SCIENCE INTEGRITY KNOWLEDGE

HUMAN HEALTH RISK ASSESSMENT REMASCO GASIFIER INSTALLATIONS KINGSVILLE ON

FINAL REPORT

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EXECUTIVE SUMMARY

Overview of the Study

REMASCO have been operating various gasifiers to generate hot water for the heating systems of the Southshore property for approximately 24 months. Over the last 12 months, the production version of the gasifier has been operated, tested, modified and tested again to ascertain performance with respect to operating efficiency, and most importantly emissions of contaminants to the atmosphere. Given the nature of the fuel being used in the gasifier, the Ministry of the Environment (MOE) require that the facility meets the A7 Emission Guidelines applied to municipal solid waste (MSW) incinerators operating in the province.
The A7 guideline is generally considered to be a technology based standard that sets a performance level for the emission control system that is deemed to be necessary for such facilities. At this performance level it is generally accepted that there will be minimal impacts on the environment and human health. This conclusion has been proven by human health risk assessment studies carried out for the only commercial MSW incinerator operating in the province, the APEFW facility in Brampton. Regardless of these findings, REMASCO undertook to conduct a Human Health Risk Assessment for its facility in Kingsville when it applied for its operating permit.

This document describes the tasks that will be undertaken for such an assessment. The assessment utilizes computer models to develop estimates of the atmospheric levels of contaminants associated with the facility and the rate of at which contaminants might be deposited on the ground in the area surrounding the facilities. Based upon these results, the Human Health Risk Assessment specialists ascertain the potential for these contaminants to enter the body of humans living in the area. Based upon their knowledge of the effects of these contaminants, they then determine the risk that these levels might pose to human health.

What is a Human Health Risk Assessment (HHRA)?

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

All chemicals (both natural and man-made) have the potential to cause effects in people and the ecosystem. It is the chemical concentration, the route of exposure, and the inherent toxicity of the chemical that determines the level of effect and potential for unacceptable risk to the exposed receptor for health risks arising from acute and chronic exposures.

As illustrated in the diagram to the right, if all three components are present (*i.e.,* where the three circles intersect), there is the potential for risk of adverse effects. If exposure is low enough, the risks may be considered "acceptable". Where technically and economically feasible, methods can be used to mitigate "unacceptable" risks.

It is acknowledged that the various uncertainties associated with the HHRA process have the potential to influence estimates of exposure and risk. The methods and assumptions used in this HHRA were designed to be highly cautious (*i.e.,* health protective), and have a built-in tendency to overestimate, rather than underestimate, potential health risks.

The current HHRA followed the standard HHRA framework (see Figure 2-1) that is composed of the following steps:

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

Typically, where potential adverse impacts are predicted through risk characterization, an additional step providing risk management goals and recommendations for mitigative measures to address these concerns is added. For the current EA process, it is this step that would provide recommendations for mitigation measures to the City of Hamilton should any unacceptable health risk related to facility emissions to the surrounding community be identified.

Who are the Sensitive Receptors in the Surrounding Community?

The proposed facility is located in an agricultural area of south-western Ontario. As such, most of the surrounding land is occupied by farmland and would fall into an agricultural land-use category. In addition to the surrounding farms and their residential dwellings, the town of Kingsville is located approximately 3 km southwest of the Agriville cluster. Various other community facilities are located in the area (schools, seniors residence, recreation facilities). To assess potential risks related to the projected emissions from the proposed greenhouse facility, key sensitive locations representative of the surrounding community were selected.

Based on provincial regulatory guidance, a sensitive receptor location is typically defined as:

- A senior citizen's residence or long-term care facility;
- A health care facility;
- A child care facility;
- An educational facility; or,
- A dwelling.

Based on these definitions, the locations of the nearest sensitive receptors were identified. The selected locations from the surrounding community were:

- Four residential areas: Agriville Residential (receptor R1), Southshore Residential S (receptor R2), Kingsville Residential (receptor R3), Southshore Residential N (receptor R4), and Residence S of Seacliff (receptor R6);
- Two schools: District School (receptor C1) and Ruthven School (receptor C2);
- Three other key community facilities: Recreation Complex (receptor C3), Seniors Residence (receptor C4), and Colisanti Facility (receptor R5); and,
- Three agricultural operations in the area: Asparagus Crop Land (receptor P1), Apple Orchard (receptor P2), and Vineyards (receptor P3).

What Chemicals were Assessed?

As the gasifiers operate in essence as incineration units, combusting pellets formulated from municipal solid waste, chemicals of concern (COC) were selected on the basis of two documents related to incineration of municipal solid waste in Ontario. These documents include a report published by the MOE (1999) entitled 'Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration. Technical Report Summary' and the Guideline A-7 which dictates combustion and air pollution control requirements for new municipal waste incinerators.

The following table provides a list of the final COCs and specific pathways evaluated in the current assessment.

Chemicals Selected for Evaluation in the Current Assessment		
Chemicals of Concern (COCs)	***Inhalation Exposures***	***Oral/Dermal Exposures***
Criteria Air Contaminants		
Sulphur Dioxide (SO_2)	✓	
Nitrogen Oxides (NOx)	✓	
Hydrogen Chloride	✓	
Particulate Matter (PM_{10})	✓	
Particulate Matter ($PM_{2.5}$)	✓	
Inorganics		
Arsenic	✓	✓
Cadmium	✓	✓
Chromium	✓	✓
Lead	✓	✓
Mercury (Inorganic)	✓	✓

Chemicals Selected for Evaluation in the Current Assessment		
Chemicals of Concern (COCs)	***Inhalation Exposures***	***Oral/Dermal Exposures***
Organics		
Vinyl Chloride	✓	✓
Benzene	✓	✓
PAHs		
Benzo(a)pyrene	✓	✓
Dioxins / Furans		
Dioxins & Furans[a]	✓	✓

[a] The polychlorinated dibenzo-*p*-dioxin and dibenzofuran family of compounds were evaluated as a group using toxic equivalency factors (TEFs) for tetrachloro dibenzo-*p*-dioxin (TCDD) as a surrogate.

How were Potential Exposures Evaluated?

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

- **Incidental Ingestion of Soil and Dust**: Through typical indoor and outdoor activities, individuals may accidentally ingest soil and/or dust particles. Children are typically more susceptible to this exposure pathway, as they spend more time in contact with the ground, and are more likely to put soiled articles, such as toys or hands, into their mouths.
- **Incidental Inhalation of Indoor Dust**: Soils impacted by particles emitted from the proposed facility were assumed to be carried indoors (*e.g.*, by wind, human and/or pet activities) and be present as indoor suspended dust for inhalation by individuals living within the home.
- **Dermal Exposure to Soils and Dusts**: Dermal exposures of human receptors may occur in both indoor and outdoor environments, through direct dermal contact with chemically-impacted soil and dust.
- **Breast Milk Consumption (infants only)**: It is assumed that infants living at each of the sensitive receptor locations will be exposed to certain chemicals *via* their mother's breast milk. This exposure pathway was evaluated for those organic COCs, such as dioxins and furans, which have the potential to "bio-accumulate".
- **Ingestion of Locally-Grown Produce**: Locally-grown produce (such as vegetables and fruits grown in backyard gardens) may itself pose a source of exposure to some COCs. As chemicals are deposited from air-borne emissions, they may come into contact with leaves and fruit of crop plants, where they may remain as a surface contaminant, or actually be absorbed into the plant. Deposition of chemicals onto soil may also result in an accumulation in plants through root uptake.

For the sake of conservatism, most of the potential pathways and exposure assumptions typically associated with a residential scenario were evaluated at all sensitive receptor locations. For example, when considering multimedia exposures (*i.e.*, non-inhalation), individuals at each of the assessed receptor locations were assumed to spend 24 hours per day, 7 days per week, 52 weeks per year at this location. This is obviously an overestimation of potential exposures for the schools. In the case of the Industrial Worker Scenario, the worker was assumed to be present on-site at the maximum ground-level air concentration for 8 hours per day, 5 days per week, 50 weeks per year.

What were the Assessment Results and Recommendations?

The purpose of the current assessment was to evaluate the potential incremental impacts of projected emissions (*i.e.*, from stack) from the gasification facilities proposed for the Kingsville area, and to determine the health implications to potentially sensitive individuals living, working, or playing in the surrounding communities, under "worst case" exposure conditions.

Acute Inhalation Assessment Results

The results of the acute inhalation assessment indicated that there are no acute impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreation/community scenarios.

Chronic Inhalation Assessment Results

The results of the chronic inhalation assessment indicated that there are no chronic impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreation/community scenarios.

Chronic Multi-Pathway Results

The results of the chronic multimedia (*i.e.*, inhalation, oral and dermal exposures) assessment indicated that there are no chronic impacts to human health expected as a result of deposition of facility emissions onto soils and home gardens of residences in the surrounding community. Furthermore, the worker scenario and the milk and produce consumer scenarios also indicated that there are no chronic impacts to human health expected as a result of these scenarios.

Cumulative Assessment Results

Evaluation of potential exposures under current and future cumulative conditions indicated marginal exceedances of acute and chronic TRVs for NOx and $PM_{2.5}$ at several receptor locations. In all cases, future cumulative risks with the proposed REMASCO facilities are equal to or lower than risks predicted under existing background conditions, indicating that there will be a net benefit in air quality with the installation and the operation of the REMASCO facilities.

Upset Scenarios

Evaluation of potential exposures under upset conditions at the maximum residential receptor location indicate that there are no acute or chronic impacts to human health expected as a result of emissions during upset conditions.

What are the Overall Conclusions?

This report has reviewed potential health impacts of the proposed REMASCO project to determine if the facility has the potential to cause impacts on the environmental and human health. This study relies on the results of an Air Quality assessment that will be published under separate cover. The assessment utilizes computer models to develop estimates of the atmospheric levels of contaminants associated with the facility and the rate of at which contaminants might be deposited on the ground in the area surrounding the facilities. Based upon these results, the Human Health Risk Assessment is able to ascertain the potential for these contaminants to enter the body of humans living in the area. Based upon this information and knowledge of the effects of these contaminants, the risk that these levels might pose to human health can be predicted. The purpose of the current assessment was to evaluate the potential incremental impacts of projected emissions (*i.e.*, from stack) from the gasification facilities proposed for the Kingsville area, and to determine the health implications to potentially sensitive individuals living, working, or playing in the surrounding communities, under "worst case" exposure conditions.

The key findings are as follows:

- The results of the acute inhalation assessment indicated that there are no acute impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreation/community scenarios.
- The results of the chronic inhalation assessment indicated that there are no chronic impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreation/community scenarios.
- Acute and chronic inhalation risks were marginally elevated for the worst-case exposures for on-site workers for the respiratory irritant group of COCs. These exceedances were not deemed significant and all predicted on-site concentrations are well below relevant occupational standards.
- The results of the chronic multimedia (*i.e.*, inhalation, oral and dermal exposures) assessment indicated that there are no chronic impacts to human health expected as a result of deposition of facility emissions onto soils and home gardens of residences in the surrounding community. Furthermore, the worker scenario and the milk and produce consumer scenarios also indicated that there are no chronic impacts to human health expected as a result of these scenarios.
- Evaluation of potential exposures under current and future cumulative conditions indicated that in all cases, future cumulative risks with the proposed REMASCO facilities are equal to or lower than risks predicted under existing background conditions. As a result, there will be a net benefit in air quality with the installation and the operation of the REMASCO facilities.

HUMAN HEALTH RISK ASSESSMENT REMASCO GASIFIER INSTALLATIONS KINGSVILLE ON

1.0 INTRODUCTION

REMASCO have been operating various gasifiers to generate hot water for the heating systems of the Southshore property for approximately 24 months. Over the last 12 months, the production version of the gasifier has been operated, tested, modified and tested again to ascertain performance with respect to operating efficiency, and most importantly emissions of contaminants to the atmosphere. Given the nature of the fuel being used in the gasifier, the MOE require that the facility meets the A7 Emission Guidelines applied to MSW incinerators operating in the province.

The A7 guideline is generally considered to be a technology based standard that sets a performance level for the emission control system that is deemed to be necessary for such facilities. At this performance level it is generally accepted that there will be minimal impacts on the environment and human health. This conclusion has been proven by human health risk assessment studies carried out for the only commercial MSW incinerator operating in the province, the APEFW facility in Brampton. Regardless of these findings, REMASCO undertook to conduct a Human Health Risk Assessment (HHRA) for its facility in Kingsville when it applied for its operating permit.

This document describes the tasks undertaken for such an assessment. The assessment utilizes computer models to develop estimates of the atmospheric levels of contaminants associated with the facility and the rate of at which contaminants might be deposited on the ground in the area surrounding the facilities. Based upon these results, the Human Health Risk Assessment specialists ascertain the potential for these contaminants to enter the body of humans living in the area. Based upon their knowledge of the effects of these contaminants, they then determine the risk that these levels might pose to human health.

To address this requirement, Intrinsik Environmental Sciences Inc. (Intrinsik) was retained by REMASCO to assess the potential human health implications associated with air emissions from the proposed facilities to the surrounding community. The primary goals of the current assessment were to evaluate the potential incremental impacts of projected emissions (*i.e.*, from stack) from the gasification facilities proposed for the Kingsville area, and to determine the health implications to potentially sensitive individuals living, working, or playing in the surrounding communities, under “worst case” exposure conditions. While this assessment has focused primarily on inhalation risks related to ground-level air concentrations predicted throughout the area, it also evaluated the potential risks associated with deposition of particulates onto soils and home gardens in the surrounding area.

Intrinsik was also requested to investigate the need for REMASCO to conduct a focused Ecological Risk Assessment (ERA) for its proposed facilities to ensure protection of local plants (including crops and greenhouse plants), invertebrates and wildlife. A screening-level ERA was conducted to address this issue.

Overall, this project is being completed to be consistent with the environmental assessment requirements outlined in *Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration* (MOE, 1999). It should be noted that these guidelines do not provide specific guidance on risk assessment methodology. Therefore, the current HHRA was conducted according to widely accepted risk assessment methodologies and guidance published and

endorsed by regulatory agencies including the Ontario Ministry of the Environment (MOE), Health Canada, the Canadian Council of Ministers of the Environment (CCME), and the United States Environmental Protection Agency (US EPA).

The current assessment was designed and conducted in the spirit of O. Reg. 153/04 (*i.e*., the overall methodological approach to risk assessments, as recommended by the MOE (2009a), but is not intended to meet the regulatory policy or administrative requirements of a brownfields RA under this regulation (*i.e*., this assessment is not being conducted for the purposes of registering a Record of Site Condition with the MOE). Furthermore, the current HHRA was conducted accordance with O. Reg. 419 and its accompanying guidance (MOE 2009b).

2.0 REVIEW OF STUDY METHODOLOGY AND ANALYSIS

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

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

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

i) problem formulation;

ii) exposure assessment;

iii) hazard assessment; and,

iv) risk characterization.

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

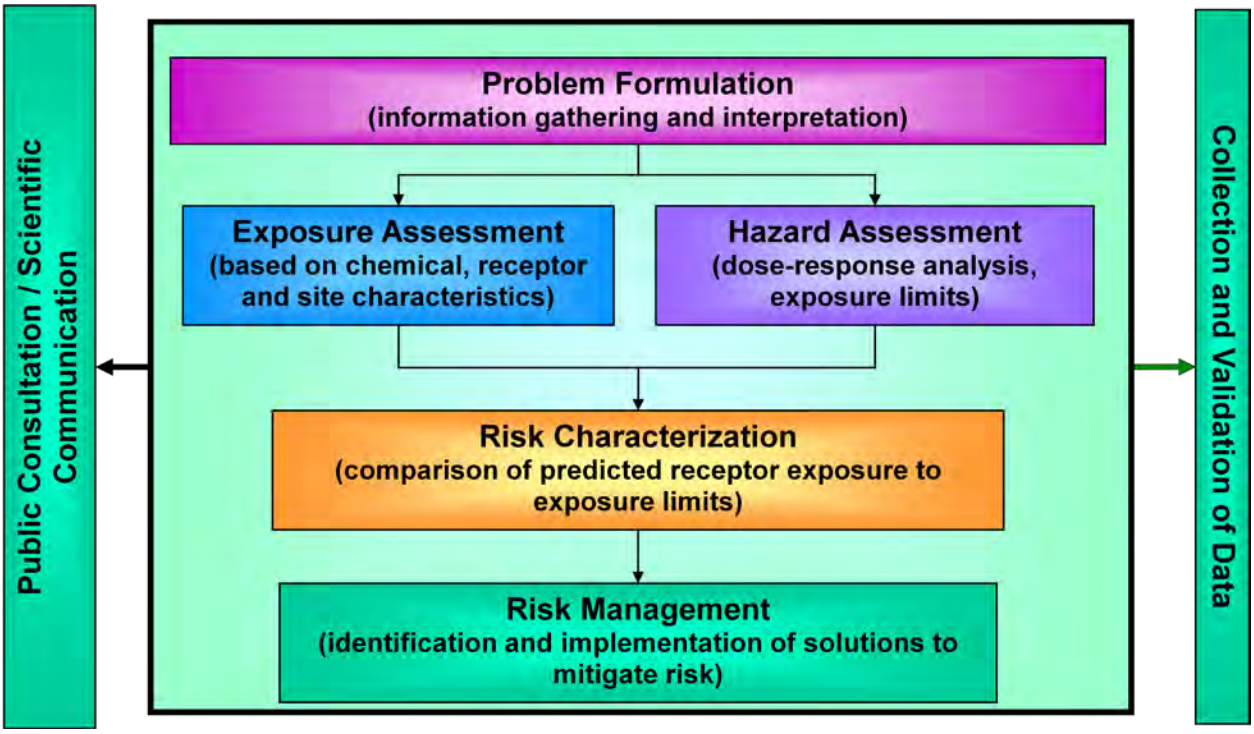


Figure 2-1 Overview of Standard HHRA Framework

2.1 Problem Formulation

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

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

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

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

2.2 Exposure Assessment

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

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

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

Given the nature of the project under assessment, and that the primary source of COCs to the environment will be *via* emissions to the atmosphere from the proposed facility, the primary route of exposure for people will be inhalation. However, for a subset of the COCs, there is the potential for deposition onto soils throughout the surrounding area, resulting in potential impacts to other exposure media. For these COCs, a multi-media assessment of potential risks related to oral and dermal exposures was conducted, in addition to the inhalation assessment.
For the inhalation exposure assessment, specific rates of exposure were not calculated. Rather, human exposures were conservatively assumed to be equal to ambient air concentrations (measured or modelled) of these substances (in $\mu g/m^3$). This inhalation assessment evaluates health risks from acute and chronic exposures (*via* direct air inhalation only) for all of the COCs at each of the sensitive receptor locations in the surrounding community.

For the multi-media assessment, the rate of exposure of the selected receptors to the COCs *via* the various exposure scenarios, pathways, and routes identified in the problem formulation step is estimated. The overall objective is to predict, using a series of conservative assumptions, the rate of exposure (in µg chemical/kg body weight/day) to the COCs *via* the oral and dermal exposure routes identified in the problem formulation. As air exposures are evaluated as part of the inhalation assessment, the multi-media assessment focussed on exposures arising from the oral and dermal pathways.

2.3 Hazard Assessment

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

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

In the case of threshold chemicals, a benchmark or threshold level must be exceeded for toxicity to occur. A NOAEL can be identified for threshold chemicals, which is the dose or amount of the chemical that results in no observable response in the most sensitive test species and test endpoint. The application of uncertainty or safety factors to the NOAEL provides an added level of protection, allowing for derivation of a *toxicity reference value* (TRV) that is expected to be safe to sensitive individuals following exposure for a prescribed period of time. Non-threshold chemicals are capable of producing cancer by altering genetic material. Regulatory agencies such as Health Canada and the US EPA assume that any level of long term exposure to carcinogenic chemicals is associated with some "hypothetical cancer risk". As a result, regulatory agencies have typically employed acceptable ILCR levels (*i.e.*, over and above baseline) between 1-in-100,000 and 1-in-1,000,000.

- Health Canada has specified an ILCR of 1-in-100,000, which is considered acceptable, tolerable or essentially negligible (Health Canada, 2004a).
- MOE considers an ILCR of 1-in-1,000,000 to be acceptable (*i.e.*, *de minimus*) for human health risk assessments in the Province of Ontario.

The CCME (2006) acknowledges that the designation of negligible cancer risk is an issue of policy rather than science. This acceptable ILCR of 1-in-1,000,000 increases a person's lifetime cancer risk from 0.400000 (based on the 40% lifetime probability of developing cancer in Canada) to 0.400001.

This HHRA is being conducted as part of an EA process in the Province of Ontario, and is being reviewed and considered by provincial regulators, including the MOE. As such, the HHRA reports ILCRs relative to the Ontario *de minimus* cancer risk benchmark of 1-in-1,000,000 (*i.e.*, one-in-one-million or 1×10^{-6}).

The terminology used to define threshold and non-threshold TRVs differs according to the source/media and type of exposure and often varies between regulatory jurisdictions. Generic nomenclature has been developed, with the following terms and descriptions commonly used.

Reference concentration (RfC): A reference concentration (or RfC) refers to the acceptable level of an airborne chemical for which the primary route of exposure is inhalation, and applies to either short term acute (*e.g.*, 1-hour or 24-hour) or long term chronic exposure periods. It is expressed as a concentration of the chemical in air (*i.e.,* micrograms per cubic metre, µg/m^3) and applies only to chemicals acting through a threshold mode of toxicological action.
For chemicals such as irritants and some combustion gases, short term or acute non-systemic toxicity is frequently observed at the points of entry into the body (*i.e.*, the respiratory tract, eyes, and skin, for air-borne contaminants). In these cases, because the toxicity is enacted simply by direct contact between the receptor and the contaminated medium, the concentration in the air to which the receptor is exposed is the important measure of exposure, rather than the internal dose associated with multiple exposure pathways. For chemicals with these characteristics, short term RfCs are used to characterize health risk, and are intended to be protective of the general population.

Reference dose (RfD): A reference dose (or RfD) refers to the acceptable level or dose of a chemical for which exposure occurs through multiple pathways (*i.e.,* inhalation, ingestion and dermal). It is most commonly expressed in terms of the total intake of the chemical per unit of body weight (*i.e.,* micrograms per kilogram of body weight per day, µg/kg bw/day), and typically represents a long term chronic exposure period. This term also applies only to chemicals acting through a threshold mode of toxicological action.

Unit risk value: The US EPA defines a unit risk value as "…the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m^3 in air…". A unit risk value of 3.0 x 10^{-5} per µg/m^3 would mean that under an upper worst-case estimate, three excess cancer cases are expected to develop per one hundred thousand (100,000) people, if exposed every day for a lifetime to 1 µg of the chemical per m^3 of air.

