

PROJECT NO. B.C.V50396

REPORT TO:

NORSKECANADA

FINAL DRAFT

**BASELINE AIR QUALITY MODELLING AND HUMAN HEALTH RISK ASSESSMENT
CURRENT DAY EMISSION FROM NORSKECANADA CROFTON**

VOLUME II: HUMAN HEALTH RISK ASSESSMENT

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EXECUTIVE SUMMARY

Jacques Whitford Limited (Jacques Whitford) was retained by NorskeCanada Crofton Division to conduct a Human Health Risk Assessment (HHRA) relating to its aerial emissions from its Crofton Division operations in Crofton, British Columbia.

The NorskeCanada Crofton Division is an integrated pulp and paper facility that produces newsprint, directory paper and market pulp. The mill is located on the southeast coast of Vancouver Island on Stuart Channel across from Saltspring Island. The mill is situated less than a kilometer north of the community of Crofton (population 2,500) and 5 km north of the City of Duncan (population 4,700).

In March 2004, NorskeCanada initiated the “Crofton Mill Community Engagement Process”. This process has two primary aims:

Strengthen NorskeCanada’s understanding of community concerns and issues related to the operations of the Crofton Mill and its impacts on the communities of the Cowichan Valley

Gather the views of a broad range of community groups and individuals representing the diverse interests of the communities of the Cowichan Valley

In April, twisurveys Inc. conducted numerous focus group sessions with local stakeholders to gather information and concerns of residents with respect to operations at the Crofton Mill. One of the Key Findings of this process was a community interest in having a baseline air quality study and accompanying human health risk assessment conducted on current day aerial emissions from the Crofton Mill.

Scope and Objectives

The human health risk assessment provided in this report is meant to inform on the potential human inhalation health



impacts from current day mill operations on health of the local community. Although, under no regulatory obligation to do so, NorskeCanada is interested in ensuring that their operations are not negatively impacting on the health of local residents. This study considers only current day emissions and mill operations and is not intended to address issues surrounding alternative fuel trials at the mill.

The scope of work consists of the following three tasks:

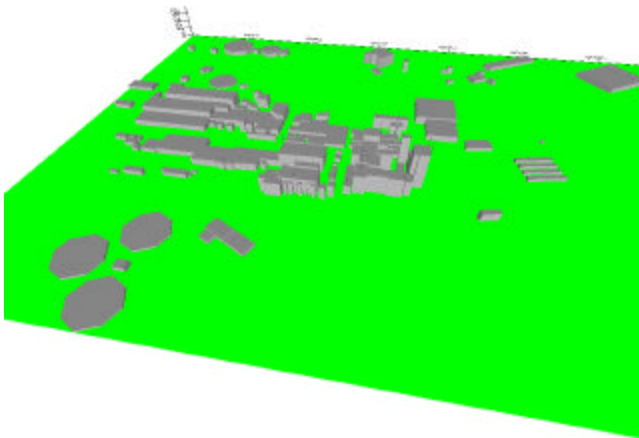
Review of ambient air quality monitoring results from the three existing monitoring stations in the Crofton Air shed to detect temporal trends and evaluate potential risk to human health from measured airborne levels of substances of potential concern (SoPC).

Modelling of ground-level concentrations of SoPC in ambient air from current day emissions from the Crofton Mill (Vol. I.)

Determining whether or not modelled concentrations of SoPCs in ambient air pose a potential risk to the health of the residents of the Crofton Air shed.

Facility Overview

The mill complex is located on flat coastal lowland immediately adjacent to Stuart Channel. Local relief on the order of 400 metres (1,300 ft) is experienced within 4 to 6 km of the site. The nearest permanently occupied dwellings are located within 100 metres of the mill property line (Crofton Mobile Home Park), however the bulk of Crofton's population reside within 1 to 3 km of the centre of the mill. Saltspring Island lies some 5 km to the east, across Stuart Channel.



Crofton Division began operation in 1957 as a single-line kraft pulp mill. Today, three paper machines and two pulp machines have an annual capacity of 680,000 tonnes of product. The mill produces 430,000 tonnes of groundwood paper annually, 280,000 tonnes of which is newsprint and 150,000 tonnes of which is directory paper. The remainder of Crofton's capacity is market pulp, some of which is used in their papermaking processes.

Crofton's paper is manufactured from combinations of mechanical pulps, kraft pulp, fillers, pigments and recycled newspaper and magazines. Thermomechanical pulp is made by heating wood chips under pressure to soften them, and then by grinding the softened chips between serrated metal plates to separate the fibres from the wood structure.

Kraft pulp is made by cooking wood chips in a solution of caustic soda and sodium sulphide to separate the wood fibres from the lignin, a glue-like binding substance. Crofton's two chemical pulping lines produce northern bleached softwood kraft pulp (NBSK). Crofton's pulp and paper products are made almost entirely from residual wood fibre, waste wood from B.C. sawmills that was once consigned to landfills or burned.

Regional Setting

The Crofton Division Mill is located adjacent to the town of Crofton, in the Cowichan Valley Regional District, on Vancouver Island, British Columbia. While the population of the Cowichan Valley is approximately 79,000, the town of Crofton has a population of 2,500.

The Cowichan Valley is located between the Cities of Victoria and Nanaimo, and is surrounded by scenic mountains and valleys. The area has been home to numerous First Nations for countless centuries and was settled by European farmers and loggers in the mid 1800s.

Residential Areas

The entire area surrounding Crofton Division is populated with residential dwellings, from the Crofton Mobile Home Park located within metres of the fenceline to the town of Crofton located approximately 1 km from the mill site.

First Nations

The traditional use of land by First Nation communities in the vicinity of Crofton Division is well documented. In fact the name "Cowichan" was derived from the Coast Salish word Khowutzun, literally translates into "The Warm Land". A number of First Nation Reserves exist within the Cowichan Valley Regional District, including: Cowichan, Chemainus, Halalt, Squaw-hay-one, Tsussie, Claoose, Kil-pah-las, Kuper Island, Lyacksun,

Shingle Point, Malachan, Malahat, Cowichan Lake, Portier Pass, Theik, Wyah , and Oyster Bay.

Given the relative proximity of these communities to the Crofton Division, it is reasonable to assume that the forested areas and waters, have historically and are currently been used by the Aboriginal Community for their hunting, fishing, and gathering activities.

By virtue of their Aboriginal Rights, First Nation communities have a direct stake in the use of the forests and natural resources for their traditional native fishing, hunting, and gathering activities. Therefore, formal discussions and information exchanges were undertaken by NorskeCanada with several First Nation communities and organizations as part of the consultation phase of the project

These meetings were aimed at sharing information about the Crofton Division operations to First Nations.

Climate

The Cowichan Valley is reported to have the highest mean annual temperature of any region in Canada. Climate data were obtained from a representative Environment Canada weather station at Duncan, B.C. The average daily temperature is a temperate 9°C, with a daily minimum temperature rarely falling below 0°C and a maximum daily temperature typically in the mid 20°C range.

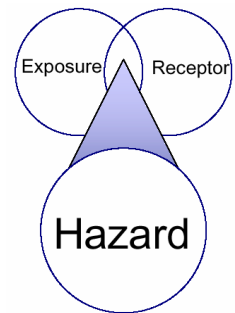
The average yearly rainfall is 993 mm and snow fall of 43 cm. Given the temperate climate the residents of this area likely spend more time outdoors than many other places in Canada and likely have their windows open for more days a year as well.

For detailed meteorological data refer to the Jacques Whitford Air Modelling report. Briefly the predominant winds in the Crofton area are from the north; however, there is a great deal of variability seasonally around the mill.

Problem Formulation

Facility Emissions

The NorskeCanada Crofton Mill emissions were developed from the mill's submission to Environment Canada (EC) for the National Pollutant release Inventory (NPRI) for the 2003 reporting year (the most recent year available). The NPRI report was developed by NorskeCanada following the reporting requirements outlined in the EC Supplementary Guide for Reporting to the National Pollutant Release Inventory 2003.



The NorskeCanada emissions estimates for their 2003 NPRI were developed from mill production data for 2003, and emissions factors for various processes developed by the National Council for Air and Stream Improvement (NCASI) or from site specific testing. The use of the NCASI emission factors for estimation is standard practice for the industry. The NCASI emission factors were supplemented (where available), with site-specific emission factors based on Crofton mill stack testing data (2002) collected as part of a study by the Forest Products Association of Canada (FPAC). These site-specific emissions factors were used in place of the NCASI factors by NorskeCanada wherever possible.

It should be noted that NorskeCanada included 106 substances listed in their NPRI calculations to determine the substances whose 2003 release rates exceeded the NPRI reporting thresholds. Only substances exceeding their NPRI reporting threshold (the thresholds are substance specific) are required to be reported to Environment Canada. Of the 106 substances, 36 were estimated to have zero emissions. The remaining 70 substances with non-zero emission rates were included in the dispersion modelling.

The SoPCs carried forward in the risk assessment are provided in the Table below.

Air Modelling

The air quality study was conducted to determine the ambient air concentrations of mill emissions at NorskeCanada's Crofton Division, British Columbia (B.C.) mill using 2003 emissions data. Since the mill is located in an area of complex terrain and ocean/land atmospheric interactions, the CALMET / CALPUFF dispersion model was selected for the analysis. CALPUFF is an integrated puff model capable of modelling instantaneous or continuous releases over distances ranging from tens of metres to hundreds of kilometres. The model contains a meteorological pre-processor (CALMET) which produces 3-dimensional flow fields of wind speed, wind direction, temperature, mixing layer height, atmospheric turbulence, etc. The meteorological pre-processor accounts for complex terrain effects on the wind field.

The primary purpose of the dispersion modelling study was to produce conservative predictions of the maximum and average ground level concentrations of approximately 80-substances emitted from the mill, as calculated for NorskeCanada inventory for internal / external reporting purposes.

The meteorological data were processed using the California Air Resources Board/Environmental Protection Agency (CARB/EPA) meteorological pre-processor CALMET. The CARB/EPA dispersion model CALPUFF was then used to calculate substance ground level concentrations over a 45-km by 55-km domain in the vicinity of the facility.

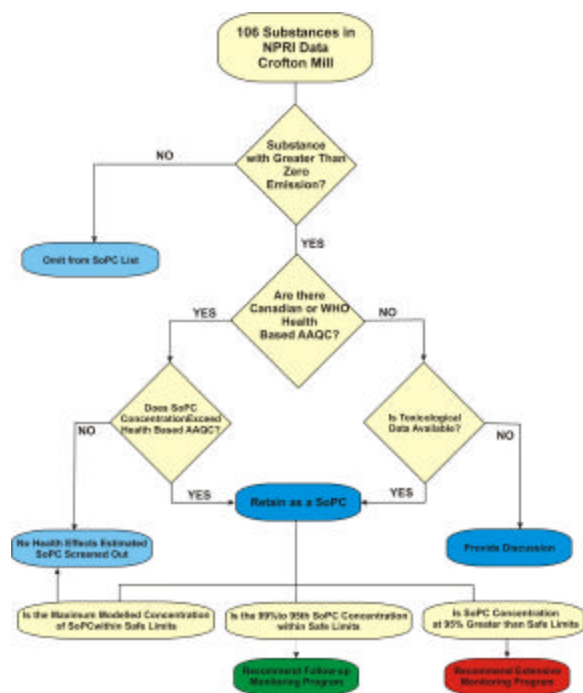
The air modeling approach and results are discussed throughout this report. However, for full details of the air modeling report refer to Volume I: Air Quality Modeling.

Final SoPC List

<u>Modelled SoPC Compounds</u>		
<u>Inorganic Elements</u> Antimony Arsenic Cadmium Chromium Chromium VI Cobalt Copper Lead Manganese Mercury Nickel Selenium Silver Zinc	<u>Combustion Gases</u> Ammonia Carbon disulphide Carbon monoxide Chlorine Chlorine dioxide Hydrochloric acid Hydrogen fluoride Hydrogen sulphide Nitrogen oxides Total particulate matter (TPM) Particulate (PM _{2.5}) Particulate (PM ₁₀) Sulphur dioxide Sulphuric acid	<u>VOCs/SVOCs/Miscellaneous</u> Acetaldehyde tetrachloroethylene Acetone 1,2,4-trichlorobenzene Acrolein 1,1,2-trichloroethane Biphenyl Trichloroethylene Carbon tetrachloride 1,2,4-Triomethylbenzene Chloroform Chloromethane Cresol Cumene Dichloromethane Formaldehyde Hexachlorobenzene (HCB) Isopropyl Alcohol Methanol Methyl ethyl ketone Methyl isobutyl ketone Napthalene Phenol Propionaldehyde styrene
	<u>Polycyclic Aromatic Hydrocarbons (PAHs)</u> Benzo(a)pyrene (TEQ) 12 Carcinogenic PAHs	
<u>Hydrocarbons</u> Benzene Toluene Xylenes - total	<u>Dioxins/Furans</u> 2,3,7,8 – TCDD (TEQ)	

SoPC Screening and Risk Assessment Framework

The SoPCs considered in this assessment were based on the Crofton Division's 2003 NPRI emissions report. The screening framework, by which the SoPCs were evaluated, screened and risk characterization for this assessment conducted is shown below.



Exposure Pathways

Although there is only one significant source of the SoPCs (stack emissions), there are numerous pathways by which human receptors could be affected by the emissions.

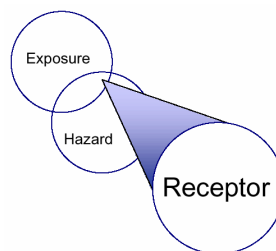


However, this report only considers the exposure pathway of inhalation of outdoor and indoor vapours and particulates resulting from stack or source emissions from Crofton Division.

SoPC concentrations in air were either obtained from ambient air monitoring program or from calculations of ambient air concentrations from CALPUFF. Air concentrations used in the evaluation of chronic health risk were estimated using annual average values whereas air concentrations used in the evaluation of acute health risks were calculated using hourly and 24 hour values.

Receptor Characteristics

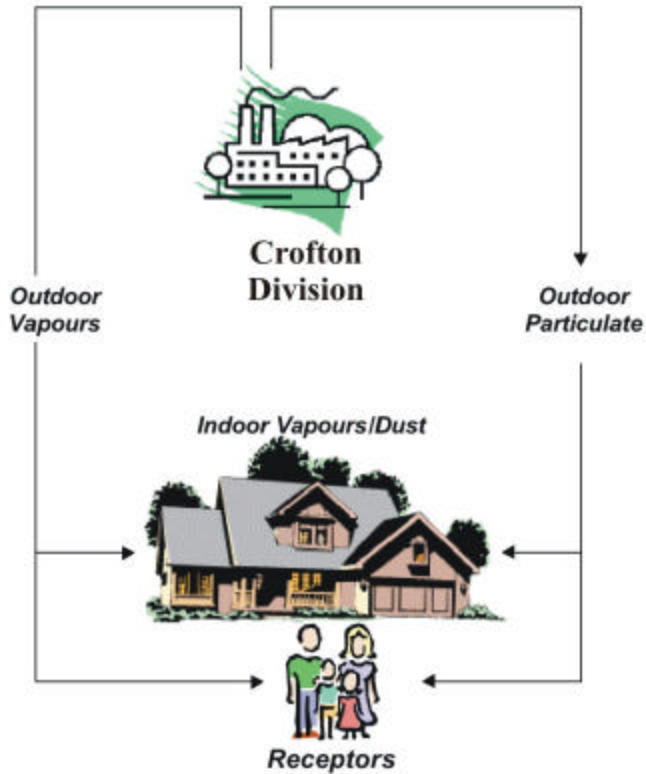
The purpose of this section is to identify the non-worker related human activities and receptors in the assessment area that may be impacted as a result of exposure to Crofton Division emissions. Receptor characterization is generally focused on identifying current and reasonably foreseeable human activities or land uses that apply to the general public. Potential worker-related exposures are governed by applicable legislation and guidelines, and is outside the scope of work for this project.



Given that only the inhalation exposure pathway was modeled, inhalation unit risks and reference concentrations were employed. Consideration was given to all human life stages, from birth to old age in the selection of toxicity reference values. Note that these are not generally age specific for inhalation exposure pathways. Residents were considered to live in the area 365 days per year over an entire lifetime.

Conceptual Model

As indicated, residents in the surrounding communities can be directly exposed to vapours and particulates both outside in the ambient air and within their homes. All SoPCs were assessed for these exposure routes.



Toxicity Assessment

The toxicity assessment provided in this assessment was based on either Health Based Ambient Air Quality Guidelines/Criteria (AAQC), or in the absence of these criteria toxicity reference values.

