration - 2004 Health Canada Recommendation ISBN: 0-662-37809-1, Cat. No.: H44-73/2004E-HTML, HC Pub. No.: 4824, © Her Majesty the Queen in Right of Canada 2004

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

MOE (Ontario Ministry of the Environment), 2004. Guideline A-7 Combustion and Air Pollution Control Requirements for New Municipal Waste Incinerators. Standards development branch. Ontario.

MOE (Ontario Ministry of the Environment), 1999. Environmental Risk of Municipal Non-Hazardous Waste Landfilling and Incineration. Standard development branch, Environmental sciences and standards division. Ontario. ISBN 0-7778-8959-5

MOE (Ontario Ministry of the Environment). 2005. Summary of Point of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria. Online. Available at: http://www.ene.govon.ca/envision/gp/2424e01.htm

Nagy, K.A. 1987. Field metabolic rate and food requirement scaling inmammals and birds. Ecological Monographs. 57(2):111–128.

Oskarsson A, Ohlin I, Schutz A, Lagerkvist B, Skerfving S. 1996. Total and inorganic mercury in breast milk and blood in relation to fish consumption and amalgam fillings in lactating women. Arch Environ Health 51:234-241 (1996).

Oskarsson A, Palminger HI, Sundberg J. 1995. Exposure to toxic elements via breast milk. Analyst 120:765-770 (1995).

Richardson, G.M. 1997. *Compendium of Canadian Human Exposure Factors for Risk Assessment.* Ottawa: O'Connor Associates Environmental Inc.

Rogan WJ, Gladen BC, McKinney JD, Carreras N, Hardy P, Thullen J, et al. 1986. Polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in human milk: Effects of maternal factors and previous lactation. Am J Public Health 1986;76:172–7.

Ryan, A.S. The resurgence of breastfeeding in the United States. Pediatrics. 1997; 99:E12.

Sample, B. E. and C. A. Arenal, 1999. Allometric Models for Interspecies Extrapolation of Wildlife. Bull. Environ. Contam Toxicol. (1999) 62:653-663

TCEQ (Texas Commission on Environmental Quality). 2003. Effects Screening Levels (ESLs). TCEQ, 12100 Park 35 Circle, Austin, Texas. Online. Available at: http://www.tceq.state.tx.us/comm_exec/tox/ESLMain.html

US EPA. (Environmental Protection Agency). 1992. Framework for Ecological Risk Assessment. EPA/630/R-92/001. Risk Assessment Forum. Washington, DC

US EPA (Environmental Protection Agency). 1997. *Mercury Study Report to Congress, Volumes I through VIII*. Office of Air Quality and Standards and ORD. EPA/453/R-97-001. December.

US EPA. 1999. Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste and Emergency Response. Washington, D.C. August 1999.

US EPA. (Environmental Protection Agency). 2002. A Review of the Reference Dose and Reference Concentration Processes. U.S. Environmental Protection Agency. Risk Assessment Forum. EPA/630/P-02/002F.

US EPA. 2005. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP), Final. EPA520-R-05-006

US EPA. 2005. Hazardous Waste Companion Database, Microsoft Access™

US EPA (Environmental Protection Agency). 2007. Integrated Risk Information System (IRIS). Available at: http://www.epa.gov/iris/index.html

WHO (World Health Organization). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91.

WHO (World Health Organization). 2001. The Optimal Duration of Exclusive Breastfeeding. Report of an Expert Consultation, Geneva, Switzerland. 28-30 March 2000.

WHO (World Health Organization) 2005. WHO air quality guidelines global update 2005. WHO 8795.