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

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

- ***Acute:*** the amount or dose of a chemical that can be tolerated without evidence of adverse health effects on a short term basis. These benchmarks are routinely applied to conditions in which exposures extend from minutes through several hours or several days only (ATSDR, 2006). For the current HHRA, risks will be evaluated based upon 1- or 24-hour exposure periods, where a relevant acute TRV for that time period is available.
- ***Chronic:*** the amount of a chemical that is expected to be without effect, even when exposure occurs continuously or regularly over extended periods, possibly lasting for periods of at least a year, and possibly extending over an entire lifetime (ATSDR, 2006).

Although it would be inappropriate to establish a generic hierarchy of source agencies by which to select TRVs given the breadth of COCs evaluated in a typical HHRA, priority was given to TRVs selected by MOE (2008; 2009a). When a TRV was selected that was different from that utilized by MOE in Reg. 153 (MOE, 2009a) or Reg. 419 (MOE, 2008), or in cases where TRV

values are not provide by MOE (2008, 2009a), toxicological profiles, including a detailed discussion of the relevant information supporting the selected TRV, are provided Appendix A are shaded. In cases where values differ from those utilized by MOE, all of the available TRVs were reviewed and the professional judgment of experienced toxicologists was used to select the most appropriate TRV. The most critical considerations in selecting TRVs were the source (it must have been derived by a reputable agency), the data used to derive the benchmark, the date the TRV was derived (it must be as up to date as possible), and its relevance in terms of duration and route of exposure. Both MOE (2009a) and Health Canada (2004b) provide lists of acceptable jurisdictions that maybe be used to determine toxicity reference values. In some occasions, additional jurisdictions outside this list can be selected based on the professional judgement of an experienced toxicologist. The TRVs employed in the HHRA have been obtained from regulatory agencies such as:

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

2.4 Risk Characterization

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

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

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

- Acute inhalation (1-hour and 24-hour durations);
- Chronic inhalation (annual average durations); and,
- Chronic multiple pathways (*i.e.,* oral and dermal exposures).

2.4.1 Estimating Potential Risk

2.4.1.1 Threshold Chemicals (Non-carcinogens)

Concentration Ratios (CR)

CR values were used to evaluate the acute and chronic health risk from exposure to chemicals *via* inhalation. CR values were calculated by dividing the predicted ground-level air concentration (for 1-hour, 24-hour or annual average exposure durations) by the appropriate toxicity reference value (*i.e.*, RfC), according to the following example equation:

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

Where:

$CR_{duration}$	=	the duration-specific *CR* (unitless), calculated for 1-hour, 24-hour and chronic durations, as appropriate
$[Air]_{duration}$	=	the predicted ground-level air concentration (µg/m^3) for the specific time duration
$RfC_{duration}$	=	the RfC (µg/m^3) for the specific time duration

Hazard Quotients (HQ)

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

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

Where:

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

2.4.1.2 Non-Threshold Chemicals (*i.e.*, Genotoxic Carcinogens)

Incremental Lifetime Cancer Risks (ILCR)

ILCR estimates were used to evaluate the increased cancer risk resulting from a lifetime of exposure to non-threshold genotoxic carcinogenic chemicals. ILCR estimates provided the incremental lifetime cancer risk resulting from the additional contributions of facility emissions into the surrounding community.

Direct Air Inhalation

ILCR estimates resulting from direct air inhalation were calculated as follows:

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

Where:

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

Multi-Media Exposure

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

$$ILCR = LADD \times CSF$$

Where:

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

2.4.2 Interpretation of Risk Estimates

The interpretation of the various risk evaluation metrics, as well as the appropriate benchmark by which to evaluate whether the predicted risk is acceptable or not, are discussed in the following section.

2.4.2.1 Threshold Chemicals (Non-carcinogens)

If the risk assessment evaluates risks associated with a single source or media (such as inhalation), the selection of a CR or HQ of 1.0 as an indication that predicted exposures do not exceed the relevant regulatory benchmark or TRV is appropriate. For example, as gaseous chemicals such as oxides of nitrogen (NO_x) only occur in air, and not in other media, the appropriate CR benchmark is 1.0 (*i.e.*, 100% of the regulatory benchmark).

For chronic multi-media exposures, the Canadian Council of Ministers of the Environment (CCME, 2006) allotted 20% of the total exposure to any one media during the derivation of its health-based soil quality criteria. This was based on the assumption that exposure to COCs may occur *via* five potential media: air, food, water, soil, and consumer products. This means that, in the absence of a multi-media assessment that takes into account multiple sources or media, the TRV should be apportioned for the single media under consideration. HQ values that are less than 0.2 represent a situation in which Project-related exposures (*e.g.*, facility-related emissions) account for less than 20% of the TRV. Therefore, no adverse health risks are expected to be associated with the estimated level of exposure. A similar source attribution or allocation model has been adopted by the MOE (2009a).

When predicted risks are greater than the benchmark level (*e.g.*, CR/HQ value greater than 1.0 or 0.2), this may indicate the potential for adverse health outcomes in sensitive individuals or in some of the exposure scenarios considered. Re-evaluation of such HQs and CRs is important since both the exposure estimates and the toxicological criteria are based on a series of conservative assumptions, particularly when considering the maximum "worst-case" exposure scenarios.

Concentration Ratio (CR)

Acute and Chronic CR values less than the selected benchmark indicate that estimated chemical concentrations in air are less than the applicable regulatory benchmark or RfC, and thus, adverse health outcomes would not be expected to occur. As this is usually a straight comparison between predicted air concentrations (*i.e.*, for 1-hour, 24-hour or annual average exposure durations) and the regulatory TRV, the resulting CR value is receptor-independent (*i.e.*, the same value is calculated for all receptor types).

Since acute TRVs are typically specific to chemical concentration within a single environmental medium (*i.e.*, air), short term TRVs are not typically apportioned for source attribution, as such an acceptable benchmark of 1.0 is selected.

For COCs only expected in a single media, such as the gases which only occur in air, and not other media, or for COCs with a published inhalation TRV, a benchmark representing the entire TRV (*i.e.*, a CR benchmark value of 1.0) is appropriate for chronic durations as well. Source allocating 20% of the TRV to the inhalation pathway (*i.e.*, a CR benchmark value of 0.2) is not necessary since this is the only pathway of exposure considered by the TRV. Where chronic exposures must consider multipathway exposures, as in the case of some of the inorganics, VOCs, PAHs and dioxins/furans, source apportionment would be appropriate and a benchmark of 0.2 is typically selected since as not all potential multi-media exposures sources of the COCs were considered.

It should be noted that some regulatory benchmarks can be dated and include policy adjustments, such as achievability based on best available technology at the time of the benchmark derivation. As a result, in cases where predicted or measured concentrations have not exceeded the regulatory benchmark, it is not necessarily indicative of a lack of potential health risk. For example, health effects for some criteria air contaminants can occur at very low levels, where no threshold has been identified (*e.g.*, particulate matter). Please refer to the detailed toxicological profiles presented in Appendix A for a more complete chemical-specific discussion of the regulatory benchmarks selected for use in the current assessment.

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

CR ≤1

- Signifies that the estimated exposure is less than or equal to the regulatory benchmark or TRV (*i.e.,* the assumed safe level of exposure). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV. An exception to this may be in the evaluation of certain criteria air contaminants where no threshold for effects has been identified.

CR >1

- Signifies the exposure estimate exceeds the regulatory benchmark or TRV. This suggests that the potential for an elevated level of risk may be present for some COCs. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e*., the margin of safety is reduced but not removed entirely).

Hazard Quotient (HQ)

If the estimated exposure to a chemical is equal to or less than the toxicological criterion (*i.e.,* the TRV) then the HQ value would be less than or equal to 1.0 and, in general, no adverse health outcomes would be expected to occur. For the current assessment, as not all potential multi-media exposures sources of the COCs were considered, a benchmark of 0.2 was selected for the evaluation of the chronic multi-pathway assessment.

In cases where the calculated HQs are below the benchmark level, no adverse health outcomes would be expected to occur, even considering sensitive members of the population.

If calculated HQs are within an order of magnitude of the benchmark level (*e.g.*, HQ <10), this would indicate situations that may require re-evaluation of model parameters and an examination of the conservative assumptions used in the assessment (*e.g.*, chemical concentration estimates, exposure parameters, and toxicological criteria) before the potential health risks can be characterized. Although there is a possibility of adverse health outcomes, such an exceedance is not necessarily indicative of actual risks. The elevated HQ may reflect the overestimation of risk due to the use of overly conservative assumptions (*e.g.*, overestimating exposures through use of maximum soil ingestion rates), and TRVs that are typically designed with uncertainty factors that can span several orders of magnitude. This approach is intended to be conservative and ensure the predicted health outcomes on human health are not under-estimated.

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

HQ ≤0.2

- Signifies that the estimated exposure is less than or equal to 20% of the TRV (*i.e.,* the assumed safe level of exposure). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV and exposure estimate.

HQ >0.2

- Signifies the exposure estimate exceeds 20% of the benchmark. This suggests that the potential for an elevated level of risk may be present for some COCs. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e.*, the uncertainty is reduced but not removed entirely).

2.4.2.2 Non-Threshold Chemicals (*i.e.*, Genotoxic Carcinogens)

Incremental Lifetime Cancer Risk (ILCR)

Non-threshold chemicals that can alter genetic material (*i.e.*, genotoxic) are capable of producing cancer. Regulatory agencies such as Health Canada and the US EPA have therefore assumed that any level of long term exposure to a carcinogenic compound is associated with some "hypothetical cancer risk". As a result, regulatory agencies have typically employed acceptable ILCR levels (*i.e.*, incremental cancer risks over and above background cancer incidence) between 1-in-100,000 and 1-in-1,000,000. ILCRs generally consider risks related to a particular facility (facility alone, excluding any contribution from other background sources) in that the cancer risks are expressed on an incremental or additional basis as compared to cancer risks related to all sources.

As this HHRA is being conducted as part of the EA process for the Province of Ontario, a benchmark ILCR of 1-in-1,000,000 (1×10^{-6}) was selected, based upon MOE policy for risk assessments in Ontario. The definition of a benchmark ILCR of 1-in-1,000,000 is a policy based decision, not a scientifically derived value. An ILCR of 1-in-1,000,000 increases a person's lifetime cancer risk from 0.400000 (based on the 40% lifetime probability of developing cancer in Canada) to 0.400001. It is recognized that some amount of the "background" cancer risk of 40% is likely associated with exposures to environmental pollution. It must be noted, however, that an ILCR of 1-in-1,000,000 (a level below which the MOE considers acceptable) represents a 0.00025% increase over the background cancer incidence, an increase that cannot be detected using epidemiological data from the study area (Health Canada, 2004a). It is noted that other regulatory agencies, including Health Canada, consider an ILCR of 1-in-100,000 as the *de minimus* risk level considered protective of public health.

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

ILCR ≤ 1.0×10^{-6} (1E-06)

- Signifies that the estimated exposure results in an incremental lifetime cancer risk less than or equal to 1-in-1,000,000 (*i.e.*, within the accepted level of risk set by MOE and 10-times lower (more conservative) than that set by the Health Canada). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV and exposure estimate.

ILCR > 1.0×10^{-6} (1E-06)

- Signifies the estimated exposure results in an incremental lifetime cancer risk greater than the acceptable regulatory-established cancer risk benchmark of 1-in-1,000,000. This suggests that the potential for an elevated level of risk may be present for some COCs. The significance of which must be balanced against the high degree of

conservatism incorporated in the risk assessment (*i.e*., the uncertainty is reduced but not removed entirely).

2.4.3 Chemical Mixtures

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

Because chemical exposures rarely occur in isolation, the potential health outcomes associated with mixtures of the COCs were assessed in the HHRA. The interaction between chemicals can take many forms, with additive interactions being assumed for the HHRA (Health Canada, 2004a). Additive interactions apply to chemicals that are structurally similar, act toxicologically through similar mechanisms or affect the same target tissue in the body (*i.e.,* share common health outcome) (Health Canada, 2004a).

The evaluation of risks related to chemical exposures in mixtures is an emerging science. There are currently no regulatory benchmarks or specific guidance (beyond those chemical groups that have established *toxic equivalency factors*, or TEFs) by which one could evaluate whether exposure to a given mixture could pose a health concern. As such, for the current assessment, cumulative risks for a given mixture group are provided for information purposes only. As there is no regulatory guidance for an appropriate comparative benchmark for mixture groups of similar toxicity, it would be inappropriate to compare these predicts to a CR benchmark of 1.0, an HQ benchmark of 0.2, or an ILCR of 1×10^{-6} (*i.e.*, one-in-one million). In particular, as noted previously, the ILCR represents the incremental risk of an individual within a given population developing cancer over his or her lifetime due to exposures from a **specific** carcinogenic chemical, and is not intended to evaluate the risk from a mixture of COCs.

For the current assessment, the health endpoint of the TRVs used in the HHRA provided the basis for the inclusion of an individual chemical in a chemical mixture.

3.0 PROBLEM FORMULATION

The current assessment followed standard risk assessment methods, and was conducted in compliance with the risk assessment procedures endorsed by regulatory agencies including Environment Canada, Health Canada, the Canadian Council of Ministers of the Environment (CCME), and the US EPA, as well as guidance provided by the MOE.

3.1 Site Characterization

REMASCO plans to operate twelve 500 hp gasifier units at two locations near Kingsville, ON (see Figure 3-1). The units will process a total of 265 tonne/day of pelletized municipal solid waste and generate a total of 4500 Gj/day thermal energy and 48 mWhr/day electrical energy. The thermal energy will be used to provide heat for 170 acres of greenhouses in the area.

The gasifiers will be located at two clusters of greenhouses, with four units at Agriville Farms Ltd. (Agriville) and seven units at Southshore Greenhouse Inc./Mucci Farms (Southshore) (see Figure 3-2). All of the greenhouses at both locations grow above ground vegetables, with specific crops as follows:

- Southshore - beefsteak tomatoes and sweet peppers.
- Mucci Farms - tomatoes, peppers, cucumbers and eggplant
- Agriville - beefsteak, cluster, and Sapori ™ cocktail tomatoes

The gasifier units will operate on pelletized fuels produced by the Dongara Pellet Plant. Dongara processes municipal solid waste into trademarked EnerPax+ fuel pellets. The residential waste goes through a vigorous separation process to ensure that minimal metals or recyclable plastics are present within the pellets. EnerPax+ pellets have an energy content similar to medium grade coal.

The maximum point of impingement (MAX) of air emissions from the proposed facilities exists near the Southshore site.

Figure 3-1 Site Location

The Town of Kingsville Interactive Mapping

SSGH/MucciPac/Agriville

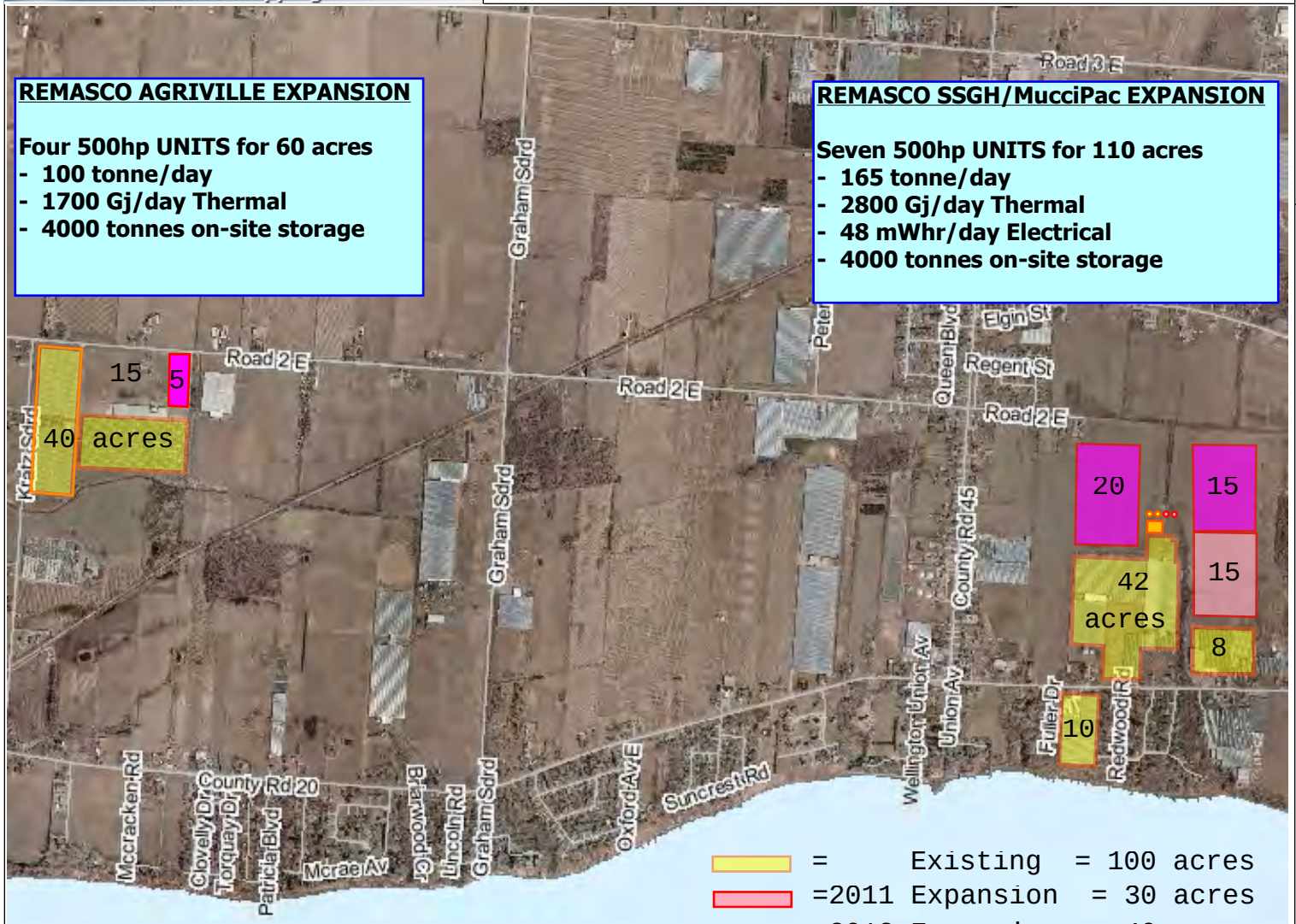


Figure 3-2 Proposed Site Location

Legend

- Municipal Boundary
- Streets
- Water

Scale: 1:25,500

The Town of Kingsville Interactive Mapping

3.1.1 The Surrounding Area

The Project is located in an agricultural area of south-western Ontario. As such, the majority of the surrounding land is occupied by farmland and would fall into an agricultural land-use category. Of note, one dairy farm exists approximately 9 km northwest of the Agriville facility.

In addition to the surrounding farms and their residential dwellings, the town of Kingsville is located approximately 3 km southwest of the Agriville cluster. Various other community facilities are located in the area, including, a school located approximately 6 km northwest of Agriville (District School), Ruthven School located approximately 1 km north of Southshore), a seniors residence located west of Agriville, a recreation complex located west of Agriville, and Colisanti's Tropical Gardens located northwest of Southshore. The City of Windsor lies 30 km to the west. Lake Erie lies approximately 1.5 km and 4 km south of the Southshore and Argiville locations, respectively.

In addition to the maximum point of impingement (MAX POI), 13 'sensitive' receptor locations were selected for evaluation in the HHRA (Figure 3-3).

Table 3-1 Sensitive Receptor Locations

Receptor Location	Location Code[a]		Easting	Northing	Approximate Distance to nearest REMASCO Installation (m)	Location Type
	HHRA	AQ Report				
Agriville Residential	R1	S5	358400	4656900	120	Residential
Southshore Residential S	R2	S13	362450	4655560	600	Residential
Kingsville Residential	R3	S3	357200	4655900	1500	Residential
District School	C1	S1	356300	4658400	2600	Community/Recreational
Ruthven School	C2	S12	362300	4657000	800	Community/Recreational
Southshore Residential N	R4	S11	362000	4656500	480	Residential
Recreation Complex	C3	S4	357500	4656700	950	Community/Recreational
Seniors Residence	C4	S2	356200	4656900	2300	Community/Recreational
Colisanti Facility	R5	S9	361300	4658200	2300	Residential/Recreational
Asparagus Crop Land	P1	S6	358600	4656000	625	Produce/Agricultural
Apple Orchard	P2	S7	360500	4655900	1750	Produce/Agricultural
Vineyards	P3	S8	360200	4657800	2250	Produce/Agricultural
Residence S of Seacliff	R6	S10	361200	4655400	1400	Residential

[a] Receptor location codes used for the Air Quality (AQ) Assessment (Chandler, 2011) and the HHRA are not consistent.

Geocortex Essentials for ArcGIS Server

Internet Mapping Framework

Legend

Essex Municipalities

<all other values>

Kingsville

Street

2008 Airohoto

Red: Band_1

Green: Band_2

Blue: Band_3

Figure
Shows locations of special receptors used for the cumulative impact assessment portion of study

Figure 3-3 Sensitive Receptor Locations

1: 23,457

1,191.6 0 595.82 1,191.6 Meters

This map is a user generated static output from an Internet mapping site and is for reference only. Data layers that appear on this map may or may not be accurate, current, or otherwise reliable.

THIS MAP IS NOT TO BE USED FOR NAVIGATION

Notes

This map was automatically generated using Geocortex Essentials.

It is important to note that by assessing the most sensitive and highly exposed receptor locations (*i.e.*, toddlers living in the closest residence to the facility, at the highest potential inhalation and deposition exposures), one is inherently being protective of all other less sensitive or highly exposed receptor locations (*i.e.*, residences or schools at a greater distance from the facility). This is a standard tenant of risk assessment. As such, one does not need to assess every single residential location within the general vicinity of the facility to ensure an accurate evaluation of community risk – those not specifically assessed are accounted for by assessing those locations that are the most sensitive.

In addition to these residential and school receptor locations, potential health risks to on-site workers were also evaluated as part of the assessment. To ensure the greatest conservatism, the on-site worker was assumed to be constantly present at the maximum point of impingement (*i.e.*, the area on the site with the maximum ground level air concentration due to facility emissions). As this facility will be operated in compliance with all Occupation Health and Safety and Ministry of Labour regulations, the assessment of occupational risks (beyond the worker exposed to the maximum point of impingement) was considered beyond the scope of the current assessment.

Two additional scenarios were also considered those being (i) a milk consumer (a person not living in the vicinity of the facilities whom consumers milk exclusively from the dairy farm located northwest of the Agriville facility); and, (ii) a vegetable consumer (a person not living in the vicinity of the facilities whom consumers vegetables exclusively from the greenhouse facilities).