B.C. Air Quality Objectives (AQO)

The B.C. Air Quality Objectives (AQO) are used for guidance in the issuance of Permits under the *Environmental Management Act* (replaced the *Waste Management Act* on July 8, 2004). They are also used in project review referrals under the B.C. Environmental Assessment Act (RWDI, 2002). The B.C. AQOs serve as benchmarks for short-term

assessments (less than 24 hours) and for long-term air shed management (annual).

National Ambient Air Quality Objectives (NAAQOs)

Canadian federal National Ambient Air Quality Objectives (NAAQOs) identify benchmark levels of protection for people and the environment for substances in the air. NAAQOs are meant to guide federal/provincial/territorial governments in making risk-management decisions, playing an important role in air quality management, and as benchmarks for developing provincial objectives and standards. NAAQOs are viewed as effects-based long-term air quality goals.

Ontario Ministry of the Environment Ambient Air Quality Criteria (AAQC)

The Ontario Ministry of the Environment (OMOE) publishes the most comprehensive set of health based guidelines for ambient air quality in Canada. These guidelines are found in:

Summary of Point of Impingement Standards, Point of Impingement Guidelines and Ambient Air Quality Criteria (AAQCs). (OMOE, 2001)

Although the guidelines contain several health based criteria, they also include odour based guidelines. For the purpose of this study, only those guidelines that were “health based” were used.

World Health Organization Guidelines

The World Health Organization has published a set of Health Based Guidelines for 35 substances, 16 of which are relevant to this study. These health based guidelines were developed to aid European countries in setting local standards for air quality. These guidelines are found in:

Air Quality Guidelines for Europe Second Edition (WHO, 2000)

The aim of the WHO guidelines is to provide a basis for protecting public health from adverse effects of air pollutants and to eliminate or reduce exposure to these compounds. In some cases the WHO guidelines are more protective, while in others the OMOE guidelines were more stringent. Therefore, modeled concentrations of SoPCs were compared to both sets of criteria.

Sources of Toxicological Information

The toxicity assessment includes toxicity values established by several regulatory agencies, including:

- Health Canada (HC);
- US EPA: Integrated Risk Information System (IRIS);
- World Health Organization (WHO);
- Agency for Toxic Substances and Disease Registry (ATSDR); and
- California Environmental Protection Agency (CalEPA).

For this assessment, preference was generally been given to toxicity values from Health Canada and IRIS. Where toxicological information was available from both of these sources, the supporting information for each value was reviewed and selection of the final TRV was based on the currency of the information and the level of protection. Other sources cited above were used when no toxicological information was available from Health Canada or IRIS.

Exposure Assessment and Risk Characterization

The exposure assessment can be divided into two distinct components; the ambient air quality monitoring that is routinely conducted by NorskeCanada and the air modeling exercise undertaken by Jacques Whitford (JWL, 2004).

The purpose of the risk characterization is to combine the information from the toxicity assessment and the results of the exposure assessment to estimate the potential risks to human health from the SoPC evaluated.

Ambient Air Monitoring

NorskeCanada is authorized to discharge substances to atmosphere from an integrated pulp and paper facility at Crofton, B.C. by the B.C. Ministry of Water, Land and Air Protection Air Permit PA-01902. The Permit is a legal document granted under the Waste Management Act that lists specific authorized discharges and related conditions. Section 13.4 of PA-01902 details the requirement of the Ambient Air Monitoring program.

The NorskeCanada Crofton ambient air quality monitoring network is currently comprised of three monitoring trailers located on a southerly transect approximately Crofton Substation (0.5 km), South Station (1.6 km) and Duncan-Deykin Station (4.6 km) distant from the facility.

At all three stations NorskeCanada is required to monitor particulate matter <math><10\mu\text{m}</math> (PM_{10}), and total reduced sulphur (TRS). It was determined that monitored concentrations of both PM_{10} and TRS did not pose a potential health threat to residents of the local air shed.

SoPCs Modelled Ground Level Concentrations With Health Based AAQC

Of the 41 compounds that have OMOE Health Based ambient air quality criteria the following were the observed to have concentrations greater than the OMOE criteria :

Fenceline Receptors

HCl (hourly and 24 hr)

NO_x (hourly)

SO₂ (hourly)

Special Receptors

HCl (hourly and 24 hr)

Maximum Gridded Receptors

HCl (hourly and 24 hr)

SO₂ (hourly)

An exceedance of an AAQC does not imply that an effect occurs, rather that it can not be ruled out entirely at this point. Therefore, HCl, NO_x and SO₂ were carried forward in the risk assessment for further understanding of the nature of the exceedances and their location. In addition SO₂ and NO_x were also found to exceed WHO guidelines. The remaining compounds had modeled concentrations below health based guidelines and therefore are at concentrations below which health based effects would be expected, thus were not considered further in the risk assessment.

Again it should be remembered that by its very nature the modeled results are meant to be conservative overestimates of the actual ground level concentrations in the local air shed. Therefore, it is possible that the concentrations of HCl, NO_x and SO₂ are actually below the guideline values.

SoPCs Modelled Ground Level Concentrations With No Health Based AAQC

Of the 16 substances of potential concern that do not have Health Based AAQC only hydrogen sulphide (H₂S) exceeded for each of the receptor groups across all of the timelines. The remaining compounds had modeled concentrations below health based toxicity reference values from other jurisdictions and therefore are at concentrations below which health based effects would be expected, thus were not considered further in the risk assessment.

Polycyclic Aromatic Hydrocarbons

The toxic equivalents (TEQs) method estimates the potential carcinogenic risk from exposure to PAHs by determining the toxic equivalence of the individual carcinogenic PAHs to benzo(a)pyrene (B(a)P), the most carcinogenic, and best studied of these compounds.

The Table below presents the results of PAH screening for the maximum concentrations of PAHs detected, which was at the Gridded Receptor. Given that PAHs are carcinogenic, then only the annual concentration is modeled using the TEQ methodology. It was determined that even at the highest concentrations of PAHs, there was no increased cancer risk above a benchmark of one person in one hundred thousand.

PAH Compound	Annual Concentration (mg/m ³)	Toxic Equivalency Factors	Annual Toxicity Equivalent (mg TEQ/m ³)
Benzo(a)anthracene	1.83E-05	0.1	1.83E-06
Benzo(a)phenanthrene	1.83E-05	0.01	1.83E-07
Benzo(a)pyrene	1.82E-05	1	1.82E-05
Benzo(b)fluoranthene	1.81E-05	0.1	1.81E-06
Benzo(g,h,i)perylene	1.81E-05	0.01	1.81E-07
Benzo(k)fluoranthene	1.81E-05	0.1	1.81E-06
Dibenzo(a,h)anthracene	1.80E-05	1	1.80E-05
Fluoranthene	2.02E-05	0.001	2.02E-08
Indeno(1,2,3-c,d)pyrene	1.81E-05	0.1	1.81E-06
Phenanthrene	2.27E-05	0.001	2.27E-08
Pyrene	8.66E-05	0.001	8.66E-08
SUM Annual Toxicity Equivalent (mg TEQ/m³)			4.40E-05
Carcinogenic Exposure Value for B(a)P (mg/m³) 1 in 100,000 cancer risk			0.32
Carried Forward in Risk Assessment			NO

Further Risk Evaluation of Selected SoPCs

Additional information on the individual compounds and their toxicity is presented in this section. Of the numerous compounds assessed for their potential to be harmful to the health of local residents only four were carried forward for detailed assessment:

- HCl
- NO_x
- SO₂
- H₂S

HCl Risk Characterization

Hydrogen chloride exceed the OMOE generic guidelines for 24 hour exposure and using scaling factor at the 1 hour exposure level for all three scenarios. Therefore, further examination of the toxicity and potential health impacts of exposure to HCl is presented.

Since the annual average concentrations of HCl (see Table 6-1) and the hourly concentrations are below the US EPA chronic RfC and CalEPA REL, no chronic or acute health effects to the local population are expected. In addition, Table 6-1 also shows the 99th and 95th percentile concentration of HCl at each of the receptors. All HCl concentrations did not exceed the toxicological standards derived by either US EPA or CalEPA. Therefore, it is very unlikely that HCl is posing a potential health threat to local residents.

NO_x Risk Characterization

The only receptor where NO_x exceed the Health Based Guidelines was at the fenceline. Concentrations of NO_x were below the Health Based Guidelines at all special receptors and the maximum gridded receptor. The NO_x Health Based Guidelines are actually based on exposure to NO₂ and not NO.

Generally for combustion, NO₂ constitutes 5 to 10% of the initial total emissions of NO_x with the remaining 90-95% being NO. The conversion of the majority of NO occurs after emission to the atmosphere. The rate of conversion depends on the oxidizing potential of the atmosphere at the time of release. For example, if the ambient concentration of O₃ is high at the time of release, the conversion might be expected to be higher than if the ambient concentration of O₃ was low. Three different conversion methods exist, however, Tier 3 Ozone conversion was considered most representative of the Crofton area.

The maximum predicted hourly and daily average NO₂ concentrations using the Tier 1, 2 and 3 methodologies are presented in the Table below. As can be seen, using the more advanced Tier 3 analysis for the conversion of NO to NO₂ produces much lower maximum hourly average ground-level NO₂ predictions than the conservative Tier 1 assumption.

The WHO Health Based guidelines for NO₂ are 200 µg/m³ for 1 hour exposure based on human clinical data (WHO, 2000) and a annual concentration of 40 µg/m³ based on the need to protect the public from exposure to nitrogen dioxide (WHO, 2000).

NO-NO ₂ Conversion Method	Averaging Period	Toxicity Reference Values from WHO (mg/m ³)	Maximum Fenceline (mg/m ³)	99 th Percentile (mg/m ³)	95 th Percentile (mg/m ³)
Tier 1 – 100% Conversion	Hourly	200	1494.60	193.27	117.0
	24 Hour		73.90	71.3	36.9
Tier 2 – 75% Conversion	Hourly	200	1121	145	88.0
	24 Hour		55.4	53.5	29.4
Tier 3 – Ozone Limiting Method	Hourly	200	245.0	115.0	107.0
	24 Hour		73.2	71.3	36.9

Given that Tier 3 conditions are the most representative of the local Crofton area and if the NO₂ Health Based Guidelines are applied to the Tier 3 conversion then there is no acute health effect expected from exposure to NO₂ (hourly 500 µg/m³ and 24 hr 200 µg/m³) at the fenceline. This is especially true of the 99th and 95th percentile concentrations.

SO₂ Risk Characterization

The SO₂ concentrations at the fenceline, as well as at the maximum off-site gridded receptor, exceeded the OMOE AAQC for acute and sub-chronic health effects and vegetative effects. An exceedance of an AAQC does not imply that an effect occurs, rather that it can not be ruled out entirely at this point.

The table below presents concentrations of SO₂ and corresponding WHO toxicity values used for the derivation of their guidelines (WHO, 2000). Although the 99th percentile concentration of SO₂ was greater than the TRV the 95th concentration was below. The annual concentrations of SO₂ were found to be below the annual toxicity reference value for the inhalation of SO₂.

Receptor	SO ₂ (mg/m ³)		
	Hourly	24 Hour	Annual
WHO toxicity based Guideline (mg/m³)		125	50
Maximum Fenceline	1250	141	18.7
Maximum Gridded Receptor	1460	204	16.3
99 th Percentile at Max Gridded Receptor	543	159	NA
95 th Percentile at Max Gridded Receptor	345	109	NA

Given that there would be very limited time spent by any one person at the fence line, or gridded receptor located in close proximity to the fenceline, and given the conservative nature of the air model it is likely that SO₂ concentrations in the area do not pose a threat to human health on either an acute or chronic basis. However, this cannot be entirely ruled out at this point.

Hydrogen Sulphide Risk Characterization

Hydrogen sulphide is a common issue surrounding the operation and maintenance of pulp and paper mills in Canada. H₂S is the primary component of total reduced sulphur, which is routinely monitored by NorskeCanada as part of their ongoing ambient air quality monitoring program.

The air modeling exercise indicated that the modeled H₂S concentrations exceeded health based toxicity reference values at the fenceline approximately 60% of the locations modeled for hourly and 24 hour timeframes. In the case of annual modeled concentrations of H₂S, over 80% of the fenceline locations exceeded the US EPA RfC of 2.0 µg/m³. The modeled ambient concentration of H₂S at the Crofton Substation and the nearby Crofton Mobile Home Park also exceed the TRVs.

Given that there was only 0.13% exceedance (Table 5-1) of the H₂S 24 hour TRV (1 day in 2 years) or 2 hours in 2 years of the hourly TRV, measured in ambient air monitoring at the Crofton Substation conservatively as TRS, Jacques Whitford believes that the air modelling exercise is likely overestimating the H₂S concentrations in the near field area of the Crofton mill.

It should also be noted that the 99th percentile of modeled H₂S at the Crofton Mobile Home Park was less than either the hourly or 24 hour TRV, as was the hourly H₂S concentration at the Crofton Substation.

Therefore, it is unlikely that there is an actual acute health risk to local residents from exposure to H₂S living nearby the mill, however it can not be ruled out at this point.

Uncertainties

A qualitative analysis of uncertainties associated with the risk assessment process supports the conclusion that the risk estimates provided in this report are very conservative and likely overstate the potential risks to the local community.

Summary of Results

- Concentrations of PM₁₀ and TRS routinely monitored at the three monitoring stations located on a transect from north to south of Crofton Division indicated that no chronic or acute adverse health effects are expected as a result of exposure to measured concentrations.
- Of the numerous substances modeled the majority had concentrations that were either below OMOE health based guidelines or toxicity reference values applicable to individual substances.

- HCl, NO_x, and SO₂ had concentrations at some receptors that exceeded health based guidelines. Further examination of each of these compounds revealed that it is unlikely that exposure to any of these substances in the local air shed would result in adverse health effects. However, at this point it can not be entirely ruled out for SO₂.
- Modelled concentrations of H₂S near field to the Crofton Division exceeded health based toxicity reference values. Further examination of the areas of exceedance indicates that the modeled is likely overestimating H₂S concentrations (e.g. Crofton Substation). However, adverse acute and chronic health effects from exposure to H₂S for residence living close to the mill could not be ruled out at this point.
- Results of the risk assessment study were in agreement with a 1990s study conducted by John Hopkins University (Matanoski *et. al.*, 1995) that indicated workers in the pulp and paper industry, who are exposed to higher concentrations of substances than general population, do not have significantly higher rates of mortality from all causes or from any specific cause of death compared to the general United States population.

Recommendations

In light of the findings of the air quality modelling and human health risk assessment, it is recommended that:

- To validate the findings respecting concentrations of H₂S and SO₂ at locations on and just outside the Crofton fenceline, ambient air quality monitoring for these parameters should be conducted for a minimum of one year at suitable locations near the fenceline.

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- If this monitoring determines that modeled concentrations in the near-field are not conservative then a similar monitoring program for HCl should be conducted at suitable locations.
 - Through initial comparison of air modelling results for H₂S and ambient measured concentrations of TRS, Jacques Whitford believes that the current position of the ambient air monitoring stations are in suitable locations. However, given the extensive air modelling exercise undertaken the ambient air quality and meteorological monitoring program associated with the Crofton mill should be reviewed with respect to the substances measured, frequency and method collection.

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GLOSSARY

AAQC: Ambient air quality criteria

ADAMS: Air data acquisition and management system

AQO: air quality objective

ATSDR: Agency for toxic substances and disease registry

Acceptable risk: A risk level that is considered by society or regulatory agencies as tolerable.

Background level: The normal ambient environmental concentration levels of a substance.

B.C.: British Columbia

BTEX: benzene, toluene, ethylbenzene, xylenes - substances typically found in petroleum products such as gasoline, heating oil, automotive oil, etc.

CACs: Criteria Air Contaminants

CalEPA: California Environmental Protection Agency

CALMET: Part of CALPUFF model for processing

CALPUFF: An air dispersion model

CARB/EPA: California Air Resources Board/Environmental Protection Agency.

Cancer: a disease characterized by malignant, uncontrolled invasive growth of body tissue cells.

Carcinogen: A chemical or substance capable or suspected to be capable of producing cancer in living organisms.

CCME: Canadian Council of Ministers of the Environment. CCME publishes Canadian Environmental Quality Guidelines for soil and other environmental media. CCME also publishes a process for assessing and clean-up of contaminated sites.

CDED: Canadian digital elevation data.

Chronic exposure: The long-term, low-level exposure to substances, i.e., the repeated exposure or doses to a substance over a long period of time. It may cause latent damage that does not appear until a later period in time.