3.2 Chemical Characterization

As the gasifiers operate in essence as incineration units, combusting pellets formulated from municipal solid waste, chemicals of concern (COC) were selected on the basis of two documents related to incineration of municipal solid waste in Ontario. The Guideline A-7 which dictates combustion and air pollution control requirements for new municipal waste incinerators provides emission limits for a list of nine (9) parameters. These nine (9) parameters have been selected for assessment in the current HHRA. In addition, MOE (1999) published a report entitled 'Environmental Risks of Municipal Non-Hazardous Waste Landfilling and Incineration. Technical Report Summary'. Within this report, MOE determined it necessary to assess fifteen (15) COCs. With the exception of silicon, iron and tin, these parameters have also been assessed in the current HHRA. A rationale for the exclusion of these naturally occurring inorganic nutrients and naturally abundant metals of low toxicity is provided below.

Iron

Iron is the fourth most common element in the earth's crust (approximately 5%) and the second most common metal (HSDB, 2008). It is most commonly found in soils as hydrous ferrous oxides. Iron compounds are commonly found in the environment and many foods, with humans exposed to it *via* inhalation of ambient air, ingestions of food, dietary supplements and drinking water. An adult body contains between 2.5 and 4 grams of iron, mostly found in red blood cell haemoglobin (HSDB, 2008).

Silicon

Silicon is an essential nutrient, particularly for plants. In humans it is involved in the formation of bone and connective tissue. Silicon is the second most abundant element in the earth's crust (26%), usually found as silica and silicate. Thus, silicon is normally found in high background concentrations, particularly in soil. In drinking water it is normally found as orthosilicic acid. In some grains, silicon can be found at concentrations exceeding 4,000 mg/kg (wet weight), and in

beer exceeding 33,000 µg/L (EVM, 2003). An oral exposure limit of 25,000 µg/kg/day has been proposed for adults (Takizawa *et al*., 1998).

Tin

The earth's crust is comprised of 0.0006 % of tin occurring in concentrations between 2 and 3 ppm (ATSDR, 2005). It forms both inorganic and organic compounds which bind to strongly to soils and sediment. However, organic compounds can be degraded into organic tin in the environment (ATSDR, 2005). Typically, exposure to tin occurs through occupational exposure to tin *via* inhalation of dust, consumption of contaminated drinking water, food and soil. Concentrations of tin in the soil are approximately 1 ppm but contaminated soils may have up to 200 ppm of tin. Children consuming soil will still have a low exposure to tin even at the rate of 200 mg/d. Exposure to tin increases when food is consumed from tin cans in particularly, unlacquered cans. Food in non metal cans contains less than 2 ppm. Exposure increases, particularly for organic tin which is present in seafood and household products such as PVC pipes. Exposure to air is normally less than 1 ppm from air and water (ATSDR, 2005).

Carbon Monoxide (CO)

Carbon monoxide (CO) was not evaluated as a COC. As discussed in Chandler (2011), emission rates for CO are very low and as such CO was not considered a potential COC.

In addition, the carcinogenic PAHs, as represented by benzo(a)pyrene, were also included in the assessment.

The final list of COCs in provided in Table 3-2.

Table 3-2 Chemicals of Concern Selected for the HHRA

Criteria Air Contaminants	Guideline A-7 Parameter	MOE (1999)
Sulphur Dioxide (SO_2)	X	X
Nitrogen Oxides (NO_x)	X	X
Hydrogen Chloride	X	X
Particulate Matter (PM_{10})	X	X
Particulate Matter ($PM_{2.5}$)	X	X
Inorganics		
Arsenic		X
Cadmium	X	X
Chromium		X
Lead	X	X
Mercury (Inorganic)	X	X
Organics		
Vinyl Chloride		X
Benzene		X
PAHs		
Benzo(a)pyrene		
Dioxins / Furans		
Dioxins & Furans	X	X

It is noted that similar assessments have been conducted for proposed waste-to-energy facilities in Ontario, including the Algonquin Energy facility in Brampton, ON (formerly KMS Peel) (Cantox, 2000) and the Durham/York Regional Waste EA (Stantec, 2009). These

assessments have considered much larger facilities and larger suites of chemicals as potential COCs. In both cases, the key parameters of concern have been captured herein, with risk estimates for other COCs falling orders of magnitude lower than the established benchmarks of concern.

As part of the pilot testing for the proposed facility, REMASCO currently owns and operates two gasification units at the Southshore location. These units have exceeded all expectations since operation began in January of 2009, consistently delivering their design rated capacity of 400 hp and passing or exceeding all applicable MOE emission standards. Chemical characterization for the facility was based on emission rates for the COCs based on the source testing at the pilot facility. Further details regarding chemical characterization and the determination of emissions rates for the proposed facility are provided in the Air Quality Report (Chandler, 2011).

3.2.1 Dispersion Modelling

AERMOD dispersion modelling was conducted by Chandler (2011) based MOE protocol and standards required for compliance with Ontario Regulation 419/05. AERMOD is an atmospheric dispersion modelling system, developed jointly by the American Meteorological Society (AMS) and the US EPA, which includes three specific modules: i) a steady-state dispersion model; ii) a meteorological data pre-processer that calculates atmospheric parameters needed by the dispersion model; and iii) a terrain pre-processor which provides a physical relationship between terrain features and the behaviour of air pollution plumes.

Modelling conducted using AERMOD permits the estimation of maximum ground-level air concentrations for each COC, as well as deposition rates for a subset of these chemicals (where deposition is an option). A detailed overview of the methodology and assumptions used by Chandler in the AERMOD dispersion modelling is provided in the Air Quality Report (Chandler, 2011).

3.3 Receptor Characterization

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

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

- Federal Contaminated Sites Risk Assessment in Canada. Part I: Guidance on Human Health Risk Preliminary Quantitative Risk Assessment (PQRA) (Health Canada, 2004a);
- Procedures for the Use of Risk Assessment under Part XV.1 of the *Environmental Protection Act*. (MOE, 2005);

- Compendium of Canadian Human Exposure Factors for Risk Assessment (Richardson, 1997);
- HHRA for Priority Substances: *Canadian Environmental Protection Act*: ISBN 0-662-22126-5 (Health Canada, 1994);
- Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (US EPA, 2004); and
- The US EPA HHRA Protocol (HHRAP) for Hazardous Waste Combustion Facilities (US EPA, 2005).

Preference was given to Canadian guidance documents and literature (*e.g.*, Richardson, 1997; Health Canada, 2004a; MOE, 2005). However, it is recognized that the US EPA publishes guidance material containing receptor characterization data not currently available in Canadian sources and, therefore, certain US EPA documents were used as a primary source of human receptor characterization data. The US EPA (2005) HHRAP document serves as a primary source for many of the fate and transport methods and general exposure scenarios. The receptor data published in the HHRAP (US EPA, 2005) has been designed to work with these fate and transport methods and the general exposure approaches. Therefore, the HHRAP was also used to help characterize human receptors.

As per Health Canada (2004a) guidance, the *residential receptor* was assumed to be represented by five discrete life stages:

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

In the case of carcinogenic COCs, potential incremental lifetime cancer risks were evaluated for a lifetime composite receptor, which combined each of the above lifestages.

The residential receptor was assumed to be born in the Kingsville area when the gasifiers begin operations, and live there during the entire period of operations (assumed 30 years). The individual was assumed to be exposed *via* inhalation of ambient air to emissions from the proposed facilities. The resident was also assumed to be exposed to COCs through contact with contaminated soil or home grown produce impacted by the deposition of the emitted COCs onto surface soils in the surrounding community. Predicted soil concentrations were conservatively assumed to be the maximum concentration that would be present after 30 years of deposition.

For the assessment of inhalation risks, as a straight comparison between predicted short term, acute (*i.e.*, for 1-hour and 24-hour exposure durations) and long term, chronic (*i.e.*, annual average exposures) air concentrations and the corresponding regulatory RfC was made, the resulting CR value is receptor-independent (*i.e.*, the same value is calculated for all receptor types). In the case of the multi-pathway assessment, oral and dermal exposures to the select COCs were evaluated for the most sensitive receptor groups living in the surrounding community – toddlers (preschool children). Consideration was also given to infants consuming breast milk.

Refer to Appendix B for further information on the assumptions used to characterize receptors evaluated in the current assessment

3.4 Identifying Exposure Scenarios and Pathways

3.4.1 Exposure Scenarios

For the current assessment, five specific exposure scenarios were evaluated: 1) residential; 2) recreational; 3) worker; 4) milk consumer and, 5) consumer.

The *residential* scenario evaluates the potential health impact related to the predicted ground-level air concentrations and deposition rates of each of the COCs at the nearest off-site residential receptor locations. The residential scenario was applied to receptor locations R1 (Agriville Residential), R2 (Southshore Residential S), R3 (Kingsville Residential), R4 (Southshore Residential N), R5 (Colisanti Facility) and R6 (Residence S of Seacliff). This scenario considered inhalation and multimedia (soil, home garden, breast milk) related pathways.

The *recreational/community* (non-residential community facility) scenario evaluates the potential health impact to the predicted ground-level air concentrations and deposition rates of each of the COCs at off-site community facilities. This scenario was applied to receptor locations C1 (District School), C2 (Ruthven School), C3 (Recreation Complex), and C4 (Seniors Residence). This scenario considered inhalation and multimedia (soil only) related pathways, with the exception of the Seniors Residence (C4) at which only inhalation related pathways were considered.

The *worker* scenario evaluates the potential health impact to an on-site worker at the predicted maximum ground-level air concentrations on the greenhouse properties (*i.e.*, MAX POI) due to emissions from the proposed gasifiers. The scenario only considered inhalation related pathways.

The *milk consumer* scenario evaluates a toddler not living in the Kingsville area whom consumer milk exclusively from the dairy farm located northwest of the Agriville facility (R1). Environmental media concentration (air, soil and silage) were predicted at the dairy farm location. Based on these environmental media concentrations, potential COC levels were then predicted for milk from the dairy cattle raised at the farm. This pathway was only evaluated for dioxins/furans as these compounds are known to be extremely bioaccumulative and as such the compounds of most concern for this scenario. As part of the *residential* scenario, toddlers living in Kingsville have also been assumed to consume this milk.

The *produce consumer* scenario evaluates a person not living in the vicinity of the facilities whom consumes produce exclusively from the greenhouse facilities or produced at the neighbouring agricultural operations The produce consumer scenario was applied to receptor locations P1 (Asparagus Crop Land), P2 (Apple Orchard), P3 (Vineyards) and MAX POI (the proposed greenhouse facilities). For the on-site (MAX POI) scenario, produce concentrations were estimated assuming maximum on-site air concentrations were drawn into the greenhouses with these COCs then depositing on the vegetables grown within these facilities. Similarly, concentrations at neighbouring agricultural operations were estimated based on predicted air concentrations and deposition at respective receptor locations. The consumer scenario was applied to receptor locations P1 (Asparagus Crop Land), P2 (Apple Orchard), P3 (Vineyards) and MAX POI (the proposed greenhouse facilities).

3.4.2 Exposure Pathways

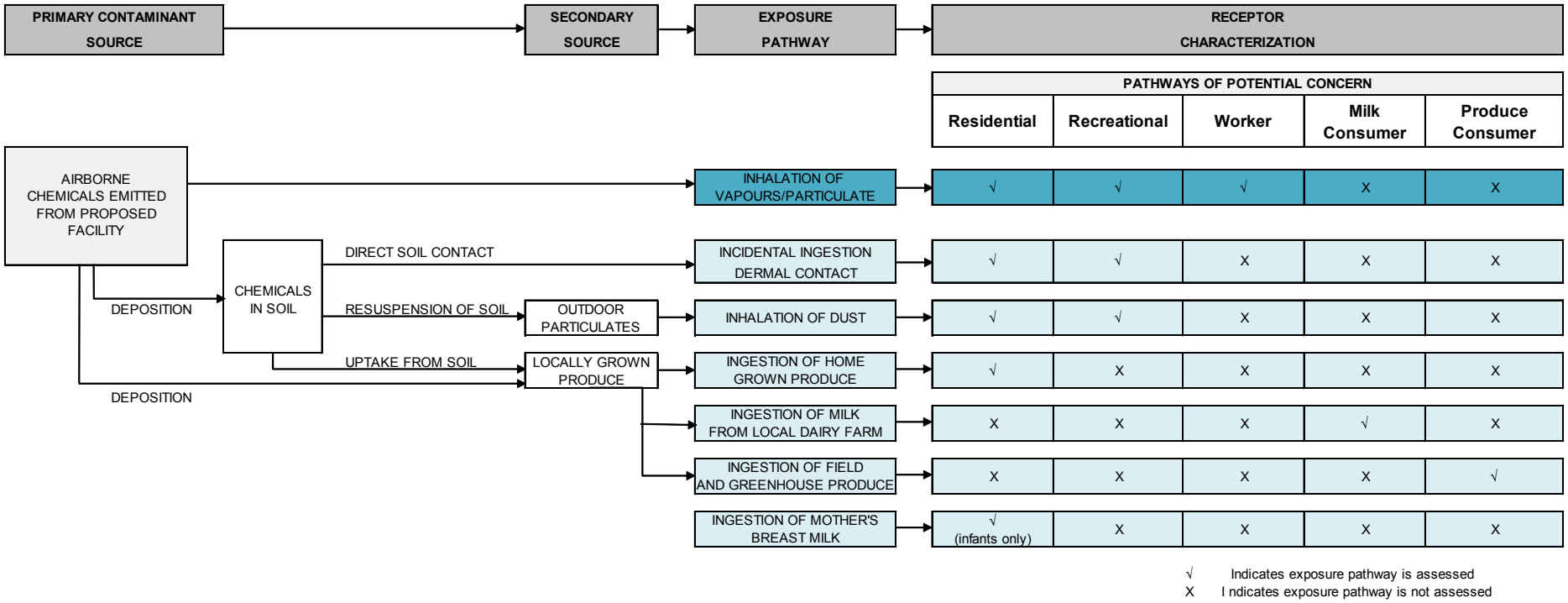
The primary exposure pathway under evaluation in the current assessment is the inhalation of the COCs by individuals living, working or playing in the surrounding community.

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

- **Incidental Ingestion of Soil and Dust**: Through typical indoor and outdoor activities, individuals may accidentally ingest soil and/or dust particles. Children are typically more susceptible to this exposure pathway, as they spend more time in contact with the ground, and are more likely to put soiled articles, such as toys or hands, into their mouths.
- **Incidental Inhalation of Indoor Dust**: Soils impacted by particles emitted from the proposed facility were assumed to be carried indoors (*e.g.*, by wind, or human and/pet activities) and present as indoor suspended dust for inhalation by individuals living within the home.
- **Dermal Exposure to Soils and Dusts**: Dermal exposures of human receptors may occur in both indoor and outdoor environments, through direct dermal contact with chemically impacted soil and dust.
- **Breast Milk Consumption (infants only)**: It is assumed that infants living at each of the sensitive receptor locations will be exposed to certain chemicals *via* their mother's breast milk. This exposure pathway was evaluated for those organic COCs, such as dioxins and furans, which have the potential to "bio-accumulate".
- **Ingestion of Locally Grown Produce**: Locally grown produce (such as vegetables and fruits grown in backyard gardens) may itself pose a source of exposure to some COCs. As chemicals are deposited from air-borne emissions, they may come into contact with leaves and fruit of crop plants, where they may remain as a surface contaminant, or actually be absorbed into the plant. Deposition of chemicals onto soil may also result in an accumulation in plants through root uptake. Due to the location of these facilities, in an agricultural area, it was assumed that residential receptors could obtain 100% of their vegetables locally.

Figure 3-4 is the Conceptual Site Model (CSM) used in the current assessment, and provides an overview of the sources of COCs and the exposure pathways associated with these sources. As noted in the CSM, for the sake of conservatism, each of the potential pathways and exposure assumptions typically associated with a residential scenario were evaluated at all receptor locations. For example, when considering multimedia exposures (*i.e.*, non-inhalation), individuals at each of the assessed receptor locations were assumed to spend 24 hours per day, 7 days per week, for 52 weeks per year at this location. This is obviously an overestimation of potential exposures for the schools. In the case of the Worker Scenario, the worker was assumed to be present on-site at the maximum ground-level air concentration for 8 hours per day, 5 days per week, 50 weeks per year.

In the case of the worker scenario, as the proposed gasifiers will be operated in compliance with all Occupation Health and Safety and Ministry of Labour regulations for the Province of Ontario, the assessment of occupational risks was considered beyond the scope of the current assessment. As such, the only pathways assessed for the worker were the inhalation of COCs.



Dark shading represents inhalation pathways; light shading represent multimedia pathways

Figure 3-4 Conceptual Site Model (CSM)

3.5 Cumulative Assessment

To assess the potential changes that would occur after the REMASCO equipment is used to replace existing boilers at the Southshore, Mucci, and Agriville greenhouses it is necessary to compare existing and future emissions from these greenhouses (Chandler, 2011). However, these facilities are only part of the existing greenhouses in the Kingsville area and together with residential and transportation related emissions they all contribute to the existing levels of contaminants in the air. Since existing ambient air quality data is not available from the immediate vicinity, the contributions of the existing greenhouses were estimated and modelled using standard emission factors (by fuel type). These results were combined with current air quality data for the nearby communities of Windsor and Chatham, collected by the Ontario Ministry of Environment, to represent cumulative air concentrations under current conditions. By using the same modelling procedures, only replacing the existing Southshore, Mucci and Agriville boilers with the proposed REMASCO facilities, the future levels were also predicted.

Cumulative risk of exposure to airborne contaminants was evaluated based on the ground-level air concentrations of selected COCs (*i.e.*, NO_2, $PM_{2.5}$) at each sensitive receptor location under both current conditions and future conditions (*i.e.*, conditions with the addition of the proposed REMASCO facilities). This evaluation was conducted for illustrative purposes and is only intended to address, at a high level, questions raised regarding the added impacts of facility emissions on local air quality. Estimates of background air concentrations were predicted for NO_2 and particulate matter, represented by $PM_{2.5}$, since emission factors for these COCs are high across all combustion fuel types used in local greenhouses (*i.e.,* coal, oil, wood, gas, REMASCO pelletized fuels) and thus, air concentrations of these COCs may be expected to be most heavily impacted by the addition and/or conversion of gasification units. This issue is further discussed in Chandler (2011).

4.0 EXPOSURE ASSESSMENT

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

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

Exposure estimation in the multi-pathway assessment portion of the HHRA was facilitated through the use of Intrinsik's integrated environmental risk assessment model. The model is spreadsheet based (Microsoft Excel™) but has a number of more advanced add-ons or features. Similar models have been used on hundreds of peer-reviewed HHRAs in Canada, including those conducted for contaminated sites, landfills, smelters, refineries, incinerators, and a variety of other industrial facilities. The current model version incorporates the techniques and procedures for exposure modelling developed by various regulatory agencies and published scientific literature sources and is capable of conducting complex exposure modelling involving all types of human receptors, and a myriad of exposure pathways and scenarios. The equations used in the current model, as well as a "worked example" of the calculations, are provided in Appendix B of this report.

4.1 Estimation of Ambient Ground-Level Air Concentrations

Estimates of the potential impacts on air quality related to emissions from the gasifiers were prepared from predicted ground-level air concentrations for each of the COCs. These were provided by Chandler (2011) at each of the receptor locations in the surrounding community, as well as at the maximum on-site location for the worker scenario. Ground-level air concentrations for each of the COCs were predicted for 1-hour, 24-hour, and annual average exposure durations, based upon the results of air dispersion modeling, taking into account emissions of the proposed gasifiers and typical meteorological information for the area.

Tables 4-1 through 4-3 provides a summary of the predicted ground-level air concentrations at each receptor location. When notation as many of the numerical values are well below 1.0. With scientific notation, any value expressed to the negative power (*i.e*., E-01) indicates a value less than 1.0, while a value expressed to a neutral (*i.e*., E+00) or positive power (*i.e*., E+01) indicate a number greater than 1.0. Therefore, in scientific notation, the value 0.000001 can be expressed as 1.0E-06 (or 1.0×10^{-6})

Table 4-1 1-Hour Ground Level Air Concentration (µg/m^3)														
COC	Receptor Location													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	7.1E+00	1.2E+00	3.1E+00	2.1E+00	1.6E+00	2.0E+00	1.8E+00	2.0E+00	2.8E+00	3.4E+00	1.4E+00	5.2E+00	3.2E+00	1.6E+00
Nitrogen Oxides (NO_x)	8.4E+01	1.5E+01	3.7E+01	2.6E+01	2.0E+01	2.3E+01	2.2E+01	2.4E+01	3.3E+01	4.1E+01	1.7E+01	6.2E+01	3.8E+01	1.9E+01
Hydrogen Chloride	1.1E+01	1.9E+00	4.7E+00	3.3E+00	2.5E+00	3.0E+00	2.8E+00	3.1E+00	4.3E+00	5.3E+00	2.2E+00	8.0E+00	4.9E+00	2.5E+00
Particulate Matter (PM_{10})	2.5E+00	4.5E-01	1.1E+00	7.7E-01	5.9E-01	7.1E-01	6.6E-01	7.2E-01	1.0E+00	1.2E+00	5.1E-01	1.9E+00	1.1E+00	5.8E-01
Particulate Matter ($PM_{2.5}$)	1.3E+00	2.2E-01	5.5E-01	3.9E-01	3.0E-01	3.5E-01	3.3E-01	3.6E-01	5.0E-01	6.2E-01	2.5E-01	9.4E-01	5.7E-01	2.9E-01
Inorganics														
Arsenic	1.4E-04	2.4E-05	5.9E-05	4.1E-05	3.1E-05	3.8E-05	3.5E-05	3.8E-05	5.3E-05	6.6E-05	2.7E-05	1.0E-04	6.1E-05	3.1E-05
Cadmium	2.3E-04	4.1E-05	1.0E-04	7.0E-05	5.4E-05	6.4E-05	6.0E-05	6.6E-05	9.1E-05	1.1E-04	4.6E-05	1.7E-04	1.0E-04	5.3E-05
Chromium	4.9E-03	8.7E-04	2.1E-03	1.5E-03	1.1E-03	1.4E-03	1.3E-03	1.4E-03	1.9E-03	2.4E-03	9.8E-04	3.6E-03	2.2E-03	1.1E-03
Lead	5.0E-04	8.9E-05	2.2E-04	1.5E-04	1.2E-04	1.4E-04	1.3E-04	1.4E-04	2.0E-04	2.4E-04	1.0E-04	3.7E-04	2.3E-04	1.2E-04
Mercury (Inorganic)	6.3E-04	1.1E-04	2.7E-04	1.9E-04	1.5E-04	1.7E-04	1.6E-04	1.8E-04	2.5E-04	3.0E-04	1.2E-04	4.6E-04	2.8E-04	1.4E-04
Organics														
Vinyl Chloride	1.0E-03	1.8E-04	4.4E-04	3.1E-04	2.3E-04	2.8E-04	2.6E-04	2.9E-04	4.0E-04	4.9E-04	2.0E-04	7.4E-04	4.5E-04	2.3E-04
Benzene	2.7E-03	4.8E-04	1.2E-03	8.2E-04	6.3E-04	7.5E-04	7.1E-04	7.7E-04	1.1E-03	1.3E-03	5.4E-04	2.0E-03	1.2E-03	6.2E-04
PAHs														
Benzo(a)pyrene	8.9E-05	1.6E-05	3.9E-05	2.7E-05	2.1E-05	2.5E-05	2.3E-05	2.5E-05	3.5E-05	4.3E-05	1.8E-05	6.6E-05	4.0E-05	2.0E-05
Dioxins / Furans														
Dioxins & Furans	8.2E-09	1.5E-09	3.6E-09	2.5E-09	1.9E-09	2.3E-09	2.1E-09	2.3E-09	3.2E-09	4.0E-09	1.6E-09	6.1E-09	3.7E-09	1.9E-09