Chronic toxicity: The occurrence of symptoms, diseases, or other adverse health effects that develop and persist over time, after exposure to a substance delivered over a relatively long period of time.

Concentration: A quantitative measure of the amount of a substance present in a sample. Typically defined in milligrams per kilogram (mg/kg) for soil samples or milligrams per litre (mg/L) for water

samples. mg/kg and mg/L are also equivalently expressed as parts per million (ppm)

Conservatism: The tendency towards caution or protection. Conservative assumptions made in a risk assessment are designed so as to over-predict the actual risks.

Dermal exposure: Exposure to a substance through skin absorption.

Dose: That amount of a substance taken in by a receptor on exposure; it is a measure of the amount of the substance received by the receptor, as a result of exposure expressed as an amount of exposure (in mg) per unit body weight of the receptor (in kg).

Dose-response: The quantitative relationship between the dose of a substance and observed health effect caused by exposure to such substance.

Dose-response evaluation: The process of quantitatively evaluating toxicity information and characterizing the relationship between the dose of a substance and the expected incidence of adverse health effects in the exposed population.

DTED: Digital terrain elevation data.

EC: Environment Canada

Effect: The response observed in the body due to exposure to a substance (e.g., decreased body weight).

Endpoint: The specific effect (e.g., liver damage) upon which a toxicity value is determined.

EPC: Exposure point concentration

Exposure: Receiving a dose of a substance; or coming in contact with a hazard.

Exposure assessment: The exposure assessment includes the identification of the receptors of interest, the identification of the relevant exposure pathways, and the quantification of the exposures from each pathway.

Exposure pathway: The mechanism by which a receptor can be exposed to a chemical hazard, such as ingestion of contaminated soil or inhalation of contaminated air.

Exposure Point Concentration (EPC): The calculated concentration of a substance that is representative of the concentration at the point of exposure (i.e., the concentration of a substance in soil, water or air that is carried forward in the risk assessment calculations for a particular exposure pathway).

Exposure Scenario: Combination of a hazard, pathway and a receptor.

FPAC: Forest Products Associations of Canada

Guidelines: Guidelines for environmental quality are defined by regulators for many substances to quickly and easily identify the concentration of a substance where no further investigation or study is required. If the concentration of a sample exceeds an established guideline, then further investigation is carried out to determine if action might be required.

H₂S: hydrogen sulphide

HHRA: Human Health Risk Assessment

Hazard: The inherent toxic potency of a substance independent of level of exposure.

Hazard identification: This is the first step in a risk assessment and is used to identify environmental hazards (e.g., SoPC) that may pose a health risk. The chemical hazards at a site are identified based on the results of data reviewed and field investigations, as well as an understanding of the toxicology of the substances of concern.

Hazard index (HI): Sum of the SoPC – specific hazard quotients for an exposure scenario.

Hazard quotient (HQ): The ratio between the calculated potential dose of a substance and the toxicity value for that substance. Values below 1.0 suggest the potential dose is below the toxicity value and no adverse health effects would be expected.

Hog: woodchips

Human health risk: The likelihood (or probability) that a given exposure or series of exposures to a hazardous substance will cause adverse health impacts on individual receptors experiencing the exposures.

Hydrocarbons: Organic substances associated with fossil fuels such as petroleum products.

Ingestion: Exposure to a substance through the mouth and into the gastrointestinal system.

Inhalation: Exposure to a substance through the respiratory tract system.

Intake: The amount of material inhaled, ingested, or dermally absorbed during a specified time period. It is a measure of exposure, expressed in mg SoPC/kg BW - day.

IRIS: Integrated Risk Information System (US EPA)

Jacques Whitford: Jacques Whitford Limited

LOAEL: lowest observed adverse effect level

Modeling: Use of mathematical algorithms to simulate and predict real events and processes.

MWLAP: British Columbia Ministry of Water, Land and Air Protection.

MRL: Minimal Risk Level

NAAQOs: National ambient air quality objectives

NBSK: northern bleached softwood kraft pulp

NCASI: National Council for Air and Stream Improvement

NOAEL: no observed adverse effect level

NPRI: National Pollutant Release Inventory

OMOE: Ontario Ministry of the Environment

PM₁₀ and PM_{2.5}: airborne particulate matter less than 10 or 2.5 µm in size

Petroleum Hydrocarbons: A class of organic substances associated with petroleum products such as gasoline, lubricating oil and home heating oil.

Polycyclic Aromatic Hydrocarbons (PAHs): A class of organic substances often associated with the burning of wood, coal, heating oil, and many industrial processes.

Potency: A measure of the relative toxicity of a substance.

ppb (parts per billion): An amount of substance in a billion parts of another material; also expressed by µg/kg or µg/L.

ppm (parts per million): An amount of substance in a million parts of another material; also expressed by mg/kg or mg/L.

Receptor: Refers to members of a potentially exposed population, e.g., persons or organisms that are potentially exposed to a particular substance. Receptors do not represent real people, but rather are used hypothetically, to represent an individual who might be expected to have a maximum potential exposure.

Receptor identification: Identification of the receptors that may be exposed to the SoPC.

Reference concentration (RFC): The maximum theoretical chronic dose of substance that the human body can absorb without experiencing chronic health effect; it is expressed in µg of substance per m³ of air. It is the estimate of lifetime daily exposure of a non-carcinogenic substance for the general human population which appears to be without an appreciable risk of deleterious effect; used interchangeably with Tolerable Daily Intake (TDI).

Risk assessment: The determination of the potential adverse health effects due to exposure to substances that may cause harm.

Risk management: The steps and processes taken to reduce, abate, or eliminate any unacceptable risks that have been identified by a risk assessment.

Risk: The probability or likelihood of an adverse consequence from a hazardous situation or hazard, or the potential for the realization of undesirable adverse consequences from impending events.

Risk characterization: The assessment of the predicted health risk from exposure to each substance by each receptor. The quantification of health risks is calculated for the identified pathways using generally accepted methods and appropriate assumptions about exposure. The risk characterization can determine if adverse health effects are expected from exposure to the substance at the exposure point.

SO₂: sulphur dioxide

Slope factor (SF): A plausible upper-bound probability estimate of a response per unit intake of a substance over a lifetime. It is used to estimate an upper bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen.

SoPC - Substance of Potential Concern: A substance that is carried forward into a risk assessment.

TC₀₅: Tumorigenic Concentration resulting in incidence or mortality cancer rate of 5 %

TEF: toxic equivalency factor

TEQ: toxic equivalents

Threshold: The lowest dose or exposure of a substance at which a specified measurable effect is observed and below which such effect is not observed.

Tolerable Daily Intake (TDI): The maximum amount of a substance that the human body can absorb without experiencing chronic health effects; it is expressed in mg of substance per kg body weight per day. It is the estimate of lifetime daily exposure of a non-carcinogenic substance for the general human population that appears to be without an appreciable risk of deleterious effects; used interchangeably with Reference Concentration (RfC).

Toxicity: The harmful effects produced by a substance. It is the quality or degree of being poisonous or harmful to human or ecological receptors.

Toxicity assessment: Toxicity reference values are obtained for the SoPC

TRS: total reduced sulphur

TRV: toxicity reference value

US EPA: United States Environmental Protection Agency

USGPM: United States gallon per minute

USGS: United States Geological Survey

Uncertainty: The degree of confidence in the estimate of a variable's magnitude or probability of occurrence.

Uncertainty assessment: A qualitative or quantitative assessment of the uncertainty associated with the risk estimation. Uncertainty may be associated with a number of components of the HHRA, including the exposure estimate, the toxicity reference value, and the assumed bioavailability of the SoPC from the exposure matrix.

Uncertainty factor (UF): Refers to a factor that is used to provide a margin of error when extrapolating from experimental animals to estimate human health risks.

UR: Unit risk

WHO: World Health Organization

1.0 INTRODUCTION

Jacques Whitford Limited (Jacques Whitford) was retained by NorskeCanada Crofton Division to conduct a Human Health Risk Assessment (HHRA) relating to its aerial emissions from its Crofton Division operations in Crofton, British Columbia.

The NorskeCanada Crofton Division is an integrated pulp and paper facility that produces newsprint, directory paper and market pulp. The mill is located on the southeast coast of Vancouver Island on Stuart Channel across from Saltspring Island. The mill is situated less than a kilometer north of the community of Crofton (population 2,500) and 5 km north of the City of Duncan (population 4,700). The location of the mill is presented in Figure 1-1 and a site plan of the mill is presented in Figure 1-2.

In August 2003, NorskeCanada applied to the British Columbia (B.C.) Ministry of Water, Land and Air Protection (MWLAP) to conduct a thirty month trial burn of three alternative fuels in its #4 wood waste power boiler (Norske, 2003). The three proposed alternative fuels are bituminous coal, tire derived fuel, and hogged waste railway ties. However, in January 2004, NorskeCanada applied to the government for a six month extension for public consultation on its proposal (NorskeCanada, 2004).

In March 2004, NorskeCanada initiated the “Crofton Mill Community Engagement Process”. This process has two primary aims:

Strengthen NorskeCanada’s understanding of community concerns and issues related to the operations of the Crofton Mill and its impacts on the communities of the Cowichan Valley

Gather the views of a broad range of community groups and individuals representing the diverse interests of the communities of the Cowichan Valley

In April, twisurveys Inc. conducted numerous focus group sessions with local stakeholders to gather information and concerns of residents with respect to operations at the Crofton Mill. One of the Key Findings of this process was a community interest in having a baseline air quality study and accompanying human health risk assessment conducted on current day aerial emissions from the Crofton Mill.

1.1 SCOPE AND OBJECTIVES

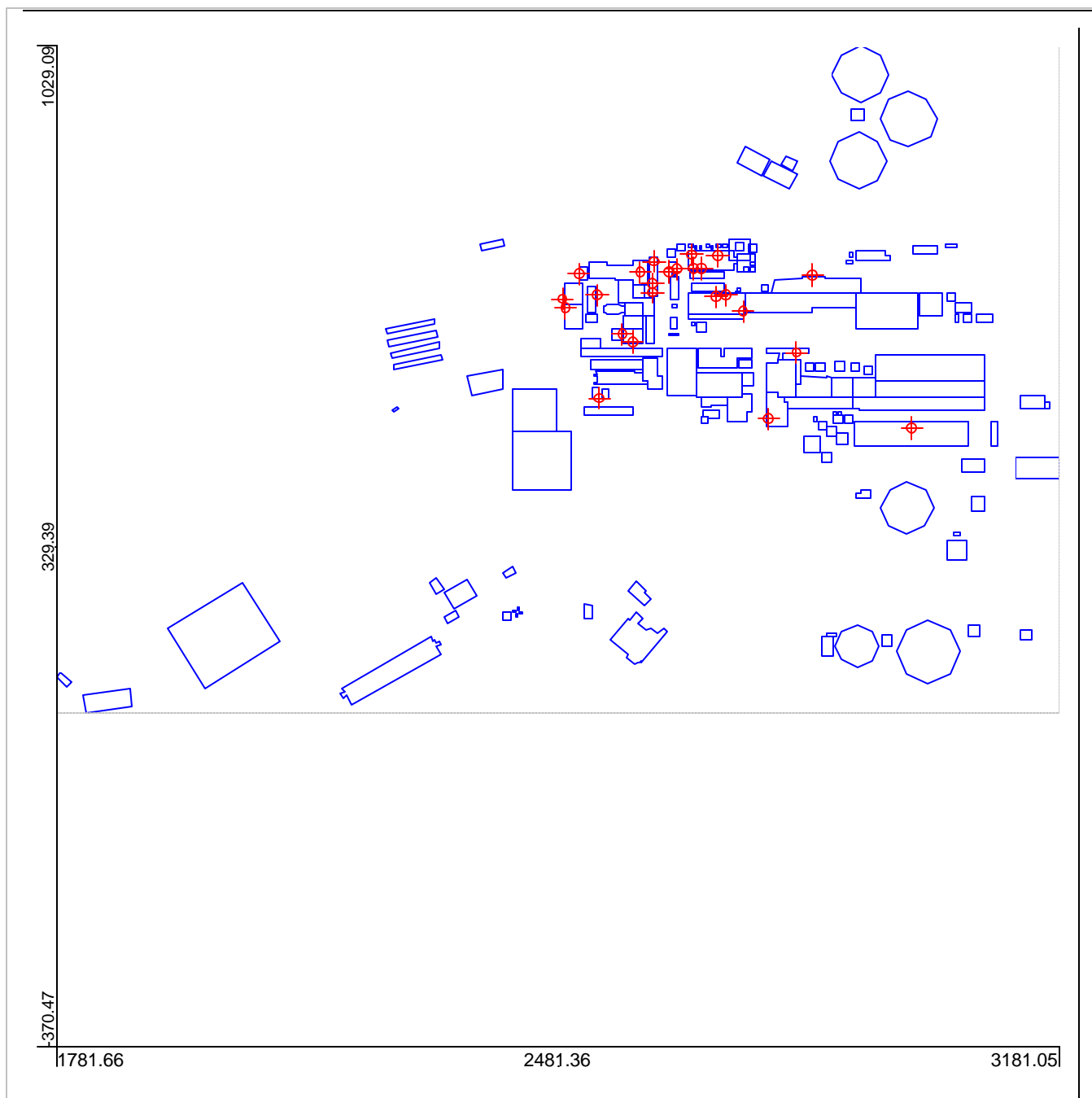
The human health risk assessment provided in this report is meant to inform on the potential human inhalation health impacts from current day mill operations on health of the local community. Although under no regulatory obligation to do so, NorskeCanada is interested in ensuring that their operations are not negatively impacting on the health of local residents. This study considers only current day emissions and mill operations and is not intended to address issues surrounding alternative fuel trials at the mill.

The scope of work consists of the following three tasks:

1. *Review of ambient air quality monitoring results from the three existing monitoring stations in the Crofton Air shed to detect temporal trends and evaluate potential risk to human health from measured airborne levels of substances of potential concern (SoPC) (Vol. II).*

Figure 1-1 Location of the Crofton Division





Legend

⊕ Stack Location

Figure 1-2 Site Plan of Crofton Division

2. *Modelling of ground-level concentrations of SoPC in ambient air from current day emissions from the Crofton Mill (Vol. I).*
3. *Determining whether or not modelled concentrations of SoPCs in ambient air pose a potential risk to the health of the residents of the Crofton Air shed (Vol. II).*

1.2 RISK ASSESSMENT APPROACH

Human Health Risk Assessment (HHRA) is an evaluation process used to describe the nature and magnitude of the risk associated with the exposure of human receptors to a potential hazard. A HHRA usually combines information on potential receptors with exposure data and identified hazards (i.e. toxicity) to determine the relative level of risk resulting from an operation.

The elements of a Human Health Risk Assessment are presented schematically in Figure 1-3.



Figure 1-3 Risk Triad

In order for a risk to exist, all three critical elements of receptors, hazard, and exposure must be present. That is, a substance must be released, a pathway must exist for the substance to travel, a pathway must exist for the substance to be absorbed into the human body, a dose must be received, and the substance must have some adverse toxicological effect (i.e. temporary or permanent damage). If any

one of these components is missing then there is no level of risk.

In other words, a particular substance does not necessarily represent a level of risk simply because it exhibits some toxic characteristic. Similarly, the mere presence of a substance in the environment does not necessarily imply a risk.

Figure 1-4 briefly describes the approach used to conduct the Human Health Risk Assessment for Crofton Division.

The following is a breakdown of the components of the HHRA:

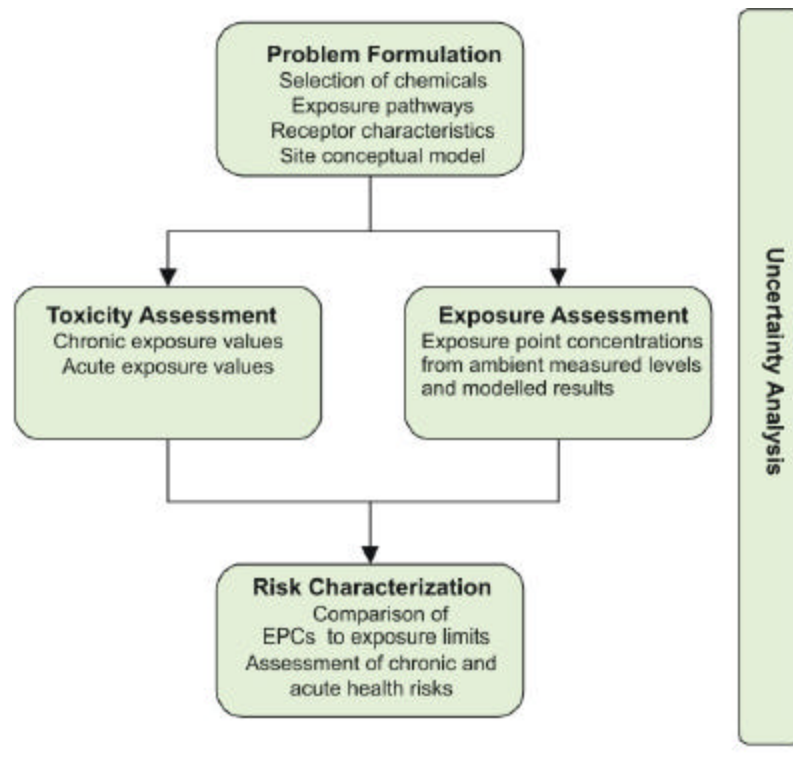


Figure 1-4 Risk Assessment Approach

- The substances of potential concern (SoPCs) will be identified, based on knowledge of the Crofton Division emissions as well as operating experience at similar facilities in Canada. Potential receptors and pathways leading to such receptors will also be identified.
- The environmental releases of the SoPCs (emissions) from the operation of the facility will then be estimated based on process emissions during normal operations (Vol. I).
- Environmental release data will then be combined with accepted dispersion models to predict ambient air concentrations (wet and dry) resulting from the operation of the facility (Vol. I).
- All identified environmental release data and predicted human exposure (i.e. ground-level air concentration) data, as applicable, will be combined to conduct the risk characterization part of the study, which will build on an exposure assessment of potential receptors combined with a toxicity assessment of each of the selected substances of potential concern.
- The risk assessment will use scientifically accepted risk characterization and risk assessment techniques, including guidance documents from the British Columbia MWLAP, the Canadian Council of Ministers of the Environment (CCME), Health Canada, United States Environmental Protection Agency (US EPA), to predict the anticipated level of risk to human health resulting from the operation of Crofton Division.
- Finally, a discussion of the uncertainties in the study resulting from a variety of factors will be presented.

1.3 AIR MODELLING APPROACH

The air quality study was conducted to determine the ambient air concentrations of mill emissions at NorskeCanada's Crofton Division, British Columbia (B.C.) mill using 2003 emissions data. Since the mill is located in an area of complex terrain and ocean/land atmospheric interactions, the CALMET / CALPUFF dispersion model was selected for the analysis. CALPUFF is an integrated puff model capable of modelling instantaneous or continuous releases over distances ranging from tens of metres to hundreds of kilometres. The model contains a meteorological pre-processor (CALMET) which produces 3-dimensional flow fields of wind speed, wind direction, temperature, mixing layer height, atmospheric turbulence, etc. The meteorological pre-processor accounts for complex terrain effects on the wind field.

The primary purpose of the dispersion modelling study was to produce conservative predictions of the maximum and average ground level concentrations of approximately 80-substances emitted from the mill, as reported to the Environment Canada (EC) National Pollutant Release Inventory (NPRI).

Meteorology was obtained from the Meteorological Service of Canada for Nanaimo, Victoria International Airport, Entrance Island, and Comox. Data were also obtained from the British Columbia Ministry of Water, Air and Land Protection (WLAP) from the Duncan and Harmac meteorological stations. The meteorological data were processed using the California Air Resources Board/Environmental Protection Agency (CARB/EPA) meteorological pre-processor CALMET. The CARB/EPA dispersion model CALPUFF was then used to calculate substance ground level concentrations over a 45-km by 55-km domain in the vicinity of the facility.

The air modeling results are discussed throughout this report. However, specific details of the air modeling study are found in Vol. I of this report:

Baseline Air Quality Modelling And Human Health Risk Assessment Of Current Day Emission From NorskeCanada Crofton Division, Crofton, British Columbia, Volume I: Air Quality Modelling (Vol. I).

1.4 REPORT ORGANIZATION

The report is presented in nine sections. Section 1 provides an introduction to the project and the scope and approach to the risk assessment. Section 2 describes the background to the study area. Section 3 outlines the problem formulation for the risk assessment. Section 4 presents the toxicity assessment and Section 5 discusses the exposure and risk assessment. Section 6 details the detailed risk characterization of several substances of potential concern. A summary of uncertainties in the risk assessment process is provided in Section 7 and the report findings are summarized in Section 8. The references for the report are found in Section 9.0. Supporting information is provided in several appendices at the end of the report.

2.0 STUDY BACKGROUND

This section presents background information regarding the project, including a brief description of the Crofton Division mill, a description of the site, and the regional setting.

2.1 FACILITY OVERVIEW

The mill complex is located on flat coastal lowland immediately adjacent to Stuart Channel, as shown in Figure 2-1. Local relief on the order of 400 metres (1,300 ft) is experienced within 4 to 6 km of the site. The nearest permanently occupied dwellings are located within 100 metres of the mill property line (Crofton Mobile Home Park), however the bulk of Crofton's population reside within 1 to 3 km of the centre of the mill. Saltspring Island lies some 5 km to the east, across Stuart Channel.

Crofton Division began operation in 1957 as a single-line kraft pulp mill. Today, three paper machines and two pulp machines have an annual capacity of 680,000 tonnes of product. The mill produces 430,000 tonnes of groundwood paper annually, 280,000 tonnes of which is newsprint and 150,000 tonnes of which is directory paper. The remainder of Crofton's capacity is market pulp, 40% of which is used in their papermaking processes.

Crofton's paper is manufactured from combinations of mechanical pulps, kraft pulp, fillers, pigments and recycled newspaper and magazines. Thermomechanical pulp is made by heating wood chips under pressure to soften them, and then by grinding the softened chips between serrated metal plates to separate the fibers from the wood structure.

Kraft pulp is made by cooking wood chips in a solution of caustic soda and sodium sulphide to separate the wood fibers from the lignin, a glue-like binding substance. Crofton's two chemical pulping lines produce northern bleached softwood kraft pulp (NBSK). Crofton's pulp and paper products are made almost entirely from residual wood fibre, waste wood from B.C. sawmills that was once consigned to landfills or burned.



Figure 2-1 Crofton Division View from the West

2.2 REGIONAL SETTING

The Crofton Division Mill is located adjacent to the town of Crofton, in the Cowichan Valley Regional District, on Vancouver Island, British Columbia (Figure 1-1). While the population of the Cowichan Valley is approximately 79,000, the town of Crofton has a population of 2,500.

The Cowichan Valley is located between the Cities of Victoria and Nanaimo, and is surrounded by scenic mountains and valleys. The area has been home to numerous First Nations for countless centuries and was settled by European farmers and loggers in the mid 1800s.

2.2.1.1 Industrial Development

The Cowichan Valley is home to numerous business sectors, including:

- Forestry
- Agriculture
- Marine
- Manufacturing
- Tourism

However, Crofton Division continues to be by far the largest employer (approximately 1,000 people) and contributor to the local municipal tax base.

2.2.2 Residential Areas

The entire area surrounding Crofton Division is populated with residential dwellings, from the Crofton Mobile Home Park located within metres of the fenceline to the town of Crofton located approximately 1 km from the mill site.

Residential areas can be seen in Figure 1-1.

2.2.3 Recreational Areas

The Cowichan Valley relies on tourism and recreation as an integral part of its economy, providing approximately 20% of the region's employment. There are many outdoors activities that can be conducted in the area both on land and on water including agri-tourism, marine, eco-tourism and adventure tourism.

2.2.4 First Nations

The traditional use of land by First Nation communities in the vicinity of Crofton Division is well documented. In fact the name "Cowichan" was derived from the Coast Salish word Khowutzun, literally translates into "The Warm Land". A number of First Nation Reserves exist within the Cowichan Valley Regional District, including: Cowichan, Cowichan, Chemanus, Halalt, Squaw-hay-one, Tsussie, Claoose, Kil-pah-las, Kuper Island, Lyacksun, Shingle Point, Malachan, Malahat, Cowichan Lake, Portier Pass, Theik, Wyah , and Oyster Bay.

Given the relative proximity of these communities to the Crofton Division, it is reasonable to assume that the forested areas and waters, have historically and are currently been used by the Aboriginal Community for their hunting, fishing, and gathering activities.

By virtue of their Aboriginal Rights, First Nation communities have a direct stake in the use of the forests and natural resources for their traditional native fishing, hunting, and gathering activities. Therefore, formal discussions and information exchanges were undertaken by NorskeCanada with several First Nation communities and organizations as part of the consultation phase of the project

These meetings were aimed at sharing information about the Crofton Division operations with First Nations.

2.2.5 Climate

The Cowichan Valley is reported to have the highest mean annual temperature of any region in Canada. Climate data were obtained from a representative Environment Canada weather station at Duncan, B.C. The average daily temperature is a temperate 9°C, with a daily minimum temperature rarely falling below 0°C and a maximum daily temperature typically in the mid 20°C range.

The average yearly rainfall is 993 mm and snow fall of 43 cm. Given the temperate climate the residents of this area likely spend more time outdoors than many other places in Canada and likely have their windows open for more days a year as well.

3.0 PROBLEM FORUMULATION

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

- Potential emissions from the Crofton Division;
- Selection of the substances of potential concern (SoPC);
- Assessment and screening of potential exposure pathways;
- Characterization of potential receptors who may be exposed to facility emissions; and,
- Development of site conceptual model that describes the potential interactions between the facility operation and the surrounding community.

3.1 FACILITY EMISSIONS

The NorskeCanada Crofton Mill emissions were developed from the mill's submission to Environment Canada (EC) for the National Pollutant Release Inventory (NPRI) for the 2003 reporting year (the most recent year available). The NPRI report was developed by NorskeCanada following the reporting requirements outlined in the EC Supplementary Guide for Reporting to the National Pollutant Release Inventory 2003.

The NorskeCanada emissions estimates for their 2003 NPRI were developed from mill production data for 2003, and emissions factors for various processes developed by the National Council for Air and Stream Improvement (NCASI) or from site specific testing. The use of the NCASI emission factors for estimation is standard practice for the industry. The NCASI emission factors were supplemented (where available), with site-specific emission factors based on Crofton mill stack testing data (2002) collected as part of a study by the Forest Products Association of Canada (FPAC). These site-specific emissions factors were used in place of the NCASI factors by NorskeCanada wherever possible. For further details on emission factors and source tested data see Vol. I.

It should be noted that NorskeCanada included 106 substances listed in their NPRI calculations to determine the substances whose 2003 release rates exceeded the NPRI reporting thresholds. Only substances exceeding their NPRI reporting threshold (the thresholds are substance specific) are required to be reported to Environment Canada. Of the 106 substances, 36 were estimated to have zero emissions. The remaining 70 substances with non-zero emission rates were included in the dispersion modelling.

Table 3-1 lists the SoPCs carried forward in the risk assessment, and those that were estimated to have zero emissions from the mill, thus were not carried forward in the assessment.

Table 3-1 List of Substances of Potential Concern Modelled as Being Emitted from the Crofton Division.

<u>Compounds with Zero Emissions Reported</u>		
1,3-butadiene, 2-Butoxyethanol, n-Butyl Alcohol, 3 - Carene (for total VOC), Chloroacetic Acid, Chlorobenzene, p-cymene (for total VOC), Cyanides, 1,2-dichloroethane, 1,2dichloroethylene, 1,2-Dimethoxyethane (for total VOC), 2,4-Dichlorophenol, Direct Blue 218, Epichlorohydrin, Ethylbenzene, 2-Ethoxyethanol, Ethylene Glycol, Hexane, Hydrazine Compounds, Limonene, 2-Methoxyethanol, Nitrate Ion, Nitric Acid, Nitrilotriacetic Acid, Nonylphenol, Nonylphenol, Polyethylene, Phosphorus, a-Pinene, b-Pinene, Benzo(j)fluoranthene, Dibenzo(a,j)acridine, Dibenzo(a,i)pyrene, 7H-Dibenzo(c,g)carbazole, Perylene		
<u>Modelled SoPC Compounds</u>		
<u>Inorganic Elements</u>	<u>Combustion Gases</u>	<u>VOCs/SVOCs/Miscellaneous</u>
Antimony Arsenic Cadmium Chromium Chromium VI Cobalt Copper Lead Manganese Mercury Nickel Selenium Silver Zinc	Ammonia Carbon disulphide Carbon monoxide Chlorine Chlorine dioxide Hydrochloric acid Hydrogen fluoride Hydrogen sulphide Nitrogen oxides Total particulate matter (TPM) Particulate (PM _{2.5}) Particulate (PM ₁₀) Sulphur dioxide Sulphuric acid	Acetaldehyde tetrachloroethylene Acetone 1,2,4-trichlorobenzene Acrolein 1,1,2-trichloroethane Biphenyl Trichloroethylene Carbon tetrachloride 1,2,4-Triomethylbenzene Chloroform Chloromethane Cresol Cumene Diochloromethane Formaldehyde Hexachlorobenzene (HCB) Isopropyl Alcohol Methanol Methyl ethyl ketone Methyl isobutyl ketone Naphthalene Phenol Propionaldehyde styrene
	<u>Polycyclic Aromatic Hydrocarbons (PAHs)</u>	
	Benzo(a)pyrene (TEQ) 12 Carcinogenic PAHs	
	<u>Dioxins/Furans</u>	
	2,3,7,8 – TCDD (TEQ)	
<u>Hydrocarbons</u>		
Benzene Toluene Xylenes - total		

3.2 AIR MODELLING METHODOLOGY

Details on the air modeling methodology used in order to establish ground level concentrations of SoPCs surrounding the mill are found in Vol. I.

3.2.1 Receptor Grid Used for Air Modelling Study

Ground-level substance concentrations were calculated over a uniform receptor grid with 250-m spacing as well as at discrete receptors placed along the fence line of the facility and at the locations of the monitoring stations (Vol. I).

In total, 36,464 gridded receptors corresponding to 172 receptors in the x direction and 212 in the y direction were used, as well as discrete receptors along the fence line and at special receptors, which are locations of public facilities such as schools, hospitals and churches. The special receptor locations tabulated in Table 3-2 and found in Figure 3-1.

Special receptor locations were selected in consultation with the air modelling team to ensure that areas where people congregate were not missed by the grid spacing.