Table 4-2 24-Hour Ground Level Air Concentration (µg/m^3)														
COC	Receptor Location													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	3.7E+00	2.1E-01	8.1E-01	1.3E+00	4.6E-01	1.2E+00	4.9E-01	3.9E-01	6.9E-01	2.1E+00	3.6E-01	1.6E+00	3.8E-01	5.7E-01
Nitrogen Oxides (NO_x)	4.5E+01	2.5E+00	9.7E+00	1.6E+01	5.5E+00	1.4E+01	5.9E+00	4.6E+00	8.2E+00	2.5E+01	4.3E+00	1.9E+01	4.5E+00	6.8E+00
Hydrogen Chloride	5.8E+00	3.3E-01	1.2E+00	2.0E+00	7.0E-01	1.8E+00	7.6E-01	5.9E-01	1.1E+00	3.2E+00	5.6E-01	2.4E+00	5.8E-01	8.7E-01
Particulate Matter (PM_{10})	1.3E+00	7.6E-02	2.9E-01	4.7E-01	1.6E-01	4.3E-01	1.8E-01	1.4E-01	2.5E-01	7.6E-01	1.3E-01	5.7E-01	1.4E-01	2.0E-01
Particulate Matter ($PM_{2.5}$)	6.7E-01	3.8E-02	1.5E-01	2.4E-01	8.2E-02	2.1E-01	8.9E-02	6.9E-02	1.2E-01	3.8E-01	6.5E-02	2.8E-01	6.8E-02	1.0E-01
Inorganics														
Arsenic	7.2E-05	4.1E-06	1.6E-05	2.5E-05	8.8E-06	2.3E-05	9.5E-06	7.4E-06	1.3E-05	4.1E-05	6.9E-06	3.0E-05	7.3E-06	1.1E-05
Cadmium	1.2E-04	6.9E-06	2.7E-05	4.3E-05	1.5E-05	3.9E-05	1.6E-05	1.3E-05	2.3E-05	6.9E-05	1.2E-05	5.2E-05	1.2E-05	1.9E-05
Chromium	2.6E-03	1.5E-04	5.7E-04	9.1E-04	3.2E-04	8.3E-04	3.4E-04	2.7E-04	4.8E-04	1.5E-03	2.5E-04	1.1E-03	2.7E-04	4.0E-04
Lead	2.7E-04	1.5E-05	5.8E-05	9.3E-05	3.3E-05	8.4E-05	3.5E-05	2.7E-05	4.9E-05	1.5E-04	2.6E-05	1.1E-04	2.7E-05	4.0E-05
Mercury (Inorganic)	3.3E-04	1.9E-05	7.2E-05	1.2E-04	4.1E-05	1.1E-04	4.4E-05	3.4E-05	6.1E-05	1.9E-04	3.2E-05	1.4E-04	3.4E-05	5.0E-05
Organics														
Vinyl Chloride	5.3E-04	3.0E-05	1.2E-04	1.9E-04	6.5E-05	1.7E-04	7.0E-05	5.5E-05	9.8E-05	3.0E-04	5.2E-05	2.2E-04	5.4E-05	8.1E-05
Benzene	1.4E-03	8.1E-05	3.1E-04	5.0E-04	1.8E-04	4.6E-04	1.9E-04	1.5E-04	2.6E-04	8.1E-04	1.4E-04	6.0E-04	1.5E-04	2.2E-04
PAHs														
Benzo(a)pyrene	4.7E-05	2.7E-06	1.0E-05	1.6E-05	5.8E-06	1.5E-05	6.2E-06	4.9E-06	8.7E-06	2.7E-05	4.6E-06	2.0E-05	4.8E-06	7.2E-06

Table 4-2 24-Hour Ground Level Air Concentration (µg/m^3)														
COC	*Receptor Location*													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Dioxins / Furans														
Dioxins & Furans	4.3E-09	2.5E-10	9.4E-10	1.5E-09	5.3E-10	1.4E-09	5.7E-10	4.5E-10	8.0E-10	2.5E-09	4.2E-10	1.8E-09	4.4E-10	6.6E-10

Table 4-3 Annual Average Ground Level Air Concentration (µg/m^3)														
COC	*Receptor Location*													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	2.0E-01	8.8E-03	6.7E-02	3.8E-02	1.3E-02	3.0E-02	3.2E-02	2.3E-02	5.4E-02	5.7E-02	1.8E-02	8.9E-02	1.9E-02	3.0E-02
Nitrogen Oxides (NO_x)	2.4E+00	1.1E-01	8.0E-01	4.5E-01	1.5E-01	3.5E-01	3.9E-01	2.8E-01	6.4E-01	6.8E-01	2.2E-01	1.1E+00	2.3E-01	3.6E-01
Hydrogen Chloride	3.1E-01	1.4E-02	1.0E-01	5.8E-02	2.0E-02	4.6E-02	5.0E-02	3.6E-02	8.3E-02	8.8E-02	2.8E-02	1.4E-01	2.9E-02	4.6E-02
Particulate Matter (PM_{10})	7.2E-02	3.2E-03	2.4E-02	1.4E-02	4.7E-03	1.1E-02	1.2E-02	8.4E-03	1.9E-02	2.1E-02	6.5E-03	3.2E-02	6.8E-03	1.1E-02
Particulate Matter ($PM_{2.5}$)	3.6E-02	1.6E-03	1.2E-02	6.8E-03	2.3E-03	5.3E-03	5.8E-03	4.2E-03	9.7E-03	1.0E-02	3.3E-03	1.6E-02	3.4E-03	5.4E-03
Inorganics														
Arsenic	3.8E-06	1.7E-07	1.3E-06	7.2E-07	2.5E-07	5.7E-07	6.2E-07	4.5E-07	1.0E-06	1.1E-06	3.5E-07	1.7E-06	3.6E-07	5.8E-07
Cadmium	6.6E-06	2.9E-07	2.2E-06	1.2E-06	4.3E-07	9.7E-07	1.1E-06	7.6E-07	1.8E-06	1.9E-06	5.9E-07	2.9E-06	6.2E-07	9.9E-07
Chromium	1.4E-04	6.2E-06	4.7E-05	2.6E-05	9.1E-06	2.1E-05	2.3E-05	1.6E-05	3.8E-05	4.0E-05	1.3E-05	6.3E-05	1.3E-05	2.1E-05
Lead	1.4E-05	6.3E-07	4.8E-06	2.7E-06	9.2E-07	2.1E-06	2.3E-06	1.7E-06	3.8E-06	4.1E-06	1.3E-06	6.4E-06	1.4E-06	2.1E-06
Mercury (Inorganic)	1.8E-05	7.8E-07	6.0E-06	3.3E-06	1.2E-06	2.6E-06	2.9E-06	2.1E-06	4.8E-06	5.1E-06	1.6E-06	7.9E-06	1.7E-06	2.7E-06
Organics														
Vinyl Chloride	2.9E-05	1.3E-06	9.6E-06	5.4E-06	1.9E-06	4.2E-06	4.6E-06	3.3E-06	7.7E-06	8.1E-06	2.6E-06	1.3E-05	2.7E-06	4.3E-06
Benzene	7.7E-05	3.4E-06	2.6E-05	1.4E-05	5.0E-06	1.1E-05	1.2E-05	8.9E-06	2.1E-05	2.2E-05	6.9E-06	3.4E-05	7.3E-06	1.2E-05
PAHs														
Benzo(a)pyrene	2.5E-06	1.1E-07	8.5E-07	4.7E-07	1.6E-07	3.7E-07	4.1E-07	2.9E-07	6.8E-07	7.2E-07	2.3E-07	1.1E-06	2.4E-07	3.8E-07
Dioxins / Furans														
Dioxins & Furans	2.3E-10	1.0E-11	7.8E-11	4.4E-11	1.5E-11	3.4E-11	3.8E-11	2.7E-11	6.3E-11	6.6E-11	2.1E-11	1.0E-10	2.2E-11	3.5E-11

4.2 Estimation of Soil and Home Garden Produce Concentrations

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

Total deposition into the environment (*e.g*., soil) was provided in total, wet, and dry deposition per year at each receptor location by Chandler (2011). Deposition rates used in the HHRA are provided in Table 4-4. These deposition fluxes were used to estimate the concentrations in multiple environmental media, as described in Appendix B of this report.

Table 4-4 Annual Deposition Rate (g/m^2)

COC	*Receptor Location*													
	MAX	**C1**	**C2**	**C3**	**C4**	**P1**	**P2**	**P3**	**R1**	**R2**	**R3**	**R4**	**R5**	**R6**
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Nitrogen Oxides (NO_x)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hydrogen Chloride	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Particulate Matter (PM_{10})	7.6E-03	2.0E-04	3.3E-03	9.1E-04	2.7E-04	1.2E-03	6.6E-04	5.1E-04	2.9E-03	1.9E-03	4.3E-04	4.0E-03	4.6E-04	6.8E-04
Particulate Matter ($PM_{2.5}$)	3.8E-03	1.0E-04	1.6E-03	4.6E-04	1.3E-04	6.0E-04	3.3E-04	2.5E-04	1.5E-03	9.5E-04	2.1E-04	2.0E-03	2.3E-04	3.4E-04
Inorganics														
Arsenic	4.1E-07	1.1E-08	1.7E-07	4.9E-08	1.4E-08	6.4E-08	3.5E-08	2.7E-08	1.5E-07	1.0E-07	2.3E-08	2.1E-07	2.4E-08	3.6E-08
Cadmium	6.9E-07	1.9E-08	3.0E-07	8.3E-08	2.4E-08	1.1E-07	6.0E-08	4.6E-08	2.6E-07	1.7E-07	3.9E-08	3.6E-07	4.1E-08	6.2E-08
Chromium	1.5E-05	4.0E-07	6.3E-06	1.8E-06	5.2E-07	2.3E-06	1.3E-06	9.8E-07	5.6E-06	3.7E-06	8.3E-07	7.8E-06	8.8E-07	1.3E-06
Lead	1.5E-06	4.0E-08	6.5E-07	1.8E-07	5.2E-08	2.4E-07	1.3E-07	1.0E-07	5.7E-07	3.8E-07	8.4E-08	7.9E-07	9.0E-08	1.3E-07
Mercury (Inorganic)	1.9E-06	5.0E-08	8.0E-07	2.2E-07	6.5E-08	3.0E-07	1.6E-07	1.2E-07	7.2E-07	4.7E-07	1.0E-07	9.9E-07	1.1E-07	1.7E-07
Organics														
Vinyl Chloride	3.0E-06	8.1E-08	1.3E-06	3.6E-07	1.1E-07	4.8E-07	2.6E-07	2.0E-07	1.2E-06	7.6E-07	1.7E-07	1.6E-06	1.8E-07	2.7E-07
Benzene	8.1E-06	2.2E-07	3.5E-06	9.7E-07	2.8E-07	1.3E-06	7.0E-07	5.4E-07	3.1E-06	2.0E-06	4.5E-07	4.3E-06	4.9E-07	7.2E-07
PAHs														
Benzo(a)pyrene	2.7E-07	7.1E-09	1.1E-07	3.2E-08	9.3E-09	4.2E-08	2.3E-08	1.8E-08	1.0E-07	6.7E-08	1.5E-08	1.4E-07	1.6E-08	2.4E-08
Dioxins / Furans														
Dioxins & Furans	2.5E-11	6.6E-13	1.1E-11	2.9E-12	8.6E-13	3.9E-12	2.1E-12	1.6E-12	9.4E-12	6.2E-12	1.4E-12	1.3E-11	1.5E-12	2.2E-12

Environmental media concentrations, including soil and vegetation, were estimated based on these facility deposition rates. These media concentrations were then used to estimate the health risks to receptors from oral and dermal contact in a multiple pathway risk assessment. Table 4-5 provides a summary of the estimated environmental media COC concentrations at the MAX POI. Estimated environmental media COC concentrations for all other receptor locations are presented in Appendix B

These media concentrations, predicted for each relevant receptor location in the surrounding community, were used to estimate potential inhalation, oral and dermal exposures. The "worst-case" predicted exposures under the residential and recreational/community scenarios (receptor location R4) are presented in Table 4-6, and "worst-case" oral (ingestion) exposure estimates under the produce consumer scenario (MAX POI) are presented in Table 4-7. The produce consumer scenario assumed the MAX POI deposition at the location of air intakes for a greenhouse and deposition of these maximum values onto produce within the greenhouse. Refer to Appendix B for residential, recreational/community, and produce consumer scenario exposure estimates at all other receptor locations and a complete "worked example" of the assumptions and equations used to complete the multi-pathway portion of the current assessment.

Table 4-5 Summary of Chemical Concentrations Used to Estimate Exposures

Receptor Location	Chemical	Soil	Surface Soil	Air	Dust	Deposition	Plant Tissue Concentrations			
							Deposition	Air	Soil	Total
		mg/kg	mg/kg	µg/m^3	µg/m^3	mg/m^2/yr	mg/kg ww	mg/kg ww	mg/kg ww	mg/kg ww
Max POI	Arsenic	3.91E-05	3.91E-04	3.84E-06	2.97E-10	4.06E-04	5.56E-07	0.00E+00	3.71E-08	5.93E-07
Max POI	Cadmium	6.67E-05	6.67E-04	6.55E-06	5.07E-10	6.93E-04	9.49E-07	0.00E+00	1.25E-06	2.20E-06
Max POI	Chromium	1.42E-03	1.42E-02	1.40E-04	1.08E-08	1.48E-02	2.02E-05	0.00E+00	1.04E-06	2.13E-05
Max POI	Lead	1.45E-04	1.45E-03	1.42E-05	1.10E-09	1.50E-03	2.06E-06	0.00E+00	2.96E-07	2.36E-06
Max POI	Mercury	1.80E-04	1.80E-03	1.77E-05	1.37E-09	1.87E-03	2.57E-06	3.19E-06	2.44E-05	3.01E-05
Max POI	Benzene	1.98E-12	1.98E-11	7.67E-05	1.50E-17	8.12E-03	1.11E-05	1.59E-11	7.02E-13	1.11E-05
Max POI	Vinyl Chloride	2.83E-14	2.83E-13	2.85E-05	2.15E-19	3.02E-03	4.13E-06	2.21E-13	2.55E-14	4.13E-06
Max POI	Dioxins & Furans (TEQ)	2.35E-10	2.35E-09	2.32E-10	1.78E-15	2.46E-08	3.37E-11	5.52E-12	1.60E-15	3.92E-11
Max POI	Benzo(a)pyrene	1.66E-06	1.66E-05	2.52E-06	1.26E-11	2.67E-04	3.65E-07	1.51E-07	2.76E-11	5.17E-07

Table 4-6 Summary of Predicted Off-Site Residential and Recreational/Community Human Exposures

Location	Receptor	COC	Estimated Daily Intake (EDI) µg/kg-day										
			Ambient Air	Soil	Dust	Plant	Berries	Root	Dermal (Hands)	Dermal (Other)[a]	Breast Milk[b]	Total EDI	Total EDI (incl air)
R4	Infant	Arsenic	4.61E-07	7.53E-07	4.20E-11	0.00E+00	0.00E+00	0.00E+00	2.41E-08	1.10E-08	-	7.88E-07	1.25E-06
R4	Toddler	Arsenic	8.64E-07	2.49E-06	7.87E-11	1.27E-06	5.04E-08	1.57E-07	1.61E-08	9.65E-09	-	4.00E-06	4.86E-06
R4	Child	Arsenic	7.57E-07	3.13E-07	6.89E-11	9.30E-07	4.46E-08	1.21E-07	1.11E-08	8.54E-09	-	1.43E-06	2.18E-06
R4	Adolescent	Arsenic	4.49E-07	1.72E-07	4.09E-11	6.27E-07	2.03E-08	9.39E-08	8.27E-09	7.45E-09	-	9.30E-07	1.38E-06
R4	Adult	Arsenic	4.03E-07	1.46E-07	3.67E-11	6.05E-07	1.41E-08	6.57E-08	7.77E-09	7.18E-09	-	8.45E-07	1.25E-06
R4	Infant	Cadmium	7.87E-07	1.29E-06	7.17E-11	0.00E+00	0.00E+00	0.00E+00	1.37E-08	6.26E-09	-	1.31E-06	2.09E-06
R4	Toddler	Cadmium	1.48E-06	4.26E-06	1.34E-10	4.71E-06	1.70E-06	2.15E-06	9.16E-09	5.50E-09	-	1.28E-05	1.43E-05
R4	Child	Cadmium	1.29E-06	5.34E-07	1.18E-10	3.45E-06	1.50E-06	1.65E-06	6.30E-09	4.86E-09	-	7.15E-06	8.45E-06
R4	Adolescent	Cadmium	7.67E-07	2.94E-07	6.98E-11	2.33E-06	6.83E-07	1.28E-06	4.71E-09	4.24E-09	-	4.60E-06	5.36E-06
R4	Adult	Cadmium	6.89E-07	2.49E-07	6.27E-11	2.25E-06	4.76E-07	8.97E-07	4.42E-09	4.09E-09	-	3.88E-06	4.56E-06
R4	Infant	Chromium	1.68E-05	2.74E-05	1.53E-09	0.00E+00	0.00E+00	0.00E+00	2.92E-06	1.33E-06	-	3.17E-05	4.84E-05
R4	Toddler	Chromium	3.15E-05	9.08E-05	2.86E-09	4.55E-05	1.42E-06	3.22E-06	1.95E-06	1.17E-06	-	1.44E-04	1.76E-04
R4	Child	Chromium	2.76E-05	1.14E-05	2.51E-09	3.34E-05	1.25E-06	2.47E-06	1.34E-06	1.04E-06	-	5.09E-05	7.84E-05
R4	Adolescent	Chromium	1.63E-05	6.27E-06	1.49E-09	2.25E-05	5.68E-07	1.92E-06	1.00E-06	9.03E-07	-	3.32E-05	4.95E-05
R4	Adult	Chromium	1.47E-05	5.30E-06	1.34E-09	2.17E-05	3.96E-07	1.34E-06	9.43E-07	8.71E-07	-	3.06E-05	4.52E-05

Table 4-6 Summary of Predicted Off-Site Residential and Recreational/Community Human Exposures

Location	Receptor	COC	Estimated Daily Intake (EDI) µg/kg-day										
			Ambient Air	Soil	Dust	Plant	Berries	Root	Dermal (Hands)	Dermal (Other)[a]	Breast Milk[b]	Total EDI	Total EDI (incl air)
R4	Infant	Lead	1.71E-06	2.79E-06	1.56E-10	0.00E+00	0.00E+00	0.00E+00	1.79E-08	8.15E-09	-	2.82E-06	4.53E-06
R4	Toddler	Lead	3.20E-06	9.25E-06	2.92E-10	5.04E-06	4.02E-07	6.55E-07	1.19E-08	7.16E-09	-	1.54E-05	1.86E-05
R4	Child	Lead	2.81E-06	1.16E-06	2.56E-10	3.70E-06	3.55E-07	5.04E-07	8.21E-09	6.33E-09	-	5.73E-06	8.54E-06
R4	Adolescent	Lead	1.66E-06	6.39E-07	1.52E-10	2.49E-06	1.61E-07	3.92E-07	6.13E-09	5.52E-09	-	3.70E-06	5.36E-06
R4	Adult	Lead	1.50E-06	5.40E-07	1.36E-10	2.40E-06	1.13E-07	2.74E-07	5.76E-09	5.32E-09	-	3.34E-06	4.84E-06
R4	Infant	Mercury	2.13E-06	3.48E-06	1.94E-10	0.00E+00	0.00E+00	0.00E+00	3.71E-07	1.69E-07	-	4.02E-06	6.14E-06
R4	Toddler	Mercury	3.99E-06	1.15E-05	3.63E-10	6.34E-05	3.31E-05	1.81E-05	2.48E-07	1.49E-07	-	1.27E-04	1.31E-04
R4	Child	Mercury	3.50E-06	1.44E-06	3.18E-10	4.65E-05	2.93E-05	1.40E-05	1.70E-07	1.31E-07	-	9.15E-05	9.50E-05
R4	Adolescent	Mercury	2.07E-06	7.96E-07	1.89E-10	3.14E-05	1.33E-05	1.08E-05	1.27E-07	1.15E-07	-	5.65E-05	5.86E-05
R4	Adult	Mercury	1.86E-06	6.72E-07	1.70E-10	3.02E-05	9.28E-06	7.58E-06	1.20E-07	1.10E-07	-	4.80E-05	4.99E-05
R4	Infant	Benzene	9.22E-06	3.81E-14	2.12E-18	0.00E+00	0.00E+00	0.00E+00	1.22E-15	5.56E-16	2.80E-09	2.80E-09	9.22E-06
R4	Toddler	Benzene	1.73E-05	1.26E-13	3.98E-18	2.38E-05	9.54E-13	7.95E-11	8.13E-16	4.88E-16	-	2.38E-05	4.10E-05
R4	Child	Benzene	1.51E-05	1.58E-14	3.48E-18	1.74E-05	8.44E-13	6.12E-11	5.60E-16	4.32E-16	-	1.74E-05	3.26E-05
R4	Adolescent	Benzene	8.98E-06	8.71E-15	2.07E-18	1.18E-05	3.83E-13	4.75E-11	4.18E-16	3.76E-16	-	1.18E-05	2.07E-05
R4	Adult	Benzene	8.07E-06	7.36E-15	1.86E-18	1.13E-05	2.67E-13	3.32E-11	3.93E-16	3.63E-16	-	1.13E-05	1.94E-05
R4	Infant	Vinyl Chloride	3.43E-06	5.46E-16	3.04E-20	0.00E+00	0.00E+00	0.00E+00	1.75E-17	7.97E-18	2.08E-10	2.08E-10	3.43E-06
R4	Toddler	Vinyl Chloride	6.43E-06	1.81E-15	5.70E-20	8.84E-06	3.47E-14	3.50E-12	1.17E-17	7.00E-18	-	8.84E-06	1.53E-05
R4	Child	Vinyl Chloride	5.63E-06	2.27E-16	5.00E-20	6.48E-06	3.07E-14	2.69E-12	8.03E-18	6.19E-18	-	6.48E-06	1.21E-05
R4	Adolescent	Vinyl Chloride	3.34E-06	1.25E-16	2.96E-20	4.38E-06	1.39E-14	2.09E-12	6.00E-18	5.40E-18	-	4.38E-06	7.71E-06
R4	Adult	Vinyl Chloride	3.00E-06	1.05E-16	2.66E-20	4.22E-06	9.72E-15	1.46E-12	5.63E-18	5.20E-18	-	4.22E-06	7.22E-06
R4	Infant	Dioxins	2.79E-11	4.52E-12	2.52E-16	0.00E+00	0.00E+00	0.00E+00	1.45E-13	6.60E-14	2.10E-09	2.10E-09	2.13E-09
R4	Toddler	Dioxins	5.23E-11	1.50E-11	4.72E-16	8.20E-11	2.18E-15	1.21E-12	9.66E-14	5.79E-14	-	9.84E-11	3.76E-09
R4	Child	Dioxins	4.59E-11	1.88E-12	4.14E-16	6.02E-11	1.92E-15	9.34E-13	6.64E-14	5.12E-14	-	6.31E-11	1.09E-10
R4	Adolescent	Dioxins	2.72E-11	1.03E-12	2.45E-16	4.06E-11	8.74E-16	7.26E-13	4.97E-14	4.47E-14	-	4.25E-11	6.96E-11
R4	Adult	Dioxins	2.44E-11	8.73E-13	2.20E-16	3.91E-11	6.10E-16	5.07E-13	4.66E-14	4.31E-14	-	4.06E-11	6.50E-11
R4	Infant	B(a)P	3.03E-07	3.19E-08	1.78E-12	0.00E+00	0.00E+00	0.00E+00	2.86E-09	1.30E-09	1.20E-06	1.24E-06	1.54E-06
R4	Toddler	B(a)P	5.68E-07	1.06E-07	3.33E-12	1.06E-06	3.74E-11	1.36E-09	1.91E-09	1.15E-09	-	1.17E-06	1.73E-06

Table 4-6 Summary of Predicted Off-Site Residential and Recreational/Community Human Exposures

Location	Receptor	COC	Estimated Daily Intake (EDI) µg/kg-day										
			Ambient Air	Soil	Dust	Plant	Berries	Root	Dermal (Hands)	Dermal (Other)[a]	Breast Milk[b]	Total EDI	Total EDI (incl air)
R4	Child	B(a)P	4.98E-07	1.33E-08	2.92E-12	7.75E-07	3.31E-11	1.05E-09	1.31E-09	1.01E-09	-	7.92E-07	1.29E-06
R4	Adolescent	B(a)P	2.95E-07	7.30E-09	1.73E-12	5.23E-07	1.50E-11	8.14E-10	9.82E-10	8.84E-10	-	5.33E-07	8.28E-07
R4	Adult	B(a)P	2.65E-07	6.17E-09	1.56E-12	5.04E-07	1.05E-11	5.69E-10	9.22E-10	8.52E-10	-	5.13E-07	7.78E-07

B(a)P Benzo(a)pyrene
Dioxins Dioxins & Furans (TEQ)
\- Exposure pathway is not relevant to the particular receptor, COC, or exposure scenario.
[a] Dermal exposure calculated based on exposed surface area of the arms and legs.
[b] Exposure *via* consumption of breast milk by an infant was calculated for those COCs for which a bio-tranfer factor (µg/kg-milk) is available; exposure *via* consumption of breast milk was not considered a relevant route of exposure under the recreational/community scenario (*i.*e., receptor locations C1, C2, C3, and C4).