Table 3-2 Special Receptor Locations

UTM Easting (km)	UTM Northing (km)	Receptor Name
452.2	5413.3	Crofton Mobile Home Park
452.33	5412.69	Crofton Motel
452.533	5412.5	Crofton School/Comm Centre
452.727	5412.68	Crofton Church 1
452.921	5412.68	Crofton Church 2
449.133	5413.83	Halalt IR2 Comm Centre
447.417	5416.78	Fuller Lake Arena
447.498	5418.86	Chemainus Church
447.199	5418.74	Chemainus School 1
447.547	5419.42	Chemainus Hospital
447.004	5419.53	Chemainus School 2
443.807	5422.74	Saltair School
442.723	5422.84	Saltair Mobile Home
442.569	5423.99	Davis Lagoon Mobile Home
441.023	5425.05	Ladysmith Shopping Centre
439.404	5426.31	Ladysmith School
439.355	5426.92	Ladysmith Hospital
440.213	5426.85	Ladysmith Municipal Hall
438.344	5427.12	Ladysmith Mobile Home
454.815	5407.32	Maple Bay School
451.691	5403.43	Cowichan Reserve Comm Centre
451.254	5404.32	Cowichan Reserve Mobile Home
447.903	5403.97	Duncan Hospital 1
446.972	5404.05	Duncan Hospital 2
448.534	5403.6	Duncan School 1
449.085	5404.03	Duncan School 2
446.09	5405.06	Haywood School
446.689	5404.9	Haywood Church
447.272	5408.29	Somenos City Hall
447.069	5408.19	Somenos School
449.926	5401.44	Eagle Height Mobile Home 1
449.36	5402.37	Eagle Height Mobile Home 2
457.5	5415	Salt Spring Dock

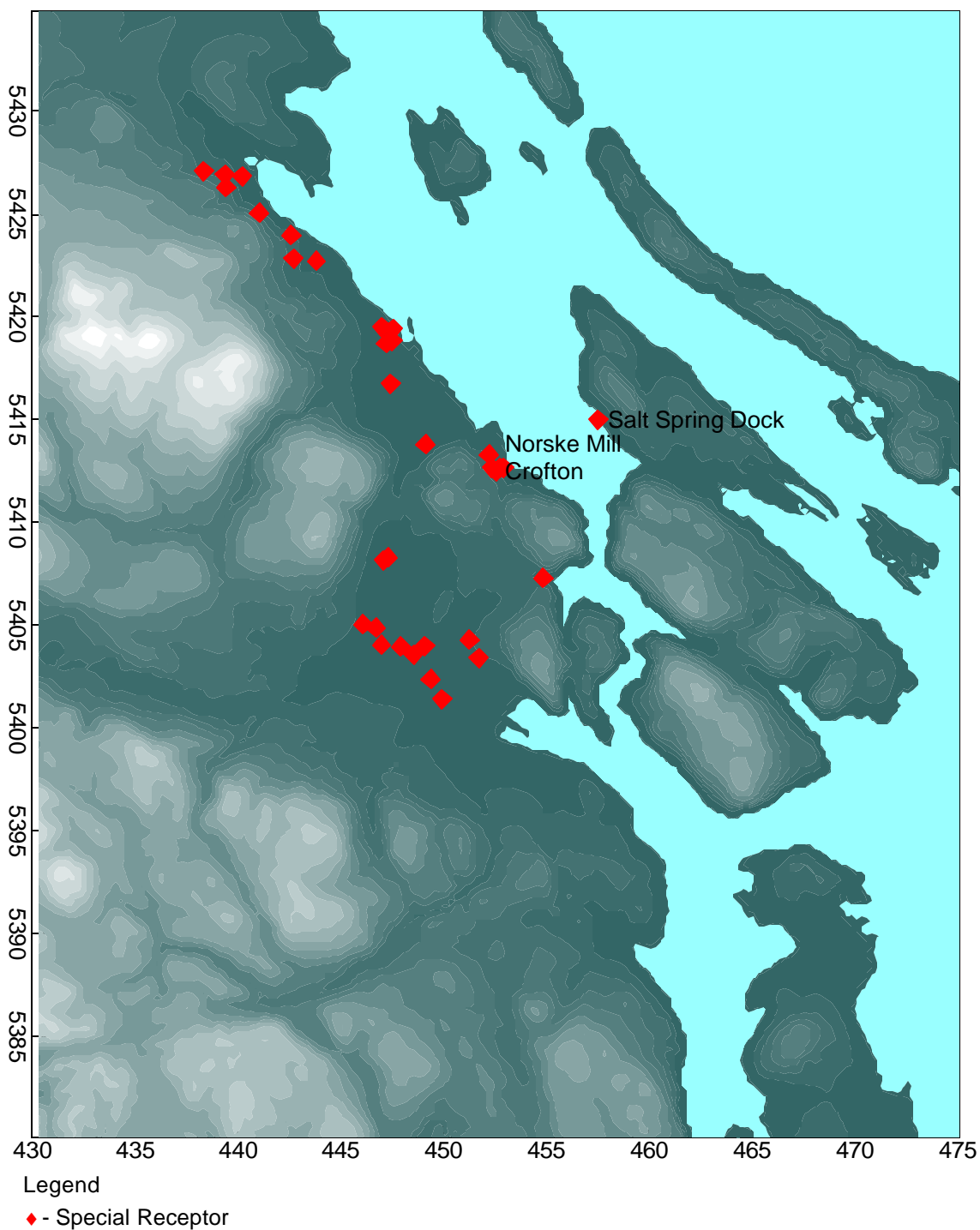


Figure 3-1 Special Receptor Locations

3.3 MODEL VALIDATION USING MONITORING STATION DATA

Ambient air monitoring concentrations for 2000-2001 are compared to CALPUFF predicted levels at the three monitoring stations, as described in Vol I. It should be noted that the dispersion model was run using 2000-2001 meteorological data and 2003 emissions data, therefore some discrepancies between the monitoring data and model predictions is to be expected as the 2000-2001 mill emissions were different than in 2003.

The agreement is quite good at the maximum (and the model generally over-predicts otherwise) for the Crofton Sub-Station monitor. The model is somewhat under-predicting the Crofton South and Duncan measurements, but most predictions fall within the factor of two range; considered as good model performance.

3.4 METHOD OF SELECTION OF SUBSTANCES OF POTENTIAL CONCERN (SoPCs)

The SoPCs considered in this assessment were based on the Crofton Division's 2003 NPRI emissions report. Figure 3-2 presents the screening framework by which the SoPCs were evaluated, screened and risk characterization for this assessment conducted. In order for a substance to be carried forward in the risk assessment it must have been reported in the NPRI emissions report at a concentration greater than zero. NorskeCanada included 106 substances listed in their NPRI calculations to determine the substances whose 2003 release rates exceeded the NPRI reporting thresholds.

Only substances exceeding their NPRI reporting threshold (the thresholds are substance specific) are required to be reported to Environment Canada. Of the 106 substances, 36 were estimated to have zero emissions and thus omitted from the SoPC list. The remaining 70 substances with non-zero emission rates were included in the dispersion modelling.

The next step was to evaluate whether or not the 70 substances had Canadian or World Health Organization (WHO) Health Based Ambient Air Quality Criteria. If a given substance did have an AAQC and its modeled or monitored concentrations were below these criteria, then it was assumed that there would be no increased risk to human health and thus it was screened out as a SoPC. In the event that a substance had a modeled or monitored concentration greater than the AAQC then the substance was retained as a SoPC. If there was no Canadian Health Based AAQC for a given substance than toxicological information was sought on its potential impact on human health, primarily through the inhalation route of exposure. If toxicological data was retrieved then the substance was carried forward as a SoPC. In the event that no toxicological data was found, then a discussion surrounding the substance was presented.

In the event that the maximum modeled concentrations of a SoPC was greater than either a Health Based AAQC or toxicological exposure value, then additional discussion on its potential impact to human health was included in the assessment.

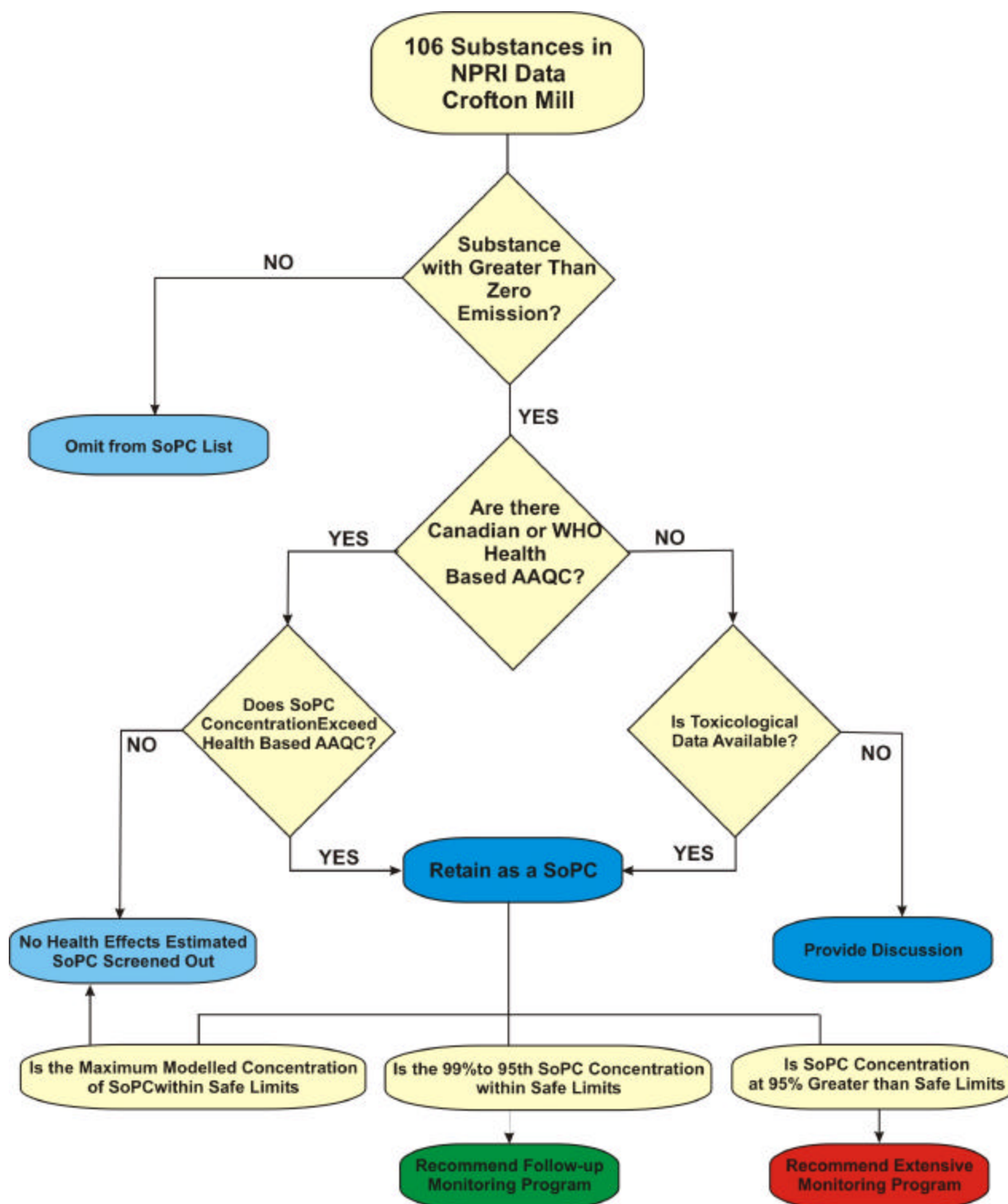


Figure 3-2 SoPC Screening Framework

3.5 EXPOSURE PATHWAYS

The scope of work for this study was limited to the consideration of the inhalation exposure pathway of outdoor and indoor vapours and particulates resulting from stack or source emissions from Crofton Division.



SoPC concentrations in air were either obtained from ambient air monitoring program or from calculations of ambient air concentrations from CALPUFF. Air concentrations used in the evaluation of chronic health risk were estimated using annual average values whereas air concentrations used in the evaluation of acute health risks were calculated using hourly and 24 hour values.

3.6 RECEPTOR CHARACTERISTICS

The purpose of this section is to identify the non-worker related human activities and receptors in the assessment area that may be impacted as a result of exposure to Crofton Division emissions. Receptor characterization is generally focused on identifying current and reasonably foreseeable human activities or land uses that apply to the general public. Potential worker-related exposures are governed by applicable legislation and guidelines, and are outside the scope of work for this project.

Given that only the inhalation exposure pathway was modeled, inhalation unit risks and reference concentrations were employed. Consideration was given to all human life stages, from birth to old age in the selection of toxicity reference values. Note that these are not generally age specific for inhalation exposure pathways. Residents were considered to live in the area 365 days per year over an entire lifetime.

3.7 CONCEPTUAL MODEL

As indicated, residents in the surrounding communities can be directly exposed to vapours and particulates both outside in the ambient air and within their homes (Figure 3-3). All SoPCs were assessed for these routes of exposure.

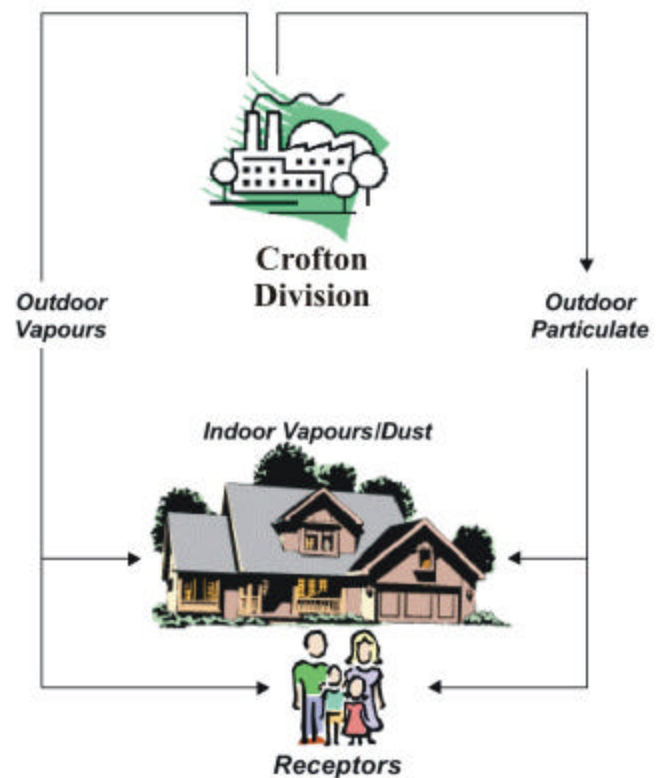


Figure 3-3 Conceptual Model

4.0 TOXICITY ASSESSMENT

Toxic effects from exposure to an environmental substance varies depending on the form of the substance (such as dissolved, or sorbed to soil), the dosage, the route of exposure (such as ingestion, inhalation, or dermal exposure), frequency and duration of exposure, physiological state, sex, and age of the exposed population. Toxicological effects may be brief or prolonged, reversible or irreversible, immediate or delayed. The purpose of a toxicity assessment is to weigh available evidence regarding the potential for the environmental substances to cause adverse effects in exposed populations and to provide an estimate of the relationship between the extent of exposure and the increased likelihood and/or severity of those adverse effects.

4.1 CANADIAN AMBIENT AIR QUALITY OBJECTIVES

4.1.1 B.C. Air Quality Objectives

The B.C. Air Quality Objectives (AQO) are used for guidance in the issuance of Permits under the *Environmental Management Act* (replaced the *Waste Management Act* on July 8, 2004). They are also used in project review referrals under the B.C. Environmental Assessment Act (RWDI, 2002). The B.C. AQOs serve as benchmarks for short-term assessments (less than 24 hours) and for long-term air shed management (annual) (Table 4-1).

The B.C. AQO are presented as three different levels A, B, and C. Level A is the Objective for new and proposed discharges, and when discharges are significantly altered in quality. Level B is intended as acceptable interim Objectives for all other discharges. Level C is the immediate Objective for discharges to meet within a minimum technically feasible period of time.

The B.C. AQO are primarily defined in Pollution Control Objectives publications released by the Pollution Control Board in the mid-to late 1970's following a series of Provincial hearings. Some Objectives were set following the drafting of Criteria in the mid-1980's by the Air Quality Standards Committee of B.C.. Final Criteria documents were not released by this Committee; however, so it is not well understood the final basis upon which they were set.

Generally it is understood that the Objectives were established on the basis of toxicity to flora and fauna, irritant effects, and thresholds for odour. Where two effects occur simultaneously (e.g. toxicity and odour) the lower threshold has been adopted as the Objective. This is the case for H₂S, where the Objectives clearly reflect thresholds for odour. For SO₂, the Objectives reflect concern for injury to sensitive vegetation, with a wide margin of safety for human health.

B.C. only has Objectives for a limited number of substances of potential concern (SoPCs) (B.C. MWLAP, 1995). Generally, the B.C. AQO do not account for chronic, long-term or acute effects of pollutants.

Table 4-1 British Columbia Ambient Air Quality Objectives and Canadian Federal National Ambient Air Quality Objectives (NAAQOs) in ug/m³ (ref: <http://wlapwww.gov.B.C..ca/air/airquality/pdfs>)

Contaminant	Averaging Period	Canada maximum desirable	Canada maximum acceptable	Canada maximum tolerable	B.C. level A	B.C. level B	B.C. level C
		objective	objective	objective	objective/ guideline	objective/ guideline	objective/ guideline
carbon monoxide	1 hour	15000	35000		14300	28000	35000
	8 hour	6000	15000	20000	5500	11000	14300
hydrogen fluoride	24 hour						
hydrogen sulphide	1 hour	1	15		7.5-14	28-45	42-45
	24 hour		5		4	6-7.5	7.5-8
lead	24 hour				4	4	6
	30 day geometric mean						
	quarterly annual geometric mean				2	2	3
nitrogen dioxide	1 hour		400	1000			
	24 hour		200	300			
	annual arithmetic mean	60	100				
ozone	1 hour	100	160	300			
	24 hour	30	50				
	annual arithmetic mean		30				
sulphur dioxide	1 hour	450	900		450	900	900-1300
	3 hour				375	665	
	24 hour	150	300	800	160	260	360
	annual arithmetic mean	30	60		25	50	80
total reduced sulphur	1 hour				7	28	
	24 hour				3	6	
total suspended particulate	24 hour		120	400	150	200	260
	annual geometric mean	60	70		60	70	75
Date of Reference		1989	1989	1989	several	several	several

Ambient Air Quality Objectives Established in 1995

formaldehyde	1 hour	Action Level = 60 Episode Level = 370 50
PM10	24 hour	

B.C. also has ambient AQOs, which were established in 1995 to provide guidance on the desired level of formaldehyde and particulate matter <math><10 \mu\text{m}</math> (PM_{10}). The B.C. Ministry of Water, Land and Air Protection issued Interim Air Quality Objective for fine particulate matter (PM_{10}) in January 1995. The Ministry chose to establish an interim air quality objective for PM_{10} before the national body recommended levels for a national PM_{10} objective to provide guidance for environmental protection decisions. The interim level of $50 \mu\text{g}/\text{m}^3$ for PM_{10} is intended to be equivalent to a maximum acceptable level in the National Ambient Air Quality Objectives (NAAQOs) (24-hour B Level).

4.1.2 National Ambient Air Quality Objectives

Canadian federal National Ambient Air Quality Objectives (NAAQOs) identify benchmark levels of protection for people and the environment for substances in the air. NAAQOs are meant to guide federal/provincial/territorial governments in making risk-management decisions, playing an important role in air quality management, and as benchmarks for developing provincial objectives and standards. NAAQOs are viewed as effects-based long-term air quality goals.

Although NAAQOs are promulgated by the federal government, provincial governments have primary responsibility in many areas of air pollution and the provinces may adopt NAAQOs through processes of their choice and implement them as they see fit. In the case of British Columbia they have adopted some of the NAAQOs, while for some substances the objectives are different.

4.1.3 Canada Wide Standards $\text{PM}_{2.5}$

Particulate matter (PM) is solid particles suspended in the air. These particles originate from a diversity of sources, such as power plants, industrial processes, car exhaust, and are formed in the atmosphere by transformation of gaseous emissions. Their chemical and physical compositions vary dramatically depending of location, time of year, and weather. Particulate matter is categorized by its particle size and can be defined as either coarse or fine particles.

Fine particles have an aerodynamic diameter less than $2.5 \mu\text{m}$ ($\text{PM}_{2.5}$). They differ from PM_{10} in origin and chemistry. These particles are formed from gas and condensation of high-temperature vapors during combustion, and they are composed of various combinations of sulfate compounds, nitrate compounds, carbon compounds, ammonium, hydrogen ion, organic compounds, metals (Pb, Cd, V, Ni, Cu, Zn, Mn, and Fe), and particle bound water. The major sources of $\text{PM}_{2.5}$ are fossil fuel combustion, vegetation burning, and the smelting and processing of metals. Lifetime of $\text{PM}_{2.5}$ can be from days to weeks and travel distance ranges from 100s to 1000s km. The fine particulate matter is also associated with decreased visibility (haze) impairment in many cities of the U.S. and Canada.

In June 2000, the provinces (except Quebec) agreed in principle to guidelines established by the Canadian Wide Standards (CWSs) for particulate matter. The guidelines encompass the fine particle fraction known as $\text{PM}_{2.5}$, having a particle size of less than 2.5 micrometers (μm). The recommended standard for $\text{PM}_{2.5}$ is $30 \mu\text{g}/\text{m}^3$. This is the 24 hour average concentration and is specified in terms of the 3 year average of the annual 98th percentiles of the 24-hour levels which should be accomplished by the year 2010.

Health impacts of particulate matter have been shown to begin at very low concentrations and increase with PM concentration, known as the continuum of effects. Such effects are a reduction of life expectancy by up to a few years, with possibly some contribution from increased infant mortality in the more highly exposed areas as increased chronic bronchitis and chronic obstructive pulmonary disease rates, reduced lung function and perhaps other chronic effects. This has resulted in many organizations and jurisdictions, such as the World Health Organization (WHO) to pursue recommendations that air quality guidelines for PM_{2.5} be further developed.

4.2 HEALTH BASED GUIDELINES

4.2.1 Ontario Ministry of the Environment

The Ontario Ministry of the Environment (OMOE) publishes the most comprehensive set of health based guidelines for ambient air quality in Canada. These guidelines are found in:

Summary of Point of Impingement Standards, Point of Impingement Guidelines and Ambient Air Quality Criteria (AAQCs). (OMOE, 2001)

Although the guidelines contain several health based criteria, they also include odour based guidelines. For the purpose of this study, only those guidelines that were “health based” were used. The AAQCs are not legally enforceable limits, rather they are meant to be the desirable ambient air quality targets for Ontario ambient air. The complete OMOE document can be found in Appendix A.

4.2.2 World Health Organization

The World Health Organization has published a set of Health Based Guidelines for 35 substances, 16 of which are relevant to this study. These health based guidelines were developed to aid European countries

in setting local standards for air quality. These guidelines are found in:

Air Quality Guidelines for Europe Second Edition (WHO, 2000)

The aim of the WHO guidelines is to provide a basis for protecting public health from adverse effects of air pollutants and to eliminate or reduce exposure to these compounds. In some cases the WHO guidelines are more protective, while in others the OMOE guidelines were more stringent. Therefore, modeled concentrations of SoPCs were compared to both sets of criteria.

In the event that there were no health based effect concentrations for the SoPCs or the concentration of a SoPC exceeded health based guidelines then toxicity reference values (TRVs) were sought for individual compounds.

4.3 SOURCES OF TOXICOLOGICAL INFORMATION

The toxicity assessment includes toxicity values established by several regulatory agencies, including:

- Health Canada (HC);
- US EPA: Integrated Risk Information System (IRIS);
- World Health Organization (WHO);
- Agency for Toxic Substances and Disease Registry (ATSDR); and
- California Environmental Protection Agency (CalEPA).

For this assessment, preference was generally been given to toxicity values from Health Canada and IRIS. Where toxicological information was available from both of these sources, the supporting information for each value was reviewed and selection of the final TRV was based on the currency

of the information and the level of protection. Other sources cited above were used when no toxicological information was available from Health Canada or IRIS.

Health Canada is responsible for the assessment of potential risks to human health posed by existing substances under the Canadian Environmental Protection Agency (CEPA, 1999). Health Canada toxicity reference values for substances were obtained from their most recent TRV document (Health Canada, 2003; based on Health Canada, 1996).

The US EPA's IRIS database (US EPA, 2002) is the most comprehensive and widely cited source for risk assessment toxicity values. Toxicity values obtained from WHO and ATSDR are used for a few substances for which HC and IRIS values are not available. In addition, ATSDR provides minimal risk levels (MRLs) for acute, intermediate and chronic toxicity.

Where available, population studies involving human exposure to substances are used for deriving toxicity values. However, in most cases, direct human toxicological data are very limited and toxicity values are based mainly upon animal laboratory studies. Conservative uncertainty factors are used in deriving human toxicity values from animal studies in order to ensure that there is a substantial margin between the toxicity values calculated for laboratory animals and the exposure level where appreciable toxicity might be expected in the human population. In addition, uncertainty factors are also used to account for deficiencies in the level of knowledge of toxicity and variability in sensitive individuals. The toxicity values from each of the agencies are often equivalent for a particular substance, but differences sometimes occur as a result of differing toxicity evaluation protocols.

4.4 NON-CARCINOGENIC EXPOSURE LIMITS

Non-carcinogenic substances are generally considered to act on the body through threshold mechanisms. At low doses, it is assumed that the body is able to deal with the substance without it causing any adverse effect. As the dose or exposure increases, the body's ability to cope with the substance is reduced. The point at which the exposure to a substance exceeds the body's ability to handle the substance without deleterious effects is called the threshold for the particular substance.

4.4.1 Short-Term (Acute) Non Carcinogenic Exposure Limits

Short-term (i.e. acute) exposure values are founded on human health effects-based ambient air quality objectives for 1 and 24 hour averaging times. Acute effects are evaluated by comparing the air concentration to the ambient air quality objective as being treated as a short term reference concentration, or as a minimum risk level (MRL).

For certain air pollutants, ambient air quality objectives are not available for the 1-hour, annual or 24 hour averaging times. In these instances, the air quality health-based limits at these averaging times were calculated from existing ambient air quality objectives using the method used by the OMOE (2001) and US EPA (1992) as follows:

Effects Based Averaging Time	Multiplying Factor (for 1 hour exposure)
24 hour	2.5
Annual	12.5

The OMOE (2001) scaling factors have been validated by the U.S. EPA, and an empirical relationship has been derived. In cases where scaling factors were applied to OMOE criteria they have been denoted in the appropriate tables.

However, no such scaling factor has been adopted by the WHO, thus these factors were not applied to WHO air quality guidelines.

4.4.2 Long-term (Chronic) exposure Limits

Long-term (i.e., chronic) exposure risks were assessed in a manner similar to the short-term risks. Chronic exposure values are founded on human health effects based on annual average air quality objectives. Chronic health effects were then evaluated by comparing the air concentration to the average annual ambient air quality objective. In instances where an annual air quality objective was not available, a reference concentration (RfC) was used; if neither were available, the chronic exposure limit is calculated from a 24-hour or 1-hour ambient air quality objective as per the method used by the OMOE (2001) and US EPA (1992).

Health Canada's Tolerable Concentration (TC) values are chronic measures of toxicity, expressed as μg of substance per cubic metre of air ($\mu\text{g}/\text{m}^3$), and are airborne concentrations to which it is believed that a person can be exposed continuously over a lifetime without deleterious effects. Health Canada's TC values are comparable, and often equivalent to, the US EPA's Reference Concentrations (RfC) values and ATSDR's chronic inhalation MRL values (ATSDR, 2002).

By comparing annual average air concentrations of SoPCs to lifetime TCs and RfCs, risk to human health will be overestimated. This is because these levels are concentrations that can be inhaled every day, 24 hours a day without appreciable risk to human health and are meant to be conservative benchmarks.

Additional details regarding the specific effects of individual pollutants are provided in Appendix B.

4.5 CARCINOGENIC EXPOSURE LIMITS

Carcinogenic effects of substances are generally considered to work through a non-threshold mechanism. Any exposure to a carcinogen is considered to be associated with some level of cancer risk. However, at very low levels of exposure, the probability that an adverse effect (i.e., development of cancer) will occur is often sufficiently small as to be considered negligible. The probability of developing cancer increases with increased exposure to the substance.

The most common toxicity values used by Health Canada for expressing inhalation carcinogenic effects of substances are the Tumorigenic Concentration 05 (TC₀₅), (HC, 1996). Health Canada's TC₀₅ values, expressed as μg of substance per cubic metre of air ($\mu\text{g}/\text{m}^3$) give quantitative estimates of the lifetime exposure levels associated with a 5% increase in the incidence or mortality caused by cancer in the general human population. The tumorigenic dose/concentration is provided for the observable region from applicable studies, and does not provide low-dose extrapolation. The Health Canada TC₀₅ values have been recently re-released in Health Canada, 2003 as unit risks and expressed in $(\mu\text{g}/\text{m}^3)^{-1}$. It is the Health Canada, 2003 values that have been cited in this report.

The US EPA expresses carcinogenic potencies as Unit Risk (UR) values for inhalation exposures. Unit Risk values, expressed as 1 per μg of substance per cubic metre of air $(\mu\text{g}/\text{m}^3)^{-1}$, represent plausible upper-bound estimates of the probability that an adverse effect (i.e., development of cancer) will occur from a lifetime of exposure.

For the purposes of this report, given that only the inhalation pathway was being examined, UR and TC₀₅ concentrations were converted to their exposure values representing the airborne concentration that would result in a one in one hundred thousand cancer risk as shown:

$$\text{Carcinogenic Exposure values (}\mu\text{g/m}^3\text{)} = \frac{0.00001}{\text{Unit Risk (}\mu\text{g/m}^3\text{)}^{-1}}$$

A summary of the TRVs used in this assessment are provided in Table 4.2. Additional details regarding the toxicity of the SoPCs can be found in Appendix B.

Table 4-2 Toxicity Reference Values Used in the Crofton Division Air Quality Study

SUBSTANCE	Chronic				Sub-Chronic (Intermediate)			Acute		
	TRV mg/m ³	Ref.	EFFECT	NOTES	TRV mg/m ³	EFFECT	Ref.	TRV mg/m ³	EFFECT	Ref/
CARCINOGENS										
Benzene	3.03	H	Leukemia	1 in 100,000 risk level						
Benzo(a)pyrene	0.32	H	respiratory tract tumours	1 in 100,000 risk level						
Hexachlorobenzene	0.02	I	Hepatocellular carcinoma	1 in 100,000 risk level						
Nickel	0.042	I	Lung and nasal tumours	1 in 100,000 risk level						
1,1,2 Trichloroethane	0.6	I	Hepatocellular carcinoma	1 in 100,000 risk level						
NON CARCINOGENS										
Acetone	30900	A	Neurological		30900	Neurological	A	61800	Neurological	A
Benzene	30	I	Decreased lymphocyte count	Medium confidence in RfC	12.8	Neurological	A	160	Immunological	A
Biphenyl	89	I	Kidney damage	Calculated based on US EPA RfD toddler						
Carbon Disulphide	700	I	Peripheral nervous system dysfunction	Medium confidence in RfC						
Cumene	400	I	Increased kidney/adrenal weights	Medium confidence in RfC						
Hydrochloric Acid	20	I	Nasal hyperplasia	Low confidence in TRV				2100	Irritation	C
Hydrogen Fluoride	14	C	Bone Density Effects					16.4	Respiratory	A
Hydrogen Sulphide	2.0	I	Nasal lesions	Low confidence in TRV	20	Nasal lesion	W	97.6	Respiratory	W
Isopropyl Alcohol	7000	C	kidney; developmental							
Methyl Isobutyl Ketone	3000	I	Reduced fetal body weight/skeletal variations	Low to medium confidence in TRV						
Nickel	0.09	A	Respiratory		0.2	Respiratory.	A			
Sulfuric Acid	1.0	C	Respiratory							
Toluene	400	I	Respiratory	Medium confidence in TRV				3770	Neurological	A
Xylene	180	H	Not Reported		3040	Developmental	A	4340	Neurological	A
No Toxicological Data Found										
Propionaldehyde										

Notes: H – Health Canada 2003, I – Integrated Risk Information System (IRIS) US EPA, W – World Health Organization, A – ATSDR Minimum Risk Level (MRL), C – California EPA

4.6 USE OF H₂S AS A SURROGATE FOR TOTAL REDUCED SULPHUR (TRS)

Total reduced sulphur (TRS) is a sum of sulphur based compounds that predominantly includes a mixture of:

- hydrogen sulphide (H₂S)
- methyl mercaptan (CH₃SH)
- dimethyl sulphide ((CH₃)₂S)
- dimethyl disulphide (CH₃SSCH₃)

The TRS compounds are often referred to as malodorous sulphur compounds. Hydrogen sulphide is a highly odorous and colorless gas with a characteristic rotten egg smell at low concentrations. Methyl mercaptan has been described as having a garlic or rotten cabbage odor. Dimethyl sulphide and dimethyl disulphide also have a pervasive and offensive odor. The odor perception threshold for H₂S varies considerably with individual sensitivities but is usually in the range of 2 µg/m³ to 17 µg/m³. The odor threshold is even lower for methyl mercaptan, at 0.0016 ppm.

The toxicity of effects of inhalation exposure to hydrogen sulphide has been well documented (see Appendix B). It is also the major component of TRS and one of the common SoPCs emitted from pulp and paper mills. However, the toxicity of the remaining sulphur compounds that comprise TRS are either less toxic than H₂S or have little to no toxicological information available.

In order to develop ambient air quality standards, a reliable monitoring method must be established. The equipment used must be able to distinguish between the compound of concern and not be sensitive to interferences from other compounds. Stack testing monitoring equipment does exist for H₂S, however, it is not practicable for continued operation in ambient environments.

While there is monitoring equipment which can quantify H₂S, that equipment is designed to be used for source emissions and not applicable to continued operation in ambient environments. Because of the limitations of available H₂S ambient monitoring equipment, the fact that good ambient monitoring technology for monitoring of TRS exists, and because of the toxicological similarities of H₂S and TRS, Jacques Whitford has considered all ambient measured TRS to be 100% H₂S.

Toxicologically this is a conservative approach for evaluating ambient concentrations of TRS. It is likely that less than 75 % of the TRS measurements are actually the most toxic of the sulphur compounds, H₂S.

4.7 USE OF SURROGATES FOR PAHS AND DIOXINS/FURANS

Polycyclic aromatic hydrocarbons present a unique problem in the assessment of risk. The PAH family contains a large number of substances with a range of biological and substance activities. PAHs are divided into a group of compounds with no apparent carcinogenic activity and a group of compounds that are carcinogenic. Reliable toxicity studies have been performed for relatively few PAH compounds; however, similar structures and substance properties have led to a surrogate approach for estimating toxicity values.

Although there is strong evidence of carcinogenicity for several PAH compounds, only benzo(a)pyrene has reliable carcinogenic toxicity values. The most common method for estimating carcinogenic toxicity values for the other PAH compounds is the Toxicity Equivalency (TEQ) approach adopted by the US EPA (1993). It is assumed that the carcinogenic PAH compounds each have the same biological mechanism of action and biological end-point, but differ in their relative potencies or degrees of carcinogenicity.