Table 4-7 Summary of Predicted Ingestion Exposures from Consumption of Produce Grown in an On-site Greenhouse (MAX POI)			
Location	***Receptor***	***COC***	***Estimated Daily Intake (EDI) (µg/kg-day)***
Max POI	Infant	Arsenic	-
Max POI	Toddler	Arsenic	2.41E-06
Max POI	Child	Arsenic	1.77E-06
Max POI	Adolescent	Arsenic	1.19E-06
Max POI	Adult	Arsenic	1.15E-06
Max POI	Infant	Cadmium	-
Max POI	Toddler	Cadmium	8.94E-06
Max POI	Child	Cadmium	6.56E-06
Max POI	Adolescent	Cadmium	4.42E-06
Max POI	Adult	Cadmium	4.26E-06
Max POI	Infant	Chromium	-
Max POI	Toddler	Chromium	8.64E-05
Max POI	Child	Chromium	6.34E-05
Max POI	Adolescent	Chromium	4.28E-05
Max POI	Adult	Chromium	4.12E-05
Max POI	Infant	Lead	-
Max POI	Toddler	Lead	9.57E-06
Max POI	Child	Lead	7.02E-06
Max POI	Adolescent	Lead	4.74E-06
Max POI	Adult	Lead	4.57E-06
Max POI	Infant	Mercury	-
Max POI	Toddler	Mercury	1.22E-04
Max POI	Child	Mercury	8.97E-05
Max POI	Adolescent	Mercury	6.05E-05
Max POI	Adult	Mercury	5.84E-05
Max POI	Infant	Benzene	-
Max POI	Toddler	Benzene	4.51E-05
Max POI	Child	Benzene	3.31E-05
Max POI	Adolescent	Benzene	2.23E-05
Max POI	Adult	Benzene	2.15E-05
Max POI	Infant	Vinyl Chloride	-
Max POI	Toddler	Vinyl Chloride	1.68E-05
Max POI	Child	Vinyl Chloride	1.23E-05
Max POI	Adolescent	Vinyl Chloride	8.31E-06
Max POI	Adult	Vinyl Chloride	8.01E-06
Max POI	Infant	Dioxins & Furans (TEQ)	-
Max POI	Toddler	Dioxins & Furans (TEQ)	1.59E-10
Max POI	Child	Dioxins & Furans (TEQ)	1.17E-10
Max POI	Adolescent	Dioxins & Furans (TEQ)	7.88E-11
Max POI	Adult	Dioxins & Furans (TEQ)	7.59E-11
Max POI	Infant	Benzo(a)pyrene	-
Max POI	Toddler	Benzo(a)pyrene	2.10E-06

Table 4-7 Summary of Predicted Ingestion Exposures from Consumption of Produce Grown in an On-site Greenhouse (MAX POI)			
Location	***Receptor***	***COC***	***Estimated Daily Intake (EDI) (µg/kg-day)***
Max POI	Child	Benzo(a)pyrene	1.54E-06
Max POI	Adolescent	Benzo(a)pyrene	1.04E-06
Max POI	Adult	Benzo(a)pyrene	1.00E-06

- Exposure pathway is not relevant to the particular receptor.

4.3 Exposure Analysis of Particulate Matter

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

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

The potential health impact of ultrafine particulate matter (*i.e.*, $PM_{0.1}$) is an emerging area of scientific enquiry. Currently there are no established regulatory benchmarks or standardized approaches to evaluation of the health impact related to exposures to this particulate matter fraction. For the current assessment, the ultrafine fraction was considered as part of the evaluation of health impacts related to the $PM_{2.5}$ (*i.e.*, particulate matter less than 2.5 microns in size) group.

4.4 Cumulative Air Concentrations

Predicted cumulative air concentrations of NO_2 and $PM_{2.5}$ under current conditions and future conditions with the addition of the proposed REMASCO facilities are presented in Table 4-8, along with predicted air concentrations attributed to the proposed facilities alone. As indicated in the table, there will be a net benefit in air quality with the installation and the operation of the REMASCO facilities.

Table 4-8 Predicted Air Concentrations under Various Operating Scenarios					
Location	*COC*	*Duration*	*Predicted Air Concentration (µg/m^3)*		
			Cumulative: Current Conditions[a,b]	*REMASCO Facilities Alone*	*Cumulative: Future Conditions with REMASCO*
R1	NO_2	1-hour	1.98E+02	3.34E+01	1.64E+02
R1	NO_2	24-hour	1.84E+02	8.16E+00	1.27E+02
R1	NO_2	Annual	4.20E+01	6.40E-01	2.87E+01
R1	$PM_{2.5}$	1-hour	-	3.60E-01	-
R1	$PM_{2.5}$	24-hour	1.29E+02	7.90E-02	7.28E+01
R1	$PM_{2.5}$	Annual	2.02E+01	2.70E-03	1.37E+01
R2	NO_2	1-hour	2.26E+02	3.57E+01	1.98E+02
R2	NO_2	24-hour	1.81E+02	2.02E+01	1.43E+02
R2	NO_2	Annual	4.99E+01	7.20E-01	3.32E+01
R2	$PM_{2.5}$	1-hour	-	6.70E-01	-
R2	$PM_{2.5}$	24-hour	1.35E+02	3.91E-01	8.50E+01
R2	$PM_{2.5}$	Annual	2.56E+01	1.43E-02	1.56E+01
R3	NO_2	1-hour	2.01E+02	9.11E+00	1.68E+02
R3	NO_2	24-hour	1.55E+02	3.30E+00	1.42E+02
R3	NO_2	Annual	3.01E+01	1.90E-01	2.86E+01
R3	$PM_{2.5}$	1-hour	-	2.70E-01	-
R3	$PM_{2.5}$	24-hour	9.21E+01	7.40E-02	7.03E+01
R3	$PM_{2.5}$	Annual	1.31E+01	1.10E-03	1.23E+01
C1	NO_2	1-hour	1.80E+02	1.28E+01	1.65E+02
C1	NO_2	24-hour	1.14E+02	2.54E+00	1.01E+02
C1	NO_2	Annual	2.52E+01	1.10E-01	2.45E+01
C1	$PM_{2.5}$	1-hour	-	1.90E-01	-
C1	$PM_{2.5}$	24-hour	4.87E+01	5.30E-02	4.29E+01
C1	$PM_{2.5}$	Annual	1.02E+01	2.10E-03	9.77E+00
C2	NO_2	1-hour	1.79E+02	4.43E+01	1.76E+02
C2	NO_2	24-hour	1.35E+02	1.41E+01	1.44E+02
C2	NO_2	Annual	3.72E+01	1.12E+00	3.75E+01
C2	$PM_{2.5}$	1-hour	-	8.60E-01	-
C2	$PM_{2.5}$	24-hour	6.50E+01	2.80E-01	6.04E+01
C2	$PM_{2.5}$	Annual	1.73E+01	1.37E-02	1.42E+01
R4	NO_2	1-hour	1.80E+02	7.63E+01	1.79E+02
R4	NO_2	24-hour	1.58E+02	2.40E+01	1.29E+02
R4	NO_2	Annual	4.31E+01	1.22E+00	3.30E+01
R4	$PM_{2.5}$	1-hour	-	1.53E+00	-
R4	$PM_{2.5}$	24-hour	8.41E+01	7.99E-01	7.70E+01
R4	$PM_{2.5}$	Annual	2.07E+01	1.81E-02	1.70E+01
C3	NO_2	1-hour	2.15E+02	2.47E+01	1.66E+02
C3	NO_2	24-hour	1.47E+02	1.19E+01	1.56E+02
C3	NO_2	Annual	3.10E+01	4.30E-01	3.12E+01
C3	$PM_{2.5}$	1-hour	-	3.70E-01	-
C3	$PM_{2.5}$	24-hour	1.02E+02	2.24E-01	6.68E+01
C3	$PM_{2.5}$	Annual	1.39E+01	7.00E-03	1.19E+01
C4	NO_2	1-hour	2.01E+02	1.54E+01	1.63E+02
C4	NO_2	24-hour	1.25E+02	5.15E+00	1.14E+02

Table 4-8 Predicted Air Concentrations under Various Operating Scenarios					
Location	*COC*	*Duration*	*Predicted Air Concentration (µg/m^3)*		
			Cumulative: Current Conditions[a,b]	*REMASCO Facilities Alone*	*Cumulative: Future Conditions with REMASCO*
C4	NO_2	Annual	2.51E+01	1.40E-01	2.42E+01
C4	$PM_{2.5}$	1-hour	-	2.90E-01	-
C4	$PM_{2.5}$	24-hour	6.97E+01	1.06E-01	5.99E+01
C4	$PM_{2.5}$	Annual	1.03E+01	3.30E-03	9.72E+00
R5	NO_2	1-hour	1.83E+02	2.79E+01	1.83E+02
R5	NO_2	24-hour	1.33E+02	4.55E+00	1.31E+02
R5	NO_2	Annual	3.14E+01	2.30E-01	2.95E+01
R5	$PM_{2.5}$	1-hour	-	3.60E-01	-
R5	$PM_{2.5}$	24-hour	6.76E+01	8.10E-02	5.95E+01
R5	$PM_{2.5}$	Annual	1.46E+01	4.60E-03	1.32E+01
P1	NO_2	1-hour	2.08E+02	2.34E+01	1.80E+02
P1	NO_2	24-hour	1.52E+02	1.41E+01	1.51E+02
P1	NO_2	Annual	3.67E+01	3.50E-01	3.31E+01
P1	$PM_{2.5}$	1-hour	-	3.70E-01	-
P1	$PM_{2.5}$	24-hour	9.30E+01	2.39E-01	9.29E+01
P1	$PM_{2.5}$	Annual	1.83E+01	5.60E-03	1.59E+01
P2	NO_2	1-hour	1.94E+02	2.20E+01	1.77E+02
P2	NO_2	24-hour	1.59E+02	5.89E+00	1.44E+02
P2	NO_2	Annual	3.90E+01	3.70E-01	3.70E+01
P2	$PM_{2.5}$	1-hour	-	3.50E-01	-
P2	$PM_{2.5}$	24-hour	1.14E+02	1.21E-01	8.78E+01
P2	$PM_{2.5}$	Annual	1.90E+01	8.10E-03	1.30E+01
P3	NO_2	1-hour	1.78E+02	2.38E+01	1.69E+02
P3	NO_2	24-hour	1.23E+02	4.25E+00	1.13E+02
P3	NO_2	Annual	3.16E+01	2.80E-01	2.97E+01
P3	$PM_{2.5}$	1-hour	-	3.00E-01	-
P3	$PM_{2.5}$	24-hour	7.21E+01	8.40E-02	6.17E+01
P3	$PM_{2.5}$	Annual	1.46E+01	5.80E-03	1.32E+01
R6	NO_2	1-hour	1.88E+02	2.25E+01	1.88E+02
R6	NO_2	24-hour	1.76E+02	6.29E+00	1.76E+02
R6	NO_2	Annual	3.84E+01	3.60E-01	3.52E+01
R6	$PM_{2.5}$	1-hour	-	4.10E-01	-
R6	$PM_{2.5}$	24-hour	1.02E+02	1.23E-01	1.02E+02
R6	$PM_{2.5}$	Annual	2.10E+01	8.00E-03	1.62E+01

[a] 1-hour NO_2 based upon the 90th percentile; 24-hour NO_2 based upon maximum 24h value from the monitoring data; 24-hour $PM_{2.5}$ based upon 90th percentile; annual NO_2 and $PM_{2.5}$ values represent annual means.

[b] Predicted existing air concentrations are inclusive of air emissions attributed to existing gasification units (*i.e.*, not using REMASCO pelletized fuels) at Agriville Farms Ltd. (Agriville) and Southshore Greenhouse Inc./Mucci Farms locations.

5.0 HAZARD ASSESSMENT

The following section provides the acute and chronic TRVs for each of the COCs evaluated in the current assessment. TRVs that are not consistent with those utilized by MOE in Reg. 153 (MOE, 2009a) and Reg. 419 (MOE, 2008) are shaded. In cases where values differ from those utilized by MOE, or in cases where TRV values are not provide by MOE (2008, 2009a), toxicological profiles, including a detailed discussion of the relevant information supporting the selected TRV, are provided Appendix A. It should be noted that in some cases short-term TRVs were utilized from recognized regulatory agencies such as AENV, TCEQ, and Cal EPA. In these cases, supplemental information is not provided in Appendix A since TRVs were utilized as reported by these agencies.

5.1 Acute Toxicity Reference Values

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

Table 5-1 Summary of Available Acute Non-carcinogenic Inhalation TRVs

Chemical of Concern	*Duration*	*Reported Exposure Limit ($\mu g/m^3$)*	*Critical Effect*	*Source*
Criteria Air Contaminants (CACs)				
Hydrogen Chloride	1-hour	75	Health Based	AENV AAQO, 2007
	24-hour	20	Health effects (based on chronic endpoints)	MOE, 2008
Oxides of Nitrogen (NO_x)	1-hour	200	Effects in the pulmonary function of asthmatics	WHO, 2005
	24-hour	200	Respiratory irritant	MOE, 2008
PM_{10}	24-hour	50	Lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase	WHO, 2005
$PM_{2.5}$	24-hour	25	Lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase	WHO, 2005
Sulphur Dioxide (SO_2)	1-hour	450	Health and environmental effects	Health Canada, 2006
	24-hour	20	Respiratory irritant	WHO, 2005
Inorganics				
Arsenic	1-hour	0.2	Decreased fetal weight in mice	Cal EPA, 2008
	24-hour	0.3	Irritation, sensitization, immunosuppression, teratogenesis, genotoxicity and carcinogenicity in exposed individuals	MOE, 2008
Cadmium	1-hour	0.1	Kidney Damage	TCEQ, 2008
	24-hour	0.025	Health effects (based on chronic endpoints)	MOE, 2008
Chromium (total)	1-hour	1	Health Based	TCEQ, 2008
	24-hour	1.5	Respiratory effects	MOE, 2008
Lead	1-hour	1.5	Impairment of hematopoietic system	AENV AAQO, 2007
	24-hour	0.5	Blood lead level of 5 µg/dL - based on neurological effects in children; weight of evidence	MOE, 2008

Table 5-1 Summary of Available Acute Non-carcinogenic Inhalation TRVs				
Chemical of Concern	***Duration***	***Reported Exposure Limit ($\mu g/m^3$)***	***Critical Effect***	***Source***
Mercury (inorganic)	1-hour	0.6	CNS disturbances in rat off-spring	Cal EPA, 2008
	24-hour	2.0	Health effects (based on chronic endpoints)	MOE, 2008
Volatile Organic Compounds (VOCs)				
Benzene	1-hour	170	Depressed peripheral lymphocytes and depressed mitogeninduced blastogenesis of femoral B-lymphocytes in mice	TCEQ, 2008
	24-hour	29	Reduces lymphocyte proliferation following mitogen stimulation	ATSDR 2008
Vinyl Chloride	1-hour	20,000	Mild headache and dryness of eyes and nose	TCEQ, 2008
	24-hour	1	Health effects (based on chronic endpoints)	MOE, 2008
Carcinogenic PAHs				
Benzo(a)pyrene	1-hour	0.001	Health Based	MOE, 2008
	24-hour	0.0011	Health effects	MOE, 2008
Dioxins/Furans				
Dioxins & Furans	24-hour	5E-06 TEQ	Health effects (based on chronic endpoints)	MOE, 2008

NV No Value (MOE, 2008 does not provide a 24-hour value for benzene)
Shaded indicates selected value is not consistent with MOE guidance or value not provided by MOE (2008; 2009a)

It should be noted that the typical regulatory approach in Canada, including in Ontario, to evaluating ambient air concentrations of the criteria air contaminants is through a comparison to Canada Wide Standards (CWS) or National Ambient Air Quality Objectives. These standards and objectives typically provide the benchmark by which emissions from a proposed project are evaluated for acceptability, from both a federal and provincial compliance point-of-view. However, it should be noted that the NAAQOs for NOx and SO_2 are not specifically health risk-based. Many of these standards and objectives are dated (*i.e.*, established in 1974/5), do not include the most recent scientific health-based knowledge, and are impacted by policy decisions in their derivation. As such, any discussion on the effect of air pollution cannot rely on the attainment of such "standards" to guarantee that health within exposed population will be protected. More recent air quality guidelines (AQGs) have been published by the World Health Organization (WHO, 2005), and could be potential candidates for the evaluation of health risks related to these compounds. However, these AQGs have not been yet accepted as appropriate regulatory standards for the evaluation of air quality in North America, and there are concerns as to whether it is appropriate to apply these guidelines to a localized area around a specific discrete emission source (such as the proposed facility), rather than for the establishment of regional air quality objectives.

5.2 Chronic Toxicity Reference Values

5.2.1 Inhalation Exposures

The chronic non-carcinogenic and carcinogenic inhalation TRVs for each of the COCs (where they were available), as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 5-2. As indicated above, TRVs that are not consistent with those utilized by MOE in Reg. 153 (MOE, 2009a) and Reg. 419 (MOE, 2008) are shaded. In cases where values differ from those utilized by MOE, or in cases where TRV values are not provide

by MOE (2008, 2009a), toxicological profiles, including a detailed discussion of the relevant information supporting the selected TRV, are provided Appendix A.

Table 5-2 Summary of Chronic Non-carcinogenic and Carcinogenic Inhalation TRVs

COC	Chronic Toxicity Reference Values					
	Non-Carcinogenic Inhalation TRVs ($\mu g/m^3$)			Carcinogenic Inhalation Unit Risk Values $(\mu g/m^3)^{-1}$		
	Value	Critical Outcome	Source	Value	Critical Outcome	Source
Criteria Air Contaminants						
Hydrogen Chloride	9	Hyperplasia of the nasal mucosa, larynx and trachea	Cal EPA, 2000	NC	-	-
Oxides of Nitrogen (NO_x)	40	Health effects	WHO, 2005	NC	-	-
$PM_{2.5}$	10	Lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase	WHO, 2005	NC	-	-
PM_{10}	20	Lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase	WHO, 2005	NC	-	-
Sulphur Dioxide (SO_2)	30	Health and environmental effects	Health Canada, 2006	NC	-	-
Inorganics						
Arsenic	0.015	Decreased intellectual function, adverse effects on neurobehavioural development in 10 year old children	Cal EPA, 2008	4.3E-03	Lung cancer	US EPA IRIS, 1998
Cadmium	0.005	Proteinuria associated with proximal tubular dysfunction, lung cancer	MOE, 2009a	9.8E-03	Detection of lung tumours	MOE, 2009a
Chromium (total)	60	Lack of kidney effects (as measured urinary levels of protein and various enzymes)	MOE, 2009a	NC	-	-
Lead	0.5	Blood lead levels	WHO, 2000	NC	-	-
Mercury (inorganic)	0.09	Nervous system, kidney, development	MOE, 2009a	NC	-	-
Volatile Organic Compounds (VOCs)						
Benzene	30	Decreased lymphocyte count	MOE, 2009a	2.2E-06	Leukemia	MOE, 2009a
Vinyl Chloride	100	Liver cell polymorphism	MOE, 2009a	8.8E-06	Liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules	MOE, 2009a

Table 5-2 Summary of Chronic Non-carcinogenic and Carcinogenic Inhalation TRVs						
COC	***Chronic Toxicity Reference Values***					
	Non-Carcinogenic Inhalation TRVs (µg/m^3)			***Carcinogenic Inhalation Unit Risk Values (µg/m^3)$^{-1}$***		
	Value	***Critical Outcome***	***Source***	***Value***	***Critical Outcome***	***Source***
Polychlorinated dibenzo-p-dioxins / polychlorinated dibenzofurans (PCDD/PCDF)						
2,3,7,8-TCDD TEQ	8.0x10^{-6}	Reproductive toxicity in male offspring, calculated from maternal body burden with a half-life of 7.6 years	MOE, 2009a; Dose extrapolation from systemic TRV (2.3 pg/kg/day)[a]	NC	-	-
Carcinogenic PAHs						
Benzo(a)pyrene TEQ	NV	-	-	1.1E-03	Male hamster respiratory tract tumour incidence	MOE, 2009a

Shaded indicates selected value is not consistent with MOE (2008; 2009a) guidance or value not provided by MOE (2008; 2009a)

NC This chemical is not considered to be a carcinogen.