The US EPA toxic equivalency factors (TEFs) were used to generate the TEQ concentrations of individual PAH compounds, which were then summed and compared to the toxicity reference value for benzo(a)pyrene.

Speciation of individual dioxins/furans was not provided in the emissions inventory. In accordance to standard practice, all dioxins/furans are represented by 2,3,7,8-TCDD equivalents.

5.0 EXPOSURE ASSESSMENT AND RISK CHARACTERIZATION

This section describes the concentrations of SoPCS in ambient air surrounding the Crofton Division Mill. The exposure assessment can be divided into two distinct components; the ambient air quality monitoring that is routinely conducted by NorskeCanada and the air modeling exercise undertaken by Jacques Whitford (Vol. I).

Vol. I described the methodologies used in estimating exposure point concentrations (EPCs) of each SoPC in ground level ambient air surrounding the mill property.

The purpose of the risk characterization is to combine the information from the toxicity assessment and the results of the exposure assessment to estimate the potential risks to human health from the SoPC evaluated. This section briefly summarizes the general approach to the risk characterization for non-carcinogenic SoPC and carcinogenic SoPC, respectively, and presents the results for each receptor evaluated in this risk assessment.

5.1 APPROACH

Risk characterization is essentially a comparison of the predicted human intake, in this case either ambient air monitored data or modeled data, of a SoPC to the TRV or Health Based Guideline for that SoPC. For evaluation of potential chronic health risks, chronic daily intakes, based on annual average air concentrations and deposition rates, are compared to chronic TRVs or Health Based Guideline. For evaluation of potential acute health risks, short-term daily intakes, based on 1-hour and 24-hour air concentrations, are compared to acute TRVs or Health Based Guideline.

5.2 AMBIENT AIR QUALITY

The following section details the results of the investigation of ambient air quality measured at the monitoring stations from 1994 to 2004 (Figure 5-1). All data were provided by NorskeCanada Crofton Division and is the same dataset as reported to the Ministry ADAMS system (Air Data Acquisition & Management System). Details of the ambient air monitoring program that is undertaken by NorskeCanada Crofton Division can be found in Section 3 of Vol. I.

The employees of Crofton Division were on strike from July 14, 1997 to April 19, 1998. During this time the mill was not in operation and thus not emitting substances into the local air shed. In addition, by March of 2002, Crofton Division had made several upgrades to the stack of Number 4 Power Boiler. These key dates were used to temporally examine the ambient air quality datasets as follows:

Prestrike	1994 to July 16, 1997
Strike	July 17, 1997 to April 19, 1998
Pre-Upgrade	April 20, 1998 to February 28, 2002
Post-Upgrade	March 1, 2002 to April 2004

Figure 5-1 Location of Ambient Air Quality Stations

5.2.1 Ambient Air Quality Monitoring Results for Total Reduced Sulphur as H₂S

There are actually 5 ambient air monitoring stations in the local air shed that monitor TRS from which data are available between January 2000 to December 2001. Figure 5-2 shows the relationship of TRS concentrations in ambient air with distance from Crofton Division. There is a significant log linear relationship with the average TRS concentration at each station and distance from the mill ($r^2 = 0.96$). Not surprisingly the highest concentrations of TRS were recorded at the Crofton Substation, located approximately 500 m from the centre of Crofton Division, still within the property boundary.

Results of Analysis of Variance testing indicated that there was no significant difference between the TRS concentrations at the three furthest stations from the mill ($p > 0.26$). This suggests that at 8 km from the mill the ambient TRS concentration in air, which are approximately $1 \mu\text{g}/\text{m}^3$ are the same and are on average below the limit of odour detection by the majority of the population. This is also below the chronic TRV of $2.0 \mu\text{g}/\text{m}^3$.

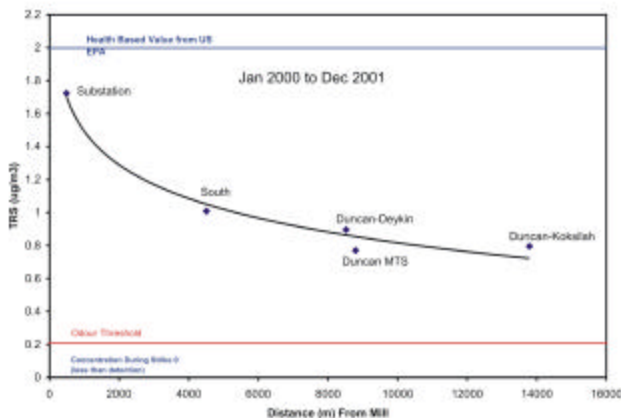


Figure 5-2 Relationship Distance of TRS by Station Away from Crofton Division

Table 5-1 shows the TRS exceedances above Level A (desired level of air quality in B.C.) and Level B (interim guidance on air quality in B.C.) B.C. odour based AQOs and a comparison to the health based toxicity reference values for H₂S. Again there was a decrease in the percent of time that TRS exceeded either Level A or Level B objectives with distance from the mill. Between March 1, 2002 and April 30, 2004 TRS exceed the odour based Level A AQOs up to 17% of the time at Substation and less than 5% of the time for Level B AQOs. There is a significant decrease in the percent exceedances with distance from the Crofton mill.

Regardless of the odour based exceedances of TRS, it can be seen that less than 0.15% of the time did the TRS concentrations, even at the substation, exceed either the acute or sub-chronic health based toxicity value for H₂S.

At the South Station and Duncan-Deykin Station there were no health based exceedances of H₂S. This suggests that although residents of the air shed may be able to detect the odorous TRS compounds (threshold approximately $2 \mu\text{g}/\text{m}^3$ for 20 days or more in any given year (4.3% exceedance of Level A 24 hr objective multiplied by 365 days per year), there are no H₂S health effects associated with the smell of the pulp and paper mill.

Table 5-1 Ambient Air Quality Exceedances of Level A and Level B Ambient Air Quality Objectives Post-Upgrade Installation (March 1, 2002 to April 30, 2004)

Monitoring Station	B.C. Level A AQO		B.C. Level B AQO		H ₂ S Health Based Toxicity Reference Values	
	% Exceedance of 1 hr (7 ug/m ³)	% Exceedance of 24 hr (3 ug/m ³)	% Exceedance of 1 hr (28 ug/m ³)	% Exceedance of 24 hr (6 ug/m ³)	% Exceedance of 1 hr Acute TRV (100 ug/m ³)	% Exceedance of 24 hr Sub Chronic TRV (20 ug/m ³)
Substation	6.5	17	0.55	4.5	0.011	0.13
South	1.7	4.3	0.041	0.65	0	0
Duncan-Deykin	0.51	0.79	0	0.13	0	0

Figure 5-3 shows the concentrations of TRS measured at Crofton Substation over varying time periods. It is apparent from the ANOVA graph that during the strike that TRS was not measured and thus reported as half the detection limit ($0.5 \mu\text{g}/\text{m}^3$) throughout this time period. It also indicates that there was a significant drop in TRS concentrations after the strike 1998 ($p=0.003$), which has remained consistent since that time.

Given that TRS could be measured at the Duncan-Koksilah, which is located approximately 14 km from the mill, it suggests that on several days throughout the year it may be possible to detect the odour from the Crofton Division at great distances from the mill. However, the level of TRS is well below the health based acute and sub-chronic TRVs for hydrogen sulphide.

In addition, the annual average monitored concentration of TRS at all three stations were below the chronic TRV of $2.0 \mu\text{g}/\text{m}^3$:

Crofton Substation	$1.9 \mu\text{g}/\text{m}^3$
South Station	$0.98 \mu\text{g}/\text{m}^3$
Duncan-Deykin	$0.76 \mu\text{g}/\text{m}^3$

Therefore, although residents in the local air shed may be able to detect the odour coming from the mill, it does not pose an acute, sub-chronic, or chronic health threat to local residents occupying the area year round.

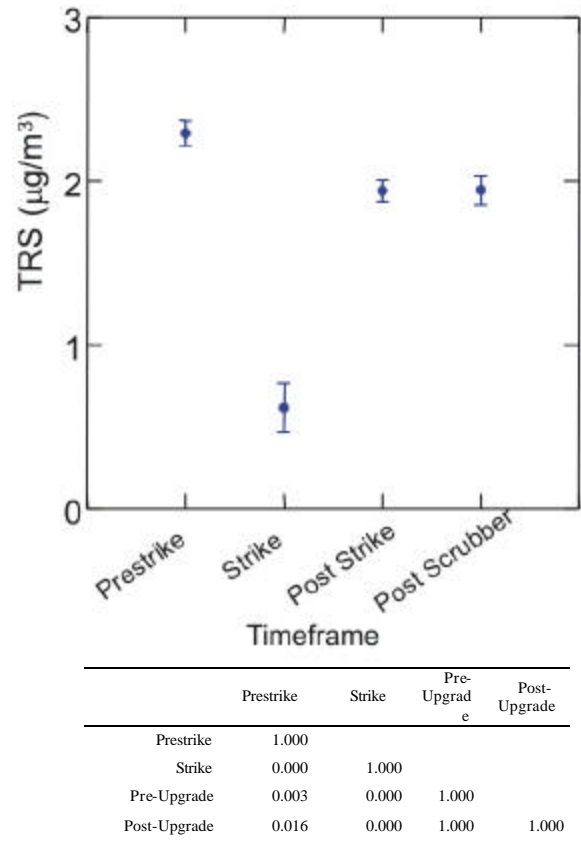


Figure 5-3 TRS concentrations measured at Crofton Substation between 1994 and 2004. Pairwise comparisons (p values) are also shown (p values <0.05 are considered significant).

5.2.1.1 Ambient Air Quality Monitoring Results for Particulate Matter <10mm

Table 5-2 shows the average concentration of PM₁₀ measured at the three monitoring stations from 1994 to 2004. Given that there are multiple sources of PM₁₀ that could be contributing to PM₁₀ levels in the area, including automobiles and wood fired stoves, the results of the modeled data appear to be a reasonable correlation with that found in the environment.

Figure 5-4 shows PM₁₀ concentrations measured at each of the monitoring stations over the years for which data were available. Each of the graphs also has the PM₁₀ and PM_{2.5} guideline concentrations illustrated on the graphs. The PM₁₀ guideline for B.C. is 50 µg/m³ over a 24 hour period. At the Substation only 5 exceedances of this criterion were observed over a seven year period or 1.1% exceedance. At the South Station there were only 2 exceedances of PM₁₀ objectives or less than 0.5% exceedances, and similar results found at the Duncan-Deykin station.

Table 5-2 PM₁₀ Monitoring Data and Modelled Data (n = number of samples)

Monitoring Station	PM10 (mg/m ³)						
	All Years		Post-Upgrade March 2002 to April 2004		Model		
	average	n	average	n	1 hr	24 hr	annual
Substation	12 ± 9.6	448	12 ± 7.8	138	70	14	1.3
South Station	9.9 ± 6.5	437	10 ± 5.6	142	30	5.7	0.65
Duncan-D	11 ± 4.5	1998	11 ± 4.5	790	18	4.6	0.4

Although PM₁₀ is monitored at the stations if it is conservatively assumed that all of the measured PM₁₀ was in fact PM_{2.5} (actual ratio is closer to 0.7) then very rarely do the 24 hour or even hourly measurements at any of the three stations exceed the CWS guideline (30 µg/m³), which will take effect in 2010. In addition, the 98th percentile of PM₁₀ concentrations was below the PM_{2.5} guideline.

Through ANOVA analysis (Figure 5-5) it was determined that there was significantly more PM₁₀ in the ambient air at the Substation over that found in the other two monitoring stations (p<0.05). However, the average concentration of PM₁₀ was well below standards provided by Canada and the B.C. government.

Examination of the temporal trends of PM₁₀ concentrations at Crofton Substation (Figure 5-6) indicate that there was no significant difference (p>0.60), albeit marginally, in PM₁₀ concentrations at the station even while the mill was shut down during the strike period. This suggests that Crofton Division is not the sole contributor of PM₁₀ to the local area.

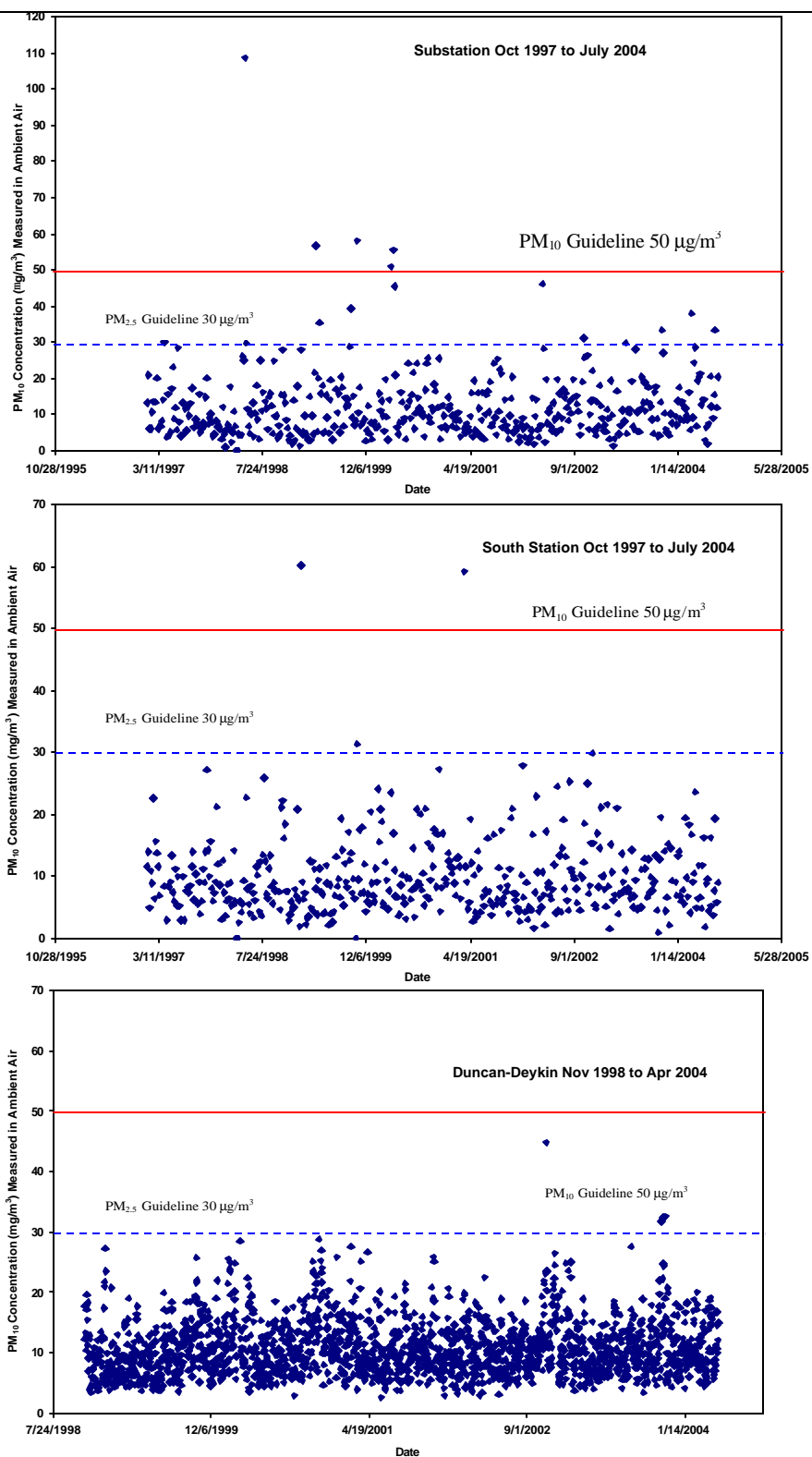


Figure 5-4 PM₁₀ Ambient Air Quality Monitoring Results for the Three Monitoring Stations.

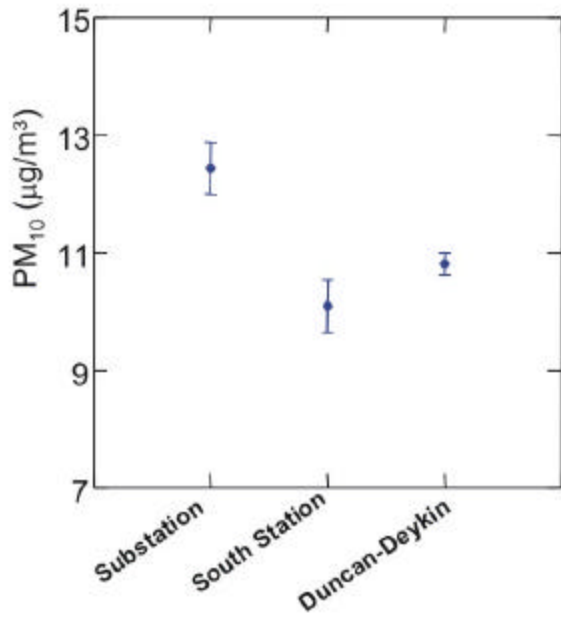


Figure 5-5 PM₁₀ ANOVA at Stations with Distance from the Crofton Division.