NV No value. No chronic TRVs are available for this COC.

[a] The primary TRV used for this COC is a systemic exposure limit including both inhalation and oral exposure. Therefore, the inhalation TRV in µg/m^3 was extrapolated from the µg/kg bw/day exposure limit by multiply by a standard bodyweight of 70 kg and dividing by a standard inhalation rate of 20 m^3/day.

5.2.2 Oral/Dermal Multi-Pathway Exposures

The chronic non-carcinogenic and carcinogenic oral/dermal TRVs for the PAH group (assuming benzo[a]pyrene-TEQ as a surrogate), as well as the key critical health outcomes and regulatory source for each TRV, are provided in Table 5-3. Refer to the toxicological profile for each of the COCs provided in an appendix to the main technical report for a detailed discussion of the relevant background information supporting the selected TRV.

Table 5-3 Summary of Chronic Non-carcinogenic and Carcinogenic Oral/Dermal TRVs						
Chemical of Concern	***Chronic Toxicity Reference Values***					
	Non-Carcinogenic Oral/Dermal TRVs (µg/kg bw/day)			***Carcinogenic Oral/Dermal Slope Factor Values (µg/kg bw/day)$^{-1}$***		
	Value	***Critical Outcome***	***Source***	***Value***	***Critical Outcome***	***Source***
Inorganics						
Arsenic	0.3	Hyperpigmentation, keratosis and possible vascular complications	MOE, 2009a	1.5E-03	Skin cancer prevalence rates	MOE, 2009a
Cadmium	0.03	Renal tubular dysfunction	MOE, 2009a	NC	-	-
Chromium (total)	1,500	No observed effects	MOE, 2009a	NC	-	-
Lead	1.85	Behavioural effects and learning disabilities in children	MOE, 1994	NC	-	-
Mercury (inorganic)	0.3	Kidney effects	MOE, 2009a	NC	-	-
Volatile Organic Compounds (VOCs)						
Benzene	4	Decreased lymphocyte Count	MOE, 2009a	8.5E-05	Leukemia	MOE, 2009a
Vinyl Chloride	3	Liver cell polymorphism	MOE, 2009a	1.4E-03	Liver angiosarcoma, hepatocellular carcinoma, and neoplastic nodules	MOE, 2009a

Table 5-3 Summary of Chronic Non-carcinogenic and Carcinogenic Oral/Dermal TRVs						
Chemical of Concern	***Chronic Toxicity Reference Values***					
	Non-Carcinogenic Oral/Dermal TRVs (µg/kg bw/day)			***Carcinogenic Oral/Dermal Slope Factor Values (µg/kg bw/day)$^{-1}$***		
	Value	***Critical Outcome***	***Source***	***Value***	***Critical Outcome***	***Source***
Polychlorinated Dibenzo-p-dioxins and Dibenzofurans						
2,3,7,8-TCDD TEQ	2.3E-06	Reproductive toxicity in male offspring, calculated from maternal body burden with a half-life of 7.6 years	MOE, 2009a	NC	-	-
Polycyclic Aromatic Hydrocarbons (PAHs)						
Benzo(a)pyrene TEQ	NV	-	-	7.3E-03	Carcinomas and papillomas	MOE, 2009a

Oral and dermal exposures to the PAH group are not evaluated by non-carcinogenic TRVs.
NV No value.

5.2.3 Toxicity Equivalence Factors for Dioxin and Furans

As all inhalation and deposition data was provided in the form of 2,3,7,8-TCDD toxicity equivalencies (*i.e.*, 2,3,7,8-TCDD TEQ) and not as the individual congeners. The WHO TEF factors (van den Berg *et al.*, 2006) were utilized to determine 2,3,7,8-TCDD equivalents.

5.3 Chemical Mixtures

For the current assessment, in addition to the dioxin/furan group of chemicals evaluated as a COC, a number of different mixture groups with specific health outcomes were evaluated. The health endpoint of the TRVs used in the HHRA provided the basis for an individual chemical's inclusion in a chemical mixture. For example, the acute inhalation TRV for hydrogen chloride is based on its ability to cause respiratory tract irritation, thus hydrogen chloride was included in the acute inhalation "respiratory irritant" mixture. The various evaluated mixture groups, as well as the COCs considered in each group, are listed in Table 5-4.

Table 5-4 Potential Additive Interactions of the Chemicals of Concern			
Exposure Characteristics	***Potential Health Endpoint of Mixture***	***Chemicals of Concern***	
Acute air exposure	respiratory irritants	hydrogen chloride, NO_x, $PM_{2.5}$, PM_{10},SO_2	
Chronic air exposure	neurological effects (neurotoxicants)	arsenic, lead, mercury (inorganic)	
	respiratory irritants	hydrogen chloride, NO_x, $PM_{2.5}$, PM_{10},SO_2	
	cancer	lung carcinogens	arsenic, cadmium, carcinogenic PAHs
		skin carcinogens	arsenic, carcinogenic PAHs

Table 5-4 Potential Additive Interactions of the Chemicals of Concern			
Exposure Characteristics	***Potential Health Endpoint of Mixture***	***Chemicals of Concern***	
Chronic oral exposure	kidney effects (renal toxicants)	cadmium, mercury (inorganic)	
	reproductive/developmental effects	arsenic, lead, dioxins/furans	
	cancer	skin carcinogens	arsenic and carcinogenic PAHs

Like the assessed COCs, the health implications of exposures to these mixture groups have been evaluated at each sensitive receptor location in the surrounding community, as well as for the on-site worker.

6.0 RISK CHARACTERIZATION

The following section provides the results of the acute and chronic assessment of risks related to emissions from the proposed incinerator gasifiers.

As noted previously, potential acute human health inhalation risks were evaluated for both 1-hour and 24-hour exposure periods for individuals living, working or playing in the surrounding community. For the assessment of potential chronic human health risks to individuals living, working, or playing in the surrounding community, ground-level air concentrations at the closest residential receptor location were evaluated based upon an annual average exposure period. In addition to inhalation risks, potential health risks related to long term exposures to soils and produce from home gardens that may have been impacted by deposited particulate from the proposed facilities are also evaluated. Additionally, worker, milk consumer and produce consumer scenarios were evaluated.

6.1 Acute Inhalation Assessment Results

The potential for acute adverse health effects were evaluated based upon potential inhalation exposures at each of the receptor locations, as well as for the worker. Tables 6-1 and 6-2 provide summaries of the predicted 1-hour and 24-hour acute inhalation risks at the receptor locations for each chemical of concern. It should be noted that results are provided only for those COCs with a TRV corresponding to the relevant duration of exposure.

Table 6-1 1-Hour Concentration Ratios

COC	Receptor Location													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	1.6E-02	2.8E-03	6.8E-03	4.8E-03	3.6E-03	4.4E-03	4.1E-03	4.4E-03	6.2E-03	7.6E-03	3.1E-03	1.2E-02	7.1E-03	3.6E-03
Nitrogen Oxides (NO_x)	4.2E-01	7.4E-02	1.8E-01	1.3E-01	9.8E-02	1.2E-01	1.1E-01	1.2E-01	1.7E-01	2.1E-01	8.4E-02	3.1E-01	1.9E-01	9.7E-02
Hydrogen Chloride	1.5E-01	2.6E-02	6.3E-02	4.4E-02	3.4E-02	4.0E-02	3.8E-02	4.1E-02	5.7E-02	7.0E-02	2.9E-02	1.1E-01	6.5E-02	3.3E-02
Particulate Matter (PM_{10})	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Particulate Matter ($PM_{2.5}$)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Inorganics														
Arsenic	6.8E-04	1.2E-04	2.9E-04	2.1E-04	1.6E-04	1.9E-04	1.8E-04	1.9E-04	2.7E-04	3.3E-04	1.3E-04	5.0E-04	3.1E-04	1.6E-04
Cadmium	2.3E-03	4.1E-04	1.0E-03	7.0E-04	5.4E-04	6.4E-04	6.0E-04	6.6E-04	9.1E-04	1.1E-03	4.6E-04	1.7E-03	1.0E-03	5.3E-04
Chromium (Total)	4.9E-03	8.7E-04	2.1E-03	1.5E-03	1.1E-03	1.4E-03	1.3E-03	1.4E-03	1.9E-03	2.4E-03	9.8E-04	3.6E-03	2.2E-03	1.1E-03
Lead	3.4E-04	5.9E-05	1.5E-04	1.0E-04	7.8E-05	9.3E-05	8.7E-05	9.5E-05	1.3E-04	1.6E-04	6.7E-05	2.5E-04	1.5E-04	7.7E-05
Mercury (Inorganic)	1.0E-03	1.8E-04	4.5E-04	3.2E-04	2.4E-04	2.9E-04	2.7E-04	3.0E-04	4.1E-04	5.1E-04	2.1E-04	7.7E-04	4.7E-04	2.4E-04
Organics														
Vinyl Chloride	5.1E-08	8.9E-09	2.2E-08	1.5E-08	1.2E-08	1.4E-08	1.3E-08	1.4E-08	2.0E-08	2.5E-08	1.0E-08	3.7E-08	2.3E-08	1.2E-08
Benzene	1.6E-05	2.8E-06	6.9E-06	4.9E-06	3.7E-06	4.4E-06	4.1E-06	4.5E-06	6.3E-06	7.8E-06	3.2E-06	1.2E-05	7.2E-06	3.7E-06
PAHs														
Benzo(a)pyrene	8.9E-02	1.6E-02	3.9E-02	2.7E-02	2.1E-02	2.5E-02	2.3E-02	2.5E-02	3.5E-02	4.3E-02	1.8E-02	6.6E-02	4.0E-02	2.0E-02
Dioxins / Furans														
Dioxins & Furans	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mixtures														
Respiratory irritants	5.8E-01	1.0E-01	2.5E-01	1.8E-01	1.4E-01	1.6E-01	1.5E-01	1.7E-01	2.3E-01	2.8E-01	1.2E-01	4.3E-01	2.6E-01	1.3E-01

\- No acute (1-hour) TRV are available.

Table 6-2 24-Hour Concentration Ratios

COC	Receptor Location													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	1.9E-01	1.1E-02	4.1E-02	6.5E-02	2.3E-02	5.9E-02	2.5E-02	1.9E-02	3.4E-02	1.1E-01	1.8E-02	7.9E-02	1.9E-02	2.8E-02
Nitrogen Oxides (NO_x)	2.2E-01	1.3E-02	4.8E-02	7.8E-02	2.7E-02	7.1E-02	2.9E-02	2.3E-02	4.1E-02	1.3E-01	2.2E-02	9.4E-02	2.3E-02	3.4E-02
Hydrogen Chloride	2.9E-01	1.6E-02	6.2E-02	1.0E-01	3.5E-02	9.1E-02	3.8E-02	3.0E-02	5.3E-02	1.6E-01	2.8E-02	1.2E-01	2.9E-02	4.4E-02
Particulate Matter (PM_{10})	2.7E-02	1.5E-03	5.9E-03	9.4E-03	3.3E-03	8.5E-03	3.5E-03	2.8E-03	5.0E-03	1.5E-02	2.6E-03	1.1E-02	2.7E-03	4.1E-03
Particulate Matter ($PM_{2.5}$)	2.7E-02	1.5E-03	5.9E-03	9.4E-03	3.3E-03	8.5E-03	3.5E-03	2.8E-03	5.0E-03	1.5E-02	2.6E-03	1.1E-02	2.7E-03	4.1E-03
Inorganics														
Arsenic	2.4E-04	1.4E-05	5.2E-05	8.4E-05	2.9E-05	7.6E-05	3.2E-05	2.5E-05	4.4E-05	1.4E-04	2.3E-05	1.0E-04	2.4E-05	3.6E-05
Cadmium	4.9E-03	2.8E-04	1.1E-03	1.7E-03	6.0E-04	1.6E-03	6.5E-04	5.1E-04	9.0E-04	2.8E-03	4.7E-04	2.1E-03	5.0E-04	7.5E-04
Chromium (Total)	1.7E-03	9.9E-05	3.8E-04	6.1E-04	2.1E-04	5.5E-04	2.3E-04	1.8E-04	3.2E-04	9.8E-04	1.7E-04	7.3E-04	1.8E-04	2.7E-04
Lead	5.3E-04	3.0E-05	1.2E-04	1.9E-04	6.5E-05	1.7E-04	7.0E-05	5.5E-05	9.8E-05	3.0E-04	5.1E-05	2.2E-04	5.4E-05	8.1E-05
Mercury (Inorganic)	1.7E-04	9.4E-06	3.6E-05	5.8E-05	2.0E-05	5.3E-05	2.2E-05	1.7E-05	3.1E-05	9.4E-05	1.6E-05	7.0E-05	1.7E-05	2.5E-05

Table 6-2 24-Hour Concentration Ratios														
COC	*Receptor Location*													
	MAX	**C1**	**C2**	**C3**	**C4**	**P1**	**P2**	**P3**	**R1**	**R2**	**R3**	**R4**	**R5**	**R6**
Organics														
Vinyl Chloride	5.3E-04	3.0E-05	1.2E-04	1.9E-04	6.5E-05	1.7E-04	7.0E-05	5.5E-05	9.8E-05	3.0E-04	5.2E-05	2.2E-04	5.4E-05	8.1E-05
Benzene	5.0E-05	2.8E-06	1.1E-05	1.7E-05	6.1E-06	1.6E-05	6.5E-06	5.1E-06	9.1E-06	2.8E-05	4.8E-06	2.1E-05	5.0E-06	7.5E-06
PAHs														
Benzo(a)pyrene	4.3E-02	2.4E-03	9.3E-03	1.5E-02	5.3E-03	1.4E-02	5.6E-03	4.4E-03	7.9E-03	2.4E-02	4.1E-03	1.8E-02	4.4E-03	6.5E-03
Dioxins / Furans														
Dioxins & Furans	8.7E-04	4.9E-05	1.9E-04	3.0E-04	1.1E-04	2.8E-04	1.1E-04	9.0E-05	1.6E-04	4.9E-04	8.4E-05	3.7E-04	8.8E-05	1.3E-04
Mixtures														
Respiratory irritants	7.5E-01	4.3E-02	1.6E-01	2.6E-01	9.2E-02	2.4E-01	9.9E-02	7.7E-02	1.4E-01	4.2E-01	7.3E-02	3.2E-01	7.6E-02	1.1E-01

The results of the acute inhalation assessment indicated that there are no acute impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreational/community scenarios. In fact, most predicted concentration ratios demonstrated that predicted ambient concentrations of the COCs were many orders of magnitude below the corresponding regulatory benchmarks.

6.2 Chronic Inhalation Assessment Results

Tables 6-3 and 6-4 provide summaries of the predicted chronic inhalation risks at the receptor location for each chemical of concern. Results of the assessment of both non-cancer and cancer risks are presented, where applicable.

Table 6-3 Annual Average Concentration Ratios

COC	Receptor Location													
	MAX	C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	6.7E-03	2.9E-04	2.2E-03	1.3E-03	4.3E-04	9.9E-04	1.1E-03	7.7E-04	1.8E-03	1.9E-03	6.0E-04	3.0E-03	6.3E-04	1.0E-03
Nitrogen Oxides (NO_x)	6.0E-02	2.6E-03	2.0E-02	1.1E-02	3.9E-03	8.8E-03	9.7E-03	6.9E-03	1.6E-02	1.7E-02	5.4E-03	2.7E-02	5.7E-03	9.0E-03
Hydrogen Chloride	3.4E-02	1.5E-03	1.1E-02	6.4E-03	2.2E-03	5.1E-03	5.5E-03	4.0E-03	9.2E-03	9.7E-03	3.1E-03	1.5E-02	3.2E-03	5.1E-03
Particulate Matter (PM_{10})	3.6E-03	1.6E-04	1.2E-03	6.8E-04	2.3E-04	5.3E-04	5.8E-04	4.2E-04	9.7E-04	1.0E-03	3.3E-04	1.6E-03	3.4E-04	5.4E-04
Particulate Matter ($PM_{2.5}$)	3.6E-03	1.6E-04	1.2E-03	6.8E-04	2.3E-04	5.3E-04	5.8E-04	4.2E-04	9.7E-04	1.0E-03	3.3E-04	1.6E-03	3.4E-04	5.4E-04
Arsenic	2.6E-04	1.1E-05	8.6E-05	4.8E-05	1.7E-05	3.8E-05	4.2E-05	3.0E-05	6.9E-05	7.3E-05	2.3E-05	1.1E-04	2.4E-05	3.9E-05
Cadmium	1.3E-03	5.8E-05	4.4E-04	2.5E-04	8.5E-05	1.9E-04	2.1E-04	1.5E-04	3.5E-04	3.7E-04	1.2E-04	5.9E-04	1.2E-04	2.0E-04
Chromium (Total)	2.3E-06	1.0E-07	7.8E-07	4.4E-07	1.5E-07	3.5E-07	3.8E-07	2.7E-07	6.3E-07	6.6E-07	2.1E-07	1.0E-06	2.2E-07	3.5E-07
Lead	2.8E-05	1.3E-06	9.6E-06	5.3E-06	1.8E-06	4.2E-06	4.6E-06	3.3E-06	7.7E-06	8.1E-06	2.6E-06	1.3E-05	2.7E-06	4.3E-06
Mercury (Inorganic)	2.0E-04	8.7E-06	6.6E-05	3.7E-05	1.3E-05	2.9E-05	3.2E-05	2.3E-05	5.3E-05	5.6E-05	1.8E-05	8.8E-05	1.9E-05	3.0E-05
Organics														
Vinyl Chloride	2.9E-07	1.3E-08	9.6E-08	5.4E-08	1.9E-08	4.2E-08	4.6E-08	3.3E-08	7.7E-08	8.1E-08	2.6E-08	1.3E-07	2.7E-08	4.3E-08
Benzene	2.6E-06	1.1E-07	8.6E-07	4.8E-07	1.7E-07	3.8E-07	4.2E-07	3.0E-07	6.9E-07	7.3E-07	2.3E-07	1.1E-06	2.4E-07	3.9E-07
PAHs														
Benzo(a)pyrene	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Dioxins / Furans														
Dioxins & Furans	2.9E-05	1.3E-06	9.8E-06	5.5E-06	1.9E-06	4.3E-06	4.7E-06	3.4E-06	7.8E-06	8.3E-06	2.6E-06	1.3E-05	2.8E-06	4.4E-06
Mixtures														
Respiratory irritants	1.1E-01	4.7E-03	3.6E-02	2.0E-02	7.0E-03	1.6E-02	1.7E-02	1.2E-02	2.9E-02	3.1E-02	9.7E-03	4.8E-02	1.0E-02	1.6E-02
Neurotoxicants	4.8E-04	2.1E-05	1.6E-04	9.0E-05	3.1E-05	7.1E-05	7.8E-05	5.6E-05	1.3E-04	1.4E-04	4.4E-05	2.2E-04	4.6E-05	7.3E-05

- Not applicable, chemical evaluated as a carcinogen only.

Table 6-4 Summary of Incremental Lifetime Cancer Risks (ILCR) at Each Receptor Location

COC	*Receptor Location*													
	MAX	**C1**	**C2**	**C3**	**C4**	**P1**	**P2**	**P3**	**R1**	**R2**	**R3**	**R4**	**R5**	**R6**
Criteria Air Contaminants														
Sulphur Dioxide (SO_2)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Nitrogen Oxides (NO_x)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Hydrogen Chloride	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Particulate Matter (PM_{10})	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Particulate Matter ($PM_{2.5}$)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Inorganics														
Arsenic	1.6E-08	7.3E-10	5.5E-09	3.1E-09	1.1E-09	2.4E-09	2.7E-09	1.9E-09	4.4E-09	4.7E-09	1.5E-09	7.4E-09	1.6E-09	2.5E-09
Cadmium	6.4E-08	2.8E-09	2.2E-08	1.2E-08	4.2E-09	9.5E-09	1.0E-08	7.4E-09	1.7E-08	1.8E-08	5.8E-09	2.9E-08	6.1E-09	9.7E-09
Chromium (Total)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lead	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mercury (Inorganic)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Organics														
Vinyl Chloride	2.5E-10	1.1E-11	8.4E-11	4.7E-11	1.6E-11	3.7E-11	4.1E-11	2.9E-11	6.8E-11	7.2E-11	2.3E-11	1.1E-10	2.4E-11	3.8E-11
Benzene	1.7E-10	7.4E-12	5.7E-11	3.2E-11	1.1E-11	2.5E-11	2.7E-11	2.0E-11	4.5E-11	4.8E-11	1.5E-11	7.6E-11	1.6E-11	2.5E-11
PAHs														
Benzo(a)pyrene	2.8E-09	1.2E-10	9.3E-10	5.2E-10	1.8E-10	4.1E-10	4.5E-10	3.2E-10	7.5E-10	7.9E-10	2.5E-10	1.2E-09	2.6E-10	4.2E-10
Dioxins / Furans														
Dioxins & Furans	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mixtures														
Lung carcinogens	8.3E-08	3.7E-09	2.8E-08	1.6E-08	5.4E-09	1.2E-08	1.4E-08	9.7E-09	2.2E-08	2.4E-08	7.6E-09	3.7E-08	7.9E-09	1.3E-08
Skin carcinogens	1.9E-08	8.5E-10	6.5E-09	3.6E-09	1.3E-09	2.9E-09	3.1E-09	2.2E-09	5.2E-09	5.5E-09	1.7E-09	8.6E-09	1.8E-09	2.9E-09

- Not applicable, chemical not evaluated as a carcinogen.

The results of the chronic inhalation assessment indicated that there are no chronic impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community under the residential and recreational/community scenarios. Most predicted concentrations ratios and incremental lifetime cancer risk levels demonstrated that predicted ambient concentrations of the COCs were many orders of magnitude below the corresponding regulatory benchmarks.

6.3 Chronic Multi-Pathway Results

For a subset of the COCs, there is the potential for exposure arising from facility deposition onto soils and home gardens (where applicable) in the surrounding community. Therefore, in addition to the assessment of inhalation risks, a multi-media assessment of the oral and dermal exposure pathways was also conducted for each of the sensitive receptor locations (both residential/recreational and on-site). Similar assessments were conducted for the produce and milk consumer scenarios.

6.3.1 Residential and Recreational/Community Scenarios

Table 6-5 through 6-7 provide summaries of the predicted chronic multimedia (*i.e.*, inhalation, oral and dermal exposures) risks at receptor location for each chemical of concern.

Risk estimates, for both non-cancer and cancer risks are presented, where applicable. For the non-cancer risk estimates, results are provided for the most sensitive receptor groups, the toddler (Table 6-5) and infant receptors (Table 6-6), the difference being breast milk consumption by the infant. Cancer risks are provided for a lifetime (composite) receptor.

Table 6-5 Summary of Toddler Hazard Quotient (HQ)

COC	Receptor Location									
	C1	C2	C3	C4	R1	R2	R3	R4	R5	R6
Inorganics										
Arsenic	7.1E-07	9.0E-06	3.1E-06	4.2E-07	1.1E-05	8.2E-06	2.0E-06	1.6E-05	2.1E-06	3.2E-06
Cadmium	1.2E-05	1.5E-04	5.3E-05	7.1E-06	3.4E-04	2.3E-04	5.5E-05	4.8E-04	5.9E-05	8.9E-05
Chromium	5.2E-09	6.7E-08	2.3E-08	3.0E-09	8.2E-08	5.9E-08	1.4E-08	1.2E-07	1.5E-08	2.3E-08
Lead	4.3E-07	5.4E-06	1.9E-06	2.5E-07	7.1E-06	5.1E-06	1.2E-06	1.0E-05	1.3E-06	2.0E-06
Mercury	3.3E-06	4.2E-05	1.5E-05	1.9E-06	3.1E-04	2.1E-04	4.9E-05	4.4E-04	5.3E-05	7.9E-05
VOCs										
Benzene	4.3E-07	3.2E-06	1.8E-06	6.3E-07	6.9E-06	5.6E-06	1.5E-06	1.0E-05	1.6E-06	2.5E-06
Vinyl Chloride	2.1E-07	1.6E-06	9.0E-07	3.1E-07	3.4E-06	2.8E-06	7.5E-07	5.1E-06	7.9E-07	1.2E-06
Dioxins & Furans										
Dioxins & Furans (TEQ)	2.6E-06	2.2E-05	1.1E-05	3.3E-06	1.6E-03	1.6E-03	1.6E-03	1.6E-03	1.6E-03	1.6E-03
PAHs										
Benzo(a)pyrene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Mixtures										
Kidney effects (renal toxicants)	1.5E-05	2.0E-04	6.8E-05	9.1E-06	6.5E-04	4.5E-04	1.0E-04	9.1E-04	1.1E-04	1.7E-04
Reproductive/developmental effects	3.7E-06	3.7E-05	1.6E-05	4.0E-06	1.6E-03	1.6E-03	1.6E-03	1.7E-03	1.6E-03	1.6E-03

- Not applicable, chemical evaluated as a carcinogen only

Table 6-6 Summary of Infant Hazard Quotient (HQ)

COC	Receptor Location									
	C1	C2	C3	C4	R1	R2	R3	R4	R5	R6
Inorganics										
Arsenic	2.8E-07	3.3E-06	1.2E-06	2.2E-07	2.8E-06	2.2E-06	5.9E-07	4.2E-06	6.2E-07	9.6E-07
Cadmium	4.8E-06	5.5E-05	2.1E-05	3.8E-06	4.7E-05	3.7E-05	9.9E-06	7.0E-05	1.1E-05	1.6E-05
Chromium	2.2E-09	2.6E-08	9.5E-09	1.6E-09	2.2E-08	1.7E-08	4.5E-09	3.2E-08	4.8E-09	7.3E-09
Lead	1.7E-07	1.9E-06	7.3E-07	1.3E-07	1.7E-06	1.3E-06	3.5E-07	2.4E-06	3.7E-07	5.7E-07
Mercury	1.4E-06	1.6E-05	6.0E-06	1.0E-06	1.4E-05	1.1E-05	2.9E-06	2.0E-05	3.0E-06	4.6E-06
VOCs										
Benzene	2.3E-07	1.7E-06	9.7E-07	3.3E-07	1.4E-06	1.5E-06	4.7E-07	2.3E-06	4.9E-07	7.8E-07
Vinyl Chloride	1.1E-07	8.6E-07	4.8E-07	1.7E-07	6.9E-07	7.3E-07	2.3E-07	1.1E-06	2.4E-07	3.8E-07
Dioxins & Furans										
Dioxins & Furans (TEQ)	1.3E-06	1.1E-05	5.6E-06	1.8E-06	6.2E-04	5.1E-04	1.4E-04	9.3E-04	1.5E-04	2.3E-04
PAHs										
Benzo(a)pyrene	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Mixtures										
Kidney effects (renal toxicants)	6.2E-06	7.1E-05	2.7E-05	4.8E-06	6.1E-05	4.8E-05	1.3E-05	9.0E-05	1.4E-05	2.1E-05
Reproductive/developmental effects	1.8E-06	1.6E-05	7.5E-06	2.1E-06	6.2E-04	5.1E-04	1.4E-04	9.3E-04	1.5E-04	2.3E-04

- Not applicable, evaluated as a carcinogen only.

Table 6-7 Summary of Incremental Lifetime Cancer Risks (ILCR)

COC	*Receptor Location*									
	C1	**C2**	**C3**	**C4**	**R1**	**R2**	**R3**	**R4**	**R5**	**R6**
Inorganics										
Arsenic	4.8E-11	5.0E-10	2.1E-10	4.6E-11	7.9E-10	6.0E-10	1.5E-10	1.1E-09	1.6E-10	2.5E-10
Cadmium	-	-	-	-	-	-	-	-	-	-
Chromium	-	-	-	-	-	-	-	-	-	-
Lead	-	-	-	-	-	-	-	-	-	-
Mercury	-	-	-	-	-	-	-	-	-	-
VOCs										
Benzene	3.6E-11	2.7E-10	1.5E-10	5.3E-11	5.5E-10	4.5E-10	1.2E-10	8.3E-10	1.3E-10	2.0E-10
Vinyl Chloride	2.2E-10	1.7E-09	9.3E-10	3.2E-10	3.4E-09	2.8E-09	7.5E-10	5.1E-09	7.9E-10	1.2E-09
Dioxins & Furans										
Dioxins & Furans (TEQ)	-	-	-	-	-	-	-	-	-	-
PAHs										
Benzo(a)pyrene	1.0E-10	8.3E-10	4.4E-10	1.5E-10	1.9E-09	1.6E-09	4.6E-10	2.9E-09	4.8E-10	7.5E-10
Mixtures										
Skin carcinogens	1.5E-10	1.3E-09	6.5E-10	1.9E-10	2.7E-09	2.2E-09	6.1E-10	4.1E-09	6.4E-10	9.9E-10

- Not applicable, chemical not evaluated as a carcinogen.

The results of the chronic multimedia (*i.e.*, inhalation, oral and dermal exposures assessment) indicated that there are no chronic impacts to human health expected as a result of deposition of facility emissions onto soils and home gardens of residences in the surrounding community. In fact, most predicted hazard quotients and incremental lifetime cancer risk levels demonstrated that predicted concentrations of the COCs in soil and home garden produce (where applicable) at the various sensitive receptor locations were many orders of magnitude below the corresponding regulatory benchmarks.

6.3.2 Produce Consumer Scenario

Table 6-8 provides a summary of the predicted risk related to the consumption of produce by a receptor living away from the Kingsville area for each chemical of concern. Risks associated with both consumption of produce grown in on-site greenhouses (MAX POI; deposition through air intakes of greenhouses) and grown at other agricultural operations in the community (*i.e.*, receptor locations P1 (Asparagus Crop Land), P2 (Apple Orchard), P3 (Vineyards)) were predicted.

Risk estimates for both non-cancer and cancer risks are presented, where applicable. For the non-cancer risk estimates, results are provided for the most sensitive receptor group, the toddler.

Table 6-8 Summary of Risks for the Produce Consumer Scenario

COC	*HQ (Toddler)*				*ILCR (Composite)*			
	MAX	**P1**	**P2**	**P3**	**MAX**	**P1**	**P2**	**P3**
Inorganics								
Arsenic	8.03E-06	1.27E-06	6.93E-07	5.35E-07	8.29E-10	1.31E-10	7.16E-11	5.53E-11
Cadmium	2.98E-04	4.72E-05	2.57E-05	1.99E-05	NA	NA	NA	NA
Chromium	5.76E-08	9.12E-09	4.98E-09	3.84E-09	NA	NA	NA	NA
Lead	5.17E-06	8.19E-07	4.47E-07	3.45E-07	NA	NA	NA	NA
Mercury	4.08E-04	6.41E-05	3.85E-05	2.93E-05	NA	NA	NA	NA
VOCs								
Benzene	1.13E-05	1.79E-06	9.75E-07	7.52E-07	8.81E-10	1.39E-10	7.61E-11	5.87E-11
Vinyl Chloride	5.60E-06	8.86E-07	4.83E-07	3.73E-07	5.40E-09	8.54E-10	4.66E-10	3.60E-10
Dioxins & Furans								
Dioxins & Furans (TEQ)	6.92E-05	1.09E-05	6.72E-06	5.09E-06	NA	NA	NA	NA
PAHs								
Benzo(a)pyrene	NA	NA	NA	NA	3.52E-09	5.47E-10	3.82E-10	2.85E-10
Mixtures								
Kidney effects (renal toxicants)	7.06E-04	1.11E-04	6.42E-05	4.92E-05	NA	NA	NA	NA
Reproductive/developmental effects	8.24E-05	1.29E-05	7.86E-06	5.97E-06	NA	NA	NA	NA
Skin carcinogens	NA	NA	NA	NA	4.35E-09	6.78E-10	4.54E-10	3.41E-10

The results of this scenario indicated that there are no chronic impacts to human health expected as a result of produce consumption. In fact, most predicted hazard quotients and incremental lifetime cancer risk levels demonstrated that predicted concentrations of the COCs in vegetables were many orders of magnitude below the corresponding regulatory benchmarks.

6.3.3 Milk Consumer Scenario

Risks were also estimated for a toddler drinking milk from the dairy farm while living outside of the Kingsville area. The scenario was only evaluated for the dioxins/furans due to their bioaccumulative nature. The predicted hazard quotient for this scenario was 0.00372, indicating no chronic impacts to human health are expected. Details are provided in Appendix B. Toddlers living at residential locations in the Kingsville area were also assumed to drink milk from the dairy farm.

6.4 Cumulative Assessment Results

Predicted 1-hour and 24-hour acute inhalation risks and chronic (annual average) inhalation risks at each sensitive receptor location under various air quality conditions are presented in Table 6-9. The current conditions ("Cumulative - Current") scenario evaluates the potential health impact related to the local significant sources of COCs. The facilities alone ("REMASCO Facilities Alone") scenario evaluates the potential health impact related to the operation of the proposed facilities alone. The future conditions ("Cumulative - Future with REMASCO") scenario evaluates the potential health impact related to the predicted ground-level air concentrations of each of the COCs contributed by the proposed facilities plus other local significant sources that contribute to background.

Table 6-9 Comparison of Predicted Cumulative Risks under Current Conditions to Future Conditions at Each Receptor Location

COC	Condition	Receptor Location												
		C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
1-hour														
Nitrogen Dioxide (NO_2)	Cumulative - Current	9.0E-01	9.0E-01	**1.1E+00**	**1.0E+00**	**1.0E+00**	9.7E-01	8.9E-01	9.9E-01	**1.1E+00**	**1.0E+00**	9.0E-01	9.2E-01	9.4E-01
	REMASCO Facilities Alone	6.4E-02	2.2E-01	1.2E-01	7.7E-02	1.2E-01	1.1E-01	1.2E-01	1.7E-01	1.8E-01	4.6E-02	3.8E-01	1.4E-01	1.1E-01
	Cumulative - Future with REMASCO	8.2E-01	8.8E-01	8.3E-01	8.2E-01	9.0E-01	8.8E-01	8.4E-01	8.2E-01	9.9E-01	8.4E-01	8.9E-01	9.2E-01	9.4E-01
Particulate Matter ($PM_{2.5}$)	Cumulative - Current	-	-	-	-	-	-	-	-	-	-	-	-	-
	REMASCO Facilities Alone	-	-	-	-	-	-	-	-	-	-	-	-	-
	Cumulative - Future with REMASCO	-	-	-	-	-	-	-	-	-	-	-	-	-
Mixtures														
Respira-tory Irritants	Cumulative - Current	**2.5E+00**	**3.3E+00**	**4.8E+00**	**3.4E+00**	**4.5E+00**	**5.4E+00**	**3.5E+00**	**6.1E+00**	**6.3E+00**	**4.5E+00**	**4.2E+00**	**3.4E+00**	**5.0E+00**
	REMASCO Facilities Alone	1.5E-02	8.2E-02	6.9E-02	3.0E-02	8.0E-02	3.4E-02	2.5E-02	4.4E-02	1.2E-01	1.9E-02	1.5E-01	2.6E-02	3.6E-02
	Cumulative - Future with REMASCO	**2.2E+00**	**3.1E+00**	**3.5E+00**	**3.2E+00**	**4.5E+00**	**4.2E+00**	**3.0E+00**	**3.5E+00**	**4.1E+00**	**3.5E+00**	**3.7E+00**	**3.0E+00**	**5.0E+00**
24-hour														
Nitrogen Dioxide (NO_2)	Cumulative - Current	5.7E-01	6.8E-01	7.3E-01	6.2E-01	7.6E-01	8.0E-01	6.1E-01	9.2E-01	9.0E-01	7.8E-01	7.9E-01	6.6E-01	8.8E-01
	REMASCO Facilities Alone	1.3E-02	7.1E-02	6.0E-02	2.6E-02	7.0E-02	2.9E-02	2.1E-02	4.1E-02	1.0E-01	1.7E-02	1.2E-01	2.3E-02	3.1E-02
	Cumulative - Future with REMASCO	5.0E-01	7.2E-01	7.8E-01	5.7E-01	7.6E-01	7.2E-01	5.7E-01	6.4E-01	7.1E-01	7.1E-01	6.4E-01	6.5E-01	8.8E-01
Particulate Matter ($PM_{2.5}$)	Cumulative - Current	**1.9E+00**	**2.6E+00**	**4.1E+00**	**2.8E+00**	**3.7E+00**	**4.6E+00**	**2.9E+00**	**5.2E+00**	**5.4E+00**	**3.7E+00**	**3.4E+00**	**2.7E+00**	**4.1E+00**
	REMASCO Facilities Alone	2.1E-03	1.1E-02	9.0E-03	4.2E-03	9.6E-03	4.8E-03	3.4E-03	3.2E-03	1.6E-02	3.0E-03	3.2E-02	3.2E-03	4.9E-03
	Cumulative - Future with REMASCO	**1.7E+00**	**2.4E+00**	**2.7E+00**	**2.7E+00**	**3.7E+00**	**3.5E+00**	**2.5E+00**	**2.9E+00**	**3.4E+00**	**2.8E+00**	**3.1E+00**	**2.4E+00**	**4.1E+00**

Table 6-9 Comparison of Predicted Cumulative Risks under Current Conditions to Future Conditions at Each Receptor Location

COC	Condition	Receptor Location												
		C1	C2	C3	C4	P1	P2	P3	R1	R2	R3	R4	R5	R6
Annual Average														
Nitrogen Dioxide (NO_2)	Cumulative - Current	6.3E-01	9.3E-01	7.8E-01	6.3E-01	9.2E-01	9.7E-01	7.9E-01	**1.0E+00**	**1.2E+00**	7.5E-01	**1.1E+00**	7.9E-01	9.6E-01
	REMASCO Facilities Alone	2.8E-03	2.8E-02	1.1E-02	3.5E-03	8.8E-03	9.3E-03	7.0E-03	1.6E-02	1.8E-02	4.8E-03	3.1E-02	5.8E-03	9.0E-03
	Cumulative - Future with REMASCO	6.1E-01	9.4E-01	7.8E-01	6.1E-01	8.3E-01	9.2E-01	7.4E-01	7.2E-01	8.3E-01	7.2E-01	8.3E-01	7.4E-01	8.8E-01
Particulate Matter ($PM_{2.5}$)	Cumulative - Current	**1.0E+00**	**1.7E+00**	**1.4E+00**	**1.0E+00**	**1.8E+00**	**1.9E+00**	**1.5E+00**	**2.0E+00**	**2.6E+00**	**1.3E+00**	**2.1E+00**	**1.5E+00**	**2.1E+00**
	REMASCO Facilities Alone	2.1E-04	1.4E-03	7.0E-04	3.3E-04	5.6E-04	8.1E-04	5.8E-04	2.7E-04	1.4E-03	1.1E-04	1.8E-03	4.6E-04	8.0E-04
	Cumulative - Future with REMASCO	9.8E-01	**1.4E+00**	**1.2E+00**	9.7E-01	**1.6E+00**	**1.3E+00**	**1.3E+00**	**1.4E+00**	**1.6E+00**	**1.2E+00**	**1.7E+00**	**1.3E+00**	**1.6E+00**
Mixtures														
Respira-tory Irritants	Cumulative - Current	**1.7E+00**	**2.7E+00**	**2.2E+00**	**1.7E+00**	**2.7E+00**	**2.9E+00**	**2.3E+00**	**3.1E+00**	**3.8E+00**	**2.1E+00**	**3.1E+00**	**2.2E+00**	**3.1E+00**
	REMASCO Facilities Alone	3.0E-03	2.9E-02	1.1E-02	3.8E-03	9.3E-03	1.0E-02	7.6E-03	1.6E-02	1.9E-02	4.9E-03	3.2E-02	6.2E-03	9.8E-03
	Cumulative - Future with REMASCO	**1.6E+00**	**2.4E+00**	**2.0E+00**	**1.6E+00**	**2.4E+00**	**2.2E+00**	**2.1E+00**	**2.1E+00**	**2.4E+00**	**1.9E+00**	**2.5E+00**	**2.1E+00**	**2.5E+00**

BOLDED and shaded indicates an exceedance of the acceptable concentration ratio of 1.0.

\- No acute (1-hour) TRV are available.

As discussed in Section 6.1 and 6.2, the results of the inhalation assessment indicated that there are no acute or chronic impacts to human health expected as a result of emissions from the proposed facilities ("REMASCO Facilities alone") to the ambient air. In most cases, there are also no acute or chronic impacts to human health expected as a result of exposure under current and future cumulative conditions.

Evaluation of potential exposures under current and future cumulative conditions indicate marginal exceedances of the chronic TRV for NOx and $PM_{2.5}$ at several receptor locations. Mixture effects are also noted at several locations. In all cases, future cumulative risks with the proposed REMASCO facilities are equal to or lower than risks predicted under existing background conditions. There were no cases in which the added emissions from the proposed REMASCO facilities alone resulted in cumulative risk above that already existing under current (background) conditions. As a result, there will be a net benefit to the installation and the operation of the REMASCO facilities.

6.5 Upset Conditions

Given the design of the proposed facilities, assessment of potential exposure and associated risk under a facility "upset scenario" was not considered relevant as part of the current assessment. Unlike some combustion/incineration systems, there are no bypass stacks on the REMASCO gasifier units. Therefore, the only way for exhaust gases to exit the system is through air pollution control systems (*i.e.*, the baghouse), thereby limiting potential for increased release and associated exposures under upset conditions.

Under facility start-up conditions, natural gas is used to heat the refractory to just below the standard operating temperatures without flue gas circulation. The fuel is then added to the hot system, thus reducing the effects of startup/shutdown in unstable conditions. The use of natural gas also allows for the conditioning of the bags prior to the introduction of the Enerpax pellets. Conditioning of the bags ensures that the baghouse is offering appropriate air quality protection. In this way, the use of natural gas during startup and recovery from unscheduled shutdowns offers a distinct advantage over the use of wood during startup. With the use wood, the baghouses cannot be used for fear of blinding the bags with tars and oils prior to the lime cake being well established on the bags.

System operation involves the recirculation of flue gas to the furnace for NOx control. When there are system upsets, the recirculation flow will decrease during the shutdown resulting in a substantial increase in the effective baghouse capacity. While both of these scenarios (*i.e.*, start-up and emergency shut-down) may give rise to short-term increases in NOx emissions, these conditions are not sustained for more than 90 minutes and may be expected to occur on an infrequent basis. In addition, all of the proposed REMASCO facilities have emergency generators that supply sufficient power to allow gasifiers to be shut down in a controlled manner.

Process upsets are further discussed and evaluated in the Air Quality Report (Chandler, 2011). There is little data available to quantitatively assess the emissions that could occur under any of these situations, however, potential upset conditions were evaluated following the approach suggested by the California Air Resources Board. These methods are further discussed in Chandler (2011). Table 6-10 contains predicted 1-hour, 24-hour and annual average air concentrations at the maximum residential receptor location resulting from the upset scenarios. Corresponding risk estimates are provided in Table 6-11.

Table 6-10 Upset Ground Level Air Concentration (ug/m^3)

COC	Maximum Residential Location			
	1-hour	24-hour	Chronic	ILCR
Criteria Air Contaminants				
Sulphur Dioxide (SO_2)	2.2E+01	1.9E+00	8.2E-02	8.2E-02
Nitrogen Oxides (NO_x)	7.9E+01	6.8E+00	3.0E-01	3.0E-01
Hydrogen Chloride	4.7E+01	4.1E+00	1.8E-01	1.8E-01
Particulate Matter (PM_{10})	1.1E+01	9.5E-01	4.2E-02	4.2E-02
Particulate Matter ($PM_{2.5}$)	5.5E+00	4.8E-01	2.1E-02	2.1E-02
Inorganics				
Arsenic	5.9E-04	5.1E-05	2.3E-06	2.3E-06
Cadmium	1.0E-03	8.7E-05	3.8E-06	3.8E-06
Chromium	2.1E-02	1.8E-03	8.2E-05	8.2E-05
Lead	2.2E-03	1.9E-04	8.3E-06	8.3E-06
Mercury (Inorganic)	2.7E-03	2.3E-04	1.0E-05	1.0E-05
Organics				
Vinyl Chloride	4.4E-03	3.8E-04	1.7E-05	1.7E-05
Benzene	1.2E-02	1.0E-03	4.5E-05	4.5E-05
PAHs				
Benz(a)pyrene	3.9E-04	3.3E-05	1.5E-06	1.5E-06
Dioxins / Furans				
Dioxins & Furans	3.6E-08	3.1E-09	1.4E-10	1.4E-10

Table 6-11 Upset Scenario CR and ILCR Estimates

COC	Maximum Residential Location			
	1-hour	24-hour	Chronic	ILCR
Criteria Air Contaminants				
Sulphur Dioxide (SO_2)	4.8E-02	9.3E-02	2.7E-03	-
Nitrogen Oxides (NO_x)	3.9E-01	3.4E-02	7.5E-03	-
Hydrogen Chloride	6.3E-01	2.0E-01	2.0E-02	-
Particulate Matter (PM_{10})	-	1.9E-02	2.1E-03	-
Particulate Matter ($PM_{2.5}$)	-	1.9E-02	2.1E-03	-
Inorganics				
Arsenic	2.9E-03	1.7E-04	1.5E-04	9.7E-09
Cadmium	1.0E-02	3.5E-03	7.7E-04	3.8E-08
Chromium (Total)	2.1E-02	1.2E-03	1.4E-06	-
Lead	1.5E-03	3.8E-04	1.7E-05	-
Mercury (Inorganic)	4.5E-03	1.2E-04	1.2E-04	-
Organics				
Vinyl Chloride	2.2E-07	3.8E-04	1.7E-07	1.5E-10
Benzene	6.9E-05	3.5E-05	1.5E-06	9.9E-11
PAHs				
Benz(a)pyrene	3.9E-01	3.0E-02	-	1.6E-09
Dioxins / Furans				
Dioxins & Furans	-	6.1E-04	1.7E-05	-
Mixtures				
Respiratory irritants	1.1E+00	3.7E-01	3.5E-02	

These results indicate that there are no acute or chronic impacts to human health expected as a result of emissions during upset conditions.

7.0 UNCERTAINTY ANALYSIS

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

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

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

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

Table 7-1 Major Assumptions Used in the HHRA		
Risk Assessment Paradigm	***Assumption***	***Discussion of Conservatism***
Problem Formulation	Four receptor locations were selected in the area surrounding the proposed facilities to represent the sensitive or highly exposed individuals living, working or playing in the surrounding community.	Care was taken to select locations in the surrounding community that would likely demonstrate the highest potential impacts from the proposed facility. Residential receptor locations were intended to represent geographical areas occupied by a number of homes near the proposed facility. However, the inclusion of unique receptors such as school, child care, and long term care facility receptors represents the inclusion of very sensitive individuals in the assessment (*e.g.*, young children or the elderly).
	A7 and MOE paper for chemical selection	Chemical selection was based on the COC lists published in relevant MOE documents. This list is considered representative of municipal solid waste incineration emissions. Changing waste compositions as well as changing facility capabilities may impact the nature of facility emissions. That said, the COC list is considered comprehensive and representative for the current assessment.
Exposure Assessment	Maximum predicted short term (*i.e.*, for 1-hour and 24-hour exposure durations) ground-level air concentrations at each receptor location were used to evaluate all acute inhalation risk estimates.	In reality, the frequency with which the maximum would occur at any one receptor location varies with respect to the chemical of concern and the receptor location. Individual exposure to a 1-hour or 24-hour maximum ground-level air concentration requires that a receptor (person) be present at the same time and duration of the maximum predicted air concentration at that particular receptor location.
	Impact on greenhouse vegetable quality	In order to evaluate the impact of the facility on vegetable grown in the greenhouses, as scenario was considered where deposition rates at the MPOI coincided with the air intake vents for the greenhouses and it was assumed that all greenhouse vegetable were impacted by these maximum deposition rates.
	Annual average ground-level air concentrations and chemical-specific deposition rates were used to predict various environmental media concentrations (*e.g.*, soil and garden vegetables) assuming that deposition had already occurred for 70 years.	For purposes of conservatism in the assessment, location-specific deposition rates and ground-level air concentrations were used to predict concentrations of the COCs in local produce at receptors. Deposition was conservatively assumed to have occurred for 70 years prior to exposure, resulting in the maximum predicted environmental media concentrations being employed.
	Given the level of uncertainty and the variability associated with both behavioural and physical characteristics displayed by individuals, receptor characteristics were purposely selected to overestimate potential exposures for all individuals.	For example, to ensure that estimation of risk was conservative, oral/dermal exposures as part of the multi-pathway assessment were evaluated for the most sensitive residential receptor at receptor location. In reality, children (the most sensitive receptors) may actually be at schools, child care facilities, or long term care facilities for some part of a 24-hour exposure period. These conservative assumptions (receptors spending all their time at that specific location) likely would greatly overestimate those risk predictions for non-residential locations (*e.g.*, shopping/commercial areas or green space).
	The facility was assumed to have an operating lifetime of 30 years.	As indicated in the Air Quality Report, it is unlikely that the boiler facilities would be decommissioned until such time that the greenhouses were no longer viable operations (Chandler, 2011). Boilers housed in buildings at greenhouse facilities would typically be refurbished with new

Table 7-1 Major Assumptions Used in the HHRA		
Risk Assessment Paradigm	***Assumption***	***Discussion of Conservatism***
		equipment at the end of the boilers effective life (typical equipment life on the order of 20 to 25 years with appropriate maintenance activities).
	Residential receptors were assumed to be present at their respective locations 24 hours/day, 7 days/week, 52 weeks/year for 30 years when evaluating multimedia (non-inhalation) exposures.	When considering cancer risks in the multimedia scenarios, the assessment did not consider any time spent away from the exposure location during the 30 year exposure period required for the evaluation of carcinogenic compounds.
	All oral exposures to COCs were assumed to have 100% "bioavailability" at the receptor.	The magnitude of direct toxicological impact of a chemical upon a receptor is dependent upon that fraction of the ingested quantity of the chemical that is actually absorbed into the blood stream, and thus available for toxicological effect at the target tissue or organ within the receptor's body. Complete absorption of a chemical almost never occurs. Some fraction is not absorbed, but is excreted from the body, and not available to produce the relevant health impact. For the current assessment it was assumed that 100% of all ingested/dermally exposed chemical concentrations were absorbed into the blood stream, and would therefore express a toxic potential.
Exposure Assessment (continued)	Transfer of COCs to aboveground vegetation was assumed to occur by wet/dry deposition, root uptake and air to-plant transfer. In aboveground protected produce, concentrations of each of the COCs were assumed to result from root uptake to aboveground produce alone. In root vegetables, chemical concentrations were assumed to be due to root uptake to belowground produce.	Vegetables are considered increase the risk of exposure to COCs by all three routes, while aboveground protected produce such as corn, squash and green peas are considered protected enough to only be exposed to COCs *via* root uptake.
Toxicity Assessment	Toxicity reference values (TRVs) have been developed by regulatory agencies with sufficient conservatism assure protection of the sensitive and more susceptible individuals within the general population (*e.g.*, infants and young children, the elderly, individuals with compromised health).	A considerable amount of conservatism is incorporated in the TRVs. These benchmarks are deliberately set by regulatory agencies with the protection of sensitive individuals in mind. Typically, the benchmarks used in the current assessment were derived from the most sensitive health-related endpoints, and then adjusted to account for differences in sensitivity to chemicals among individuals. The use of uncertainty factors is directed, in part, toward the protection of sensitive individuals. The most sensitive toxicological endpoint (for example, decreased growth, body weight loss/gain, reproductive effects) was selected for each chemical from the available scientific literature to represent the exposure limit (TRV).

Table 7-1 Major Assumptions Used in the HHRA		
Risk Assessment Paradigm	***Assumption***	***Discussion of Conservatism***
	Canadian National Ambient Air Quality Objectives (NAAQO) were not used as benchmarks for a number of the CACs (specifically SO_2 and NOx).	An alternative to the use of the NAAQOs for these two COCs are the air quality guidelines (AQGs) recommended by WHO (2005). However, these AQGs have not been yet accepted as appropriate regulatory standards for the evaluation of air quality, and there are concerns as to whether it is appropriate to apply these guidelines to a localized area around a specific discrete emission source, rather than for the establishment of regional air quality objectives. It should be noted that the Ontario MOE has not yet replaced the current NAAQOs with the WHO AQGs (or other more recent proposed guidelines) as part of their air quality regulatory framework.
Toxicity Assessment (continued)	Ontario MOE 24-hour Ambient Air Quality Criteria (AAQC) were used to evaluate acute health risks for the 24-hour exposure period.	In the absence of any available acute-specific TRVs in the regulatory literature, 24-hour AAQC specified by the MOE under O. Reg. 419 were used to evaluate acute 24-hour health risks. Though they are expressed as a 24-hour exposure period criteria, due to the nature of how the MOE derives their 24-hour AAQC, these typically are based on **chronic** endpoints, rather than acute. As chronic TRVs are typically much more conservative than acute TRVs for the same COC, it is expected that this will result in an overestimation of potential acute risks. In the absence of acute TRVs, this is viewed as a conservative approach to evaluating risks for this exposure period.
	For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions.	The existence of enzymes and biological pathways that routinely repair damage to genetic material (DNA) is well documented in the scientific literature. The potential adverse health outcomes arising from damage to DNA is usually observed only when the ability of these repair enzymes to "fix" the damage is blocked or exceeded.
	Large uncertainty factors (*i.e.,* 100-fold or greater) were used in the estimation of the TRVs for threshold type chemicals.	Uncertainty factors were applied at exposure levels reported in animal or human studies where no adverse effects were observed (*i.e.,* NOAEL). Thus, exceeding the toxicological criterion should not mean that adverse health outcomes would occur. Rather, it means that the uncertainty factor beyond the no-effect exposure is somewhat reduced.
Toxicity Assessment (continued)	Possible interactions of the COCs present in emissions from the proposed facility that might lead to enhanced toxicity were evaluated in the assessment.	Additive interactions were included as part of the assessment after consideration of factors including chemical structure, target tissue(s), and mechanism of toxic action. However, the evaluation of risks related to chemical exposures in mixtures is an emerging science. There are currently no regulatory benchmarks or specific guidance (beyond those chemical groups that have established toxic equivalency factors or TEFs) by which one could evaluate whether exposure to a given chemical mixture could pose a health concern. As such, comparisons of the additive CR, HQ, or ILCR values for each mixture with the relevant benchmark (*i.e.,* 0.2, 1.0, and 1-in-1,000,000, respectively) are provided for information only.
	Evaluation of the cPAHs	Benzo(a)pyrene was selected to be representative of the carcinogenic PAHs. We acknowledge that this underestimates carcinogenic risk; however, given the three to four orders of magnitude of safety in all B(a)P

Table 7-1 Major Assumptions Used in the HHRA		
Risk Assessment Paradigm	***Assumption***	***Discussion of Conservatism***
		carcinogenic risk estimates, we are confident that consideration additional carcinogenic PAHs would not affect the conclusions of the HHRA.
	Humans were assumed to be the most sensitive species with respect to toxic effects of the COC.	For obvious reasons, toxicity assays are not generally conducted on humans, so toxicological data from the most sensitive laboratory species were used in the estimation of toxicological criteria for humans.

8.0 CONCLUSIONS AND RECOMMENDATIONS

The purpose of the current assessment was to evaluate the potential human health implications associated with air emissions from the proposed REMASCO Gasifier Installations. Based on conservative air dispersion and deposition modeling, the potential impacts of projected emissions from the REMASCO Gasifier Installations were estimated in order to determine the health implications to potentially sensitive individuals living, working, or playing in the surrounding communities, under "worst case" exposure conditions.

8.1 Acute Inhalation Assessment Results

The results of the acute inhalation assessment indicated that there are no acute impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community. In fact, most predicted concentrations ratios demonstrated that predicted ambient concentrations of the COCs were many orders of magnitude below the corresponding regulatory benchmarks.

8.2 Chronic Inhalation Assessment Results

The results of the chronic inhalation assessment indicated that there are no chronic impacts to human health expected as a result of facility emissions to the ambient air of the surrounding community. Most predicted concentrations ratios and incremental lifetime cancer risk levels demonstrated that predicted ambient concentrations of the COCs were many orders of magnitude below the corresponding regulatory benchmarks.

8.3 Chronic Multi-Pathway Results

The results of the chronic multimedia (*i.e.*, inhalation, oral and dermal exposures) assessment indicated that there are no chronic impacts to human health expected as a result of deposition of facility emissions onto soils and home gardens of residences in the surrounding community. In fact, most predicted hazard quotients and incremental lifetime cancer risk levels demonstrated that predicted concentrations of each of the COCs in soil and home garden produce (where applicable) at the various sensitive receptor locations were many orders of magnitude below the corresponding regulatory benchmarks.

Furthermore, the worker scenario and the milk and produce consumer scenarios also indicated that there are no chronic impacts to human health expected as a result of these scenarios.

8.4 Cumulative Assessment Results

Evaluation of potential exposures under current and future cumulative conditions indicate marginal exceedances of the acute and chronic TRVs for NOx and $PM_{2.5}$ at several receptor locations. Mixture effects are also noted at several locations. In all cases, future cumulative risks with the proposed REMASCO facilities are equal to or lower than risks predicted under existing background conditions. There were no cases in which the added emissions from the proposed REMASCO facilities alone resulted in cumulative risk above that already existing under current (background) conditions. As a result, there will be a net benefit to the installation and the operation of the REMASCO facilities.

8.5 Upset Scenarios

Evaluation of potential exposures under upset conditions at the maximum residential receptor location indicate that there are no acute or chronic impacts to human health expected as a result of emissions during upset conditions.

9.0 SCREENING LEVEL ECOLOGICAL RISK ASSESSMENT

REMASCO have been operating various gasifiers to generate hot water for the heating systems of the Southshore property for approximately 24 months. Over the last 12 months, the production version of the gasifier has been operated, tested, modified and tested again to ascertain performance with respect to operating efficiency, and most importantly, emissions of contaminants to the atmosphere. Given the nature of the fuel being used in the gasifier, the MoE requires that the facility meets the A7 Emission Guidelines applied to MSW incinerators operating in the province. Testing has shown that the gasifiers can meet this standard.

The A7 Guideline is generally considered to be a technology-based standard that sets a performance level for the emission control system that is deemed to be necessary for such facilities. At this performance level, it is generally accepted that there will be minimal impacts on the environment and human health. Regardless of this assumption, a screening-level ecological risk assessment (ERA) for the proposed REMASCO facilities in Kingsville was conducted to ensure protection of local plants (including crops and greenhouse plants), invertebrates and wildlife. The assessment was based on the predicted concentrations of chemicals in soil and air as presented in the HHRA (see Section 4).

Screening of COCs in Soil

Table 9-1 shows a comparison between the predicted chemical concentrations in surface soil at the Maximum Point of Impingement (MAX POI) to the ecological component values of the Ontario Ministry of the Environment Site Condition Standards (Table 2, agricultural land use, coarse-textured soil; MOE, 2009a). Predicted surface soil concentrations are several orders of magnitude below concentrations assumed to be protective of plants, soil invertebrates, birds and mammals.

Table 9-1 Screening of Predicted Soil Concentrations to Ecological Component Values

Chemical	*Estimated Surface Soil Concentration (µg/g)*	*Ecological Component Value[a] (µg/g)*	
		Plants/Soil Invertebrates	*Mammals/Birds*
Arsenic	3.91E-04	20	51
Cadmium	6.67E-04	12	1.9
Chromium	1.42E-02	310 (total), 8 (Cr IV)	160 (total), 910 (Cr IV)
Lead	1.45E-03	250	32
Mercury	1.80E-03	10	20
Benzene	1.98E-11	25	370
Vinyl Chloride	2.83E-13	3.4	6.8
Dioxins & Furans (TEQ)	2.35E-09	NV	1.30E-05
Benzo(a)pyrene	1.66E-05	20	1600

[a] MOE Table 2 value for agricultural land use, coarse-textured soil (MOE, 2009a)

Based the comparison of predicted surface soil concentrations to ecological component values, there is no need to consider exposure of plants, soil invertebrates, birds or mammals to chemicals in soil.

Screening of COCs in Air

Table 9-2 shows a comparison between the predicted maximum chemical concentrations in air to the Ontario, World Health Organization, and Health Canada Ambient Air Quality Criteria

(MOE, 2008; WHO, 2005; Health Canada, 2006). Predicted air concentrations are below concentrations assumed to be protective of plants.

Table 9-2 Screening of Predicted Air Concentrations to Air Quality Criteria

Chemical	*Predicted Maximum Air Concentration ($\mu g/m^3$)*	*MOE AAQC ($\mu g/m^3$)[a]*	*WHO AAQG ($\mu g/m^3$)[b]*	*HC NAAQO ($\mu g/m^3$)[c]*	*Averaging Time*
Sulphur dioxide	7.06	690	500	450	1 h
	3.74	275	20	150	24 h
	0.20	55		30	annual
Nitrogen oxide[d]	76.3	400	200	NV	1 h
	24	200	NV	NV	24 h
	1.22	NV	40	60	annual
Hydrogen chloride	10.9	NV	NV	NV	1 h
	5.75	20	NV	NV	24 h
	0.31	NV	NV	NV	annual

NV No Value
[a] Ontario's Ambient Air Quality Criteria (MOE, 2008)
[b] World Health Organization Air Quality Guidelines (WHO, 2005)
[c] Health Canada National Ambient Air Quality Objectives (Health Canada, 2006)
[d] Predicted maximum air concentrations measured as nitrogen oxides (NO_x).

Based on the comparison of predicted maximum air concentrations to air quality criteria, there is no need to consider exposure of plants to chemicals in air. However, actual plant protection benchmarks were not obtained or derived for this comparison. In addition, predicted cumulative concentrations of sulphur dioxide, nitrogen dioxide and hydrogen chloride are expected to be significantly greater than those concentrations shown in Table 9-2 resulting from the proposed facilities. Therefore, a preliminary review of the literature was completed to compile plant-specific benchmarks in order to assess risks specifically for plants, including crop. The data used to derive preliminary benchmarks are described below and benchmarks provided in Table 9-3.

Concentrations as low as 6,500 $\mu g/m^3$ HCl caused 10% injury to ornamental plants (flowers) when exposed for 20 minutes (Lerman *et al*., 1976). Concentrations from 15,000 to 19,000 $\mu g/m^3$ HCl caused foliar injury in pinto beans when exposed for 20 minutes (Swiecki *et al*., 1982). Exposure to 27,000 $\mu g/m^3$ for 20 minutes, 12-19 times over one day, resulted in a 29% increase in number of leaves showing injury in pinto beans and radish (Granett and Taylor, 1981).

Exposure to 2,500 $\mu g/m^3$ NO for 1 hour caused a 12% reduction in photosynthesis in lettuce (Caporn, 1989). This was the only 1-hour exposure experiment found during the preliminary review of the literature. Concentrations as low as 78 $\mu g/m^3$ NOx caused severe damage to epistomatal wax structures in Norway spruce seedlings exposed for 19 days (Viskari *et al*., 2000). Use of this concentration as a benchmark for 24-hr exposures is conservative due to the longer time of exposure, and the presence of other VOCs in the exhaust gas to which the seedlings were exposed. Exposure to 15 $\mu g/m^3$ NO for 6-7 months caused up to an 11% decrease in shoot weight of grass (Lane and Bell, 1984).

Colls *et al*., (1992) concluded that for SO_2 concentrations ranging from 77 to 500 $\mu g/m^3$, plant response is determined by the cumulative dose or the average concentrations rather than by

intermittent peak exposures. Van der Eerden and Tonneijck (1988) found that the average ambient SO_2 concentration in the Netherlands (24 µg/m^3) result in a 1% total crop volume loss. Dueck *et al*., (1992) used a probabilistic approach to determine a threshold of 8 µg/m^3 SO_2 at which 95% of the species in a Dutch heathland community would be protected over a 42-day period.

Lorenzini *et al*., (1990) found an average yield reduction in four wheat cultivars exposed to 210 µg/m^3 (74 ppb) of SO_2 of 15.5%. Depressed biomass and yield, but no visible signs of injury, were also observed in barley, maize, perennial ryegrass and Italian ryegrass following long-term exposures to low levels of SO_2.

SO_2 exposure can also modify the response of plants to biotic and abiotic stresses, often exacerbating their adverse impacts (WHO, 2000; Adaros *et al*., 1991a,b; Ashmore *et al*., 1988; Mooi, 1984). There is evidence that concurrent exposure to SO_2, and ozone (O_3), and/or nitrogen dioxide (NO_2) at concentrations near their critical levels can produce markedly increased adverse impacts under some circumstances (WHO, 2000). It has also been shown that the interaction of low-temperature stress with low concentrations of SO_2 can lead to increased damage (Makela *et al*., 1987).

Predicted maximum air concentrations associated with the proposed REMASCO facilities ("REMASCO alone") and maximum predicted cumulative air concentrations for the area ("Future Conditions with REMASCO - Cumulative") were screened against these benchmarks (Table 9-3). Predicted cumulative air concentrations ("Current Conditions - Cumulative") under existing conditions are also provided to illustrate the anticipated decrease in cumulative local air concentrations with the addition/conversions of the proposed REMASCO facilities.

Table 9-3 Screening of Predicted Air Concentrations to Plant Benchmarks

Chemical	*Maximum Air Concentrations (µg/m^3)*			*Benchmarks (µg/m^3)*		*Averaging Time*
	Current Conditions - Cumulative	*REMSCO Alone*	*Future Conditions with REMASCO - Cumulative*	*Tree/Ornamental Plant Benchmark*	*Crop Benchmark*	
Sulphur dioxide	NV	7.06	NV	NV	NV	1 h
	NV	3.74	NV	NV	NV	24 h
	NV	0.20	NV	8	24	annual
Nitrogen oxide[a]	226	76.3	198	NV	2500	1 h
	180	24	176	78	620	24 h
	49.9	1.22	37.5	15	NV	annual
Hydrogen chloride	NV	10.9	NV	6500	15,000	1 h
	NV	5.75	NV	NV	27,000	24 h
	NV	0.31	NV	NV	NV	annual

NV No Value
[a] Predicted air concentrations measured as nitrogen oxides (NOx).

Predicted maximum air concentrations, attributed to the proposed facilities alone, are below all plant-specific benchmarks (Table 9-3). Predicted maximum cumulative air concentrations for nitrogen oxides are below crop benchmarks while marginally exceeding 24-hour and annual average tree/ornamental plant benchmarks.

These marginal exceedances are not considered likely to be of ecological significance given that the magnitude of exceedance is low. Also, consideration may be given to the basis of the annual average tree/ornamental plant benchmark. The critical effect level of 15 µg/m^3 NO represents adverse effects on two grass species exposed over 6-7 months. Toxicity of NO_2 to plants is less than that of NO. Therefore, this preliminary benchmark may be conservative because it assumes exposure only to NO. However, there are limited data available investigating the toxicity of nitrogen oxides to plants and thus, there is uncertainty surrounding the level of conservatism built into the preliminary benchmarks for nitrogen oxide.

As demonstrated in Table 9-3, predicted cumulative air concentrations are below those anticipated under existing background conditions. While benchmarks are exceeded at the maximum cumulative air concentrations, overall, cumulative air quality is improving with the addition/conversion of the proposed REMASCO facilities.

Conclusions

Based on the comparison of predicted surface soil concentrations to ecological component values, no unacceptable impacts to plants, soil invertebrates, birds or mammals from exposure to chemicals in soil are expected. Based on comparison of predicted maximum air concentrations emitted from the proposed facilities to air quality guidelines and preliminary plant-specific benchmarks, no unacceptable impacts to plants are expected. While marginal exceedances of preliminary plant-specific benchmarks for nitrogen oxides were predicted at future cumulative air concentrations, overall, anticipated risks to plants would decrease compared with existing conditions.

10.0 DOCUMENT SIGN-OFF

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

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