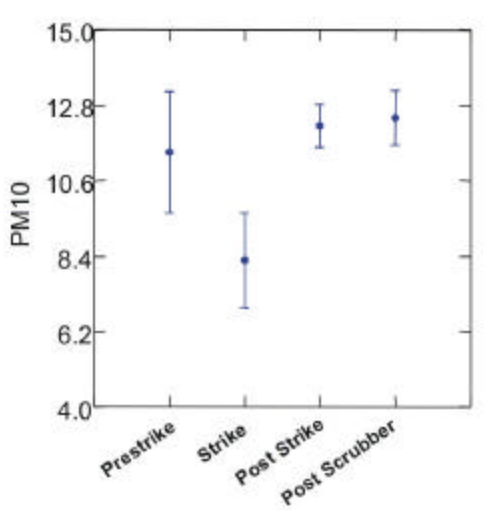


Figure 5-6 PM₁₀ ANOVA concentrations measured at Crofton Substation between 1994 and 2004. Pairwise comparisons (p values) are also shown.

	Prestrike	Strike	Pre-Upgrade	Post-Upgrade
Prestrike	1.000			
Strike	0.973	1.000		
Pre-Upgrade	1.000	0.063	1.000	
Post-Upgrade	1.000	0.060	1.000	1.000

In 2003, the B.C. Ministry of Water, Land and Air Protection (MLWAP) released a report entitled:

Particulate Matter in British Columbia: A Report on PM₁₀ and PM_{2.5} Mass Concentrations up to 2000.

This report provides a summary of PM₁₀ and PM_{2.5} concentrations in ambient air over several monitoring stations throughout British Columbia. Overall mean PM₁₀ concentrations for the entire period of record up to 2000 ranged from 7 to 26 µg/m³ at monitoring sites with at least one year of data (Table 5-3). The highest concentrations were observed in the interior of the province, particularly at sites in Golden, Prince George, Quesnel and Boston Bar, which each recorded overall mean 24-hour concentrations in excess of 20 µg/m³. However, the majority of sites in coastal areas recorded mean concentrations of less than 15 µg/m³.

Approximately two-thirds of these monitoring sites reported exceedance frequencies of 1% or less of the 50 µg/m³, while the remaining one-third of all sites experienced exceedance frequencies ranging from 2 to 7% of the time. The Crofton Substation exceeded the B.C. Ministry guideline only 1% of the time. Comparison of the data from the three monitoring stations to other areas in British Columbia indicate that the PM₁₀ levels are lower than many other cities or towns of a similar size to Crofton, B.C.

Overall PM₁₀ levels in the local air shed are low enough that under current ambient air quality guidelines and objectives they do not pose an unreasonable threat to the health of the local residents. In fact, given that the ambient concentrations of PM are among the lowest measured in the province of B.C. no further consideration was given to PM in this study.



Table 5-3 Ranked mean 24-hour average PM₁₀ concentrations (in µg/m³) based on all data from sites in British Columbia with at least one year of data (MLWAP, 2003)

ID	Station Name	Start Date	End Date	N	Overall Mean
E235070	Golden Hospital	11-Feb-99	24-Dec-00	625	26
E224013	Prince George B.C.Rail	29-Sep-96	31-Dec-00	1548	24
E221885	Quesnel Pinecrest	10-Jun-95	31-Dec-00	1954	22
E238240	Boston Bar RCMP Station	09-Jul-99	31-Dec-00	533	22
E208096	Quesnel Sr. Sec.	17-Apr-94	31-Dec-00	2419	22
0450307	PG Plaza 400	01-Mar-92	31-Dec-00	3042	21
E206243	Cranbrook Swimming Pool	01-May -92	15-Sep-93	432	21
0550502	Williams Lake	17-Dec-92	31-Dec-00	2646	20
M107004	Houston Firehall	20-Sep-94	31-Dec-00	2213	20
E237431	Merritt Granite-Garcia Mobile	16-May -99	28-May -00	376	19
E225267	Burns Lake Fire Centre	08-Mar-97	31-Dec-00	1319	19
E221199	Creston PC School	27-Oct-94	31-Dec-00	1730	18
E206589	Smithers St. Josephs	15-May -92	31-Dec-00	2956	18
E206612	Chilliwack Work Yard	02-Nov-94	28-Feb-95	101	17
0250009	Trail Butler Park	12-Apr-94	08-May -00	1751	17
E217029	Abbotsford Library	20-Jul-94	14-Sep-98	1391	17
E220203	Cranbrook PR3	21-Apr-94	29-Nov-98	1615	17
E216667	Quesnel Maple Dr.	25-May -95	31-Dec-00	1997	16
E225868	Prince George Hart Highlands	05-Apr-97	21-Mar-98	351	16
E230557	Telkwa	05-Feb-98	31-Dec-00	1010	16
0500886	Kelowna College	23-Jan-94	31-Dec-00	2511	16
E206898	Kamloops Brocklehurst	01-Jan-94	31-Dec-00	2521	15
0310162	Port Moody Rocky Pt. Park	01-Nov-93	31-Dec-00	2449	15
0450270	PG Gladstone	07-Dec-95	31-Dec-00	1828	15
E207417	Richmond South	28-Oct-93	31-Dec-00	2557	14
0310175	Kitsilano	15-Dec-93	31-Dec-00	2527	14
E232246	Vancouver International Airport #2	01-Feb-98	31-Dec-00	1055	14
E207418	Burnaby South	30-Mar-94	31-Dec-00	2443	14
M107028	Terrace B.C. Access Centre	18-Dec-96	31-Dec-00	1459	14
0310172	Squamish	12-Sep-94	14-Dec-00	2176	14
E220891	Chilliwack Airport	01-Mar-95	31-Dec-00	2067	13

ID	Station Name	Start Date	End Date	N	Overall Mean
E207723	North Delta	17-Dec-93	31-Dec-99	2088	13
E238212	Abbotsford Bevan Ave.	19-Sep-98	31-Dec-00	814	13
E206271	Surrey East	07-Jan-94	31-Dec-00	2487	13
E209178	Langley Central	01-Jan-94	31-Dec-00	2398	13
*	Crofton Substation	01-Oct-97	01-Jul-04	448	12
E229798	Campbell River Tyee Split	12-Dec-97	31-Dec-00	1103	12
0310177	Burnaby Kensington Park	14-May -94	31-Dec-00	2379	12
E232245	Maple Ridge Golden Ears Elem.	21-Feb-98	31-Dec-00	1020	12
E222520	Elk Falls	09-Dec-95	31-Dec-00	1586	12
*	Duncan-Deykin	01-Nov-98	01-Apr-04	1998	11
E225378	Quadra Island Lighthouse	23-Feb-97	31-Dec-00	1361	11
E234670	Duncan Deykin Ave.	01-Nov-98	31-Dec-00	778	11
E223756	Hope Airport	04-Dec-96	31-Dec-00	1477	11
*	South Station	01-Oct-97	01-Jul-04	437	10
E225377	Harmac Cedar Woobank	08-Jul-97	31-Dec-00	978	10
E224014	Prince George Glenview School	11-Jun-98	31-Dec-00	927	10
E222778	Langdale Elem.	06-Jan-96	24-Dec-00	1465	10
0220204	Powell River Cranberry Lake	12-Jan-96	31-Dec-00	1764	10
E223827	Kitimat Rail	18-Aug-98	31-Dec-00	857	10
E228065	Port Alberni Townsite	16-Sep-97	31-Dec-00	1183	10
E223616	Kitimat Haul Rd.	11-Aug-98	31-Dec-00	868	9
0220205	Powell River Wildwood	19-Jun-97	31-Dec-00	1276	9
0110203	Gold River Pumphouse	06-Feb-97	08-Nov-98	626	8
E216670	Kitimat Riverlodge	11-Aug-98	31-Dec-00	870	7
E231838	PrinceRupert Galloway Rapids	24-Apr-98	31-Dec-00	890	7
E225184	Port Edward Pacific	30-Apr-98	04-Nov-00	879	7

Note: * inserted by Jacques Whitford based on station monitoring data.

5.3 RESULTS OF AIR MODELLING CONCENTRATIONS OF SoPCs

The air modelling results were divided into three receptor groups:

- Maximum Fenceline Concentration
- Maximum Concentration at Special Receptors
- Maximum Concentration at Gridded Receptors

Therefore, concentrations reported for these areas will be representative of the maximum concentrations of SoPCs off property of the Crofton Division mill.

5.3.1 Modelled SoPCs with Health Based AAQC

Table 5-4 to Table 5-6 presents the maximum predicted hourly, 24-hour and annual average concentrations for all receptors and SoPCs that have OMOE health based criteria. These concentrations are based on the screening runs, which were for a smaller computational domain (25-km by 35-km) than the meteorological domain.

The majority of the MOE health based criteria are published as the 24 hour concentration, with the exception of acrolein (1 hr), trichloroethylene (24 hr and annual), NO_x (1 hr, 24 hr) PM (24 hr, annual) and SO₂ (1 hr, 24 hr and annual). Criteria for the remaining timelines were scaling using the OMOE (2001) and US EPA (1992) scaling factors.

Of the 41 compounds that have OMOE Health Based ambient air quality criteria the following were the observed to have concentrations greater than the OMOE criteria (shown in **bold** in the tables):

Fenceline Receptors

HCl (hourly and 24 hr)
NO_x (hourly)
SO₂ (hourly)

Maximum Gridded

Receptors

HCl (hourly and 24 hr)
SO₂ (hourly)

Special Receptors

HCl (hourly and 24 hr)

Tables 5-7 to 5-9 present the same maximum predicted hourly, 24-hour and annual average concentrations for all receptors and SoPCs, however they are compared to WHO Air Quality Guidelines for Europe (2000).

Of the 16 SoPCs that have WHO Health Based ambient air quality criteria only SO₂ and NO_x was observed to have concentrations greater than the WHO criteria (shown in **bold** in Table 5-9) at the maximum gridded receptor located in close proximity to the fenceline.

Therefore, HCl, NO_x and SO₂ were carried forward in the risk assessment for further understanding of the nature of the exceedances and their location. The remaining compounds had modeled concentrations below health based guidelines and therefore are at concentrations below which health based effects would be expected, thus were not considered further in the risk assessment.

Again it should be remembered that by its very nature the modeled results are meant to be conservative overestimates of the actual ground level concentrations in the local air shed. Therefore, it is possible that the concentrations of HCl, NO_x and SO₂ are actually below the guideline values.

Table 5-4 Maximum Modelled Fenceline Concentrations and MOE Criteria (exceedances – bold)

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			MOE Health Based AAQ Criteria ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Acetaldehyde	18.6	2.53	0.59	1250	500	100	
Acrolein	0.509	5.6E-02	9.1E-03	23.3	9.32	1.86	
Ammonia	57.8	10.6	1.69	250	100	20	
Antimony	2.5E-02	1.3E-03	1.7E-04	62.5	25	5	
Arsenic	6.8E-03	3.5E-04	4.8E-05	0.75	0.3	0.06	
Cadmium	6.3E-03	4.7E-04	6.9E-05	5	2	0.4	
Carbon Tetrachloride	0.55	0.14	0.02	6.25	2.4	0.48	
Chlorine	10.4	1.03	0.16	25	10	2	
Chlorine Dioxide	12.2	1.2	0.18	75	30	6	
Chloroform	1.57	0.38	5.1E-02	2.5	1	0.2	
Chloromethane	0.115	2.1E-02	3.7E-03	17500	7000	1400	
Chromium	5.4E-03	4.1E-04	6.6E-05	3.75	1.5	0.3	
Cobalt	2.9E-02	1.5E-03	1.9E-04	0.25	0.1	0.02	
Copper	2.7E-02	2.4E-03	3.6E-04	125	50	10	
Cresol	11.6	3.22	0.43	187.5	75	15	
Dichloromethane	1.47	9.1E-02	8.2E-03	550	220	44	
Formaldehyde	5.81	0.704	0.124	162.5	65	13	
Chromium VI	2.4E-02	1.5E-03	1.5E-04	3.75	1.5	0.3	
Hydrochloric Acid	1080	53.1	2.88	50	20	4	YES
Lead	3.4E-02	2.1E-03	1.9E-04	5	2	0.4	
Manganese	0.330	1.7E-02	1.2E-03	6.25	2.5	0.5	
Methanol	620	115	18.9	10000	4000	800	
Methyl Ethyl Ketone	3.09	0.920	0.140	2500	1000	200	
Naphthalene	2.73	0.181	0.029	56.25	22.5	4.5	
Phenol	6.05	1.52	0.206	250	100	20	
Selenium	1.5E-02	7.7E-04	5.8E-05	25	10	2	
Silver	2.3E-03	2.4E-04	3.9E-05	2.5	1	0.2	
Styrene	0.87	0.130	2.4E-02	1000	400	80	
Tetrachloroethylene	1.35	0.255	4.1E-02	900	360	72	
1,2,4-Trichlorobenzene	3.77	0.385	7.2E-02	1000	400	80	
Trichloroethylene	0.569	0.143	1.9E-02	287.5	115	23	
1,2,4-Trimethylbenzene	5.3E-03	5.4E-04	8.4E-05	2500	1000	200	
Zinc	2.64	0.142	1.9E-02	300	120	24	
Dioxins & Furans	2.7E-06	1.3E-07	6.6E-09	1.25E-02	5.0E-03	1.0E-03	
Mercury	3.2E-03	1.9E-04	2.0E-05	5	2	0.4	
CO	1430	102	10.4	39250	15700	3140	
NOx	1490	73.9	6.77	400	200	40	YES
PM _{2.5}	305	17.1	2.09	NV	25	5	
PM ₁₀	436	24	2.89	NV	50	10	
SO₂	1250	105	14.1	695	275	55	YES
TPM	504	27.4	3.32	NV	150	60	

Table 5-5 Maximum Modelled Concentration at Special Receptors and Health Based (exceedances – bold)

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			MOE Health Based AAQ Criteria ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Acetaldehyde	21.9	2.36	0.37	1250	500	100	
Acrolein	0.896	7.1E-02	6.3E-03	23.3	9.32	1.86	
Ammonia	31.4	6.19	0.92	250	100	20	
Antimony	6.0E-03	8.2E-04	1.4E-04	62.5	25	5	
Arsenic	1.6E-03	2.3E-04	4.0E-05	0.75	0.3	0.06	
Cadmium	1.7E-03	3.2E-04	6.0E-05	5	2	0.4	
Carbon Tetrachloride	0.414	3.5E-02	6.1E-03	6.25	2.4	0.48	
Chlorine	4.93	0.846	7.8E-02	25	10	2	
Chlorine Dioxide	5.77	0.989	9.1E-02	75	30	6	
Chloroform	1.13	0.103	1.9E-02	2.5	1	0.2	
Chloromethane	9.2E-02	1.2E-02	2.2E-03	17500	7000	1400	
Chromium	2.1E-03	2.9E-04	5.8E-05	3.75	1.5	0.3	
Cobalt	7.0E-03	9.4E-04	1.6E-04	0.25	0.1	0.02	
Copper	6.8E-03	1.6E-03	2.8E-04	125	50	10	
Cresol	8.06	0.900	0.169	187.5	75	15	
Dichloromethane	0.23	0.04	0.01	550	220	44	
Formaldehyde	3.42	0.496	8.1E-02	162.5	65	13	
Chromium VI	4.1E-03	7.2E-04	1.2E-04	3.75	1.5	0.3	
Hydrochloric Acid	123	23.9	2.51	50	20	4	YES
Lead	5.1E-03	9.8E-04	1.6E-04	5	2	0.4	
Manganese	4.1E-02	8.1E-03	1.0E-03	6.25	2.5	0.5	
Methanol	571	43.0	8.24	10000	4000	800	
Methyl Ethyl Ketone	2.82	0.320	6.5E-02	2500	1000	200	
Naphthalene	1.25	0.168	2.3E-02	56.25	22.5	4.5	
Phenol	5.03	0.461	8.4E-02	250	100	20	
Selenium	1.7E-03	3.6E-04	5.0E-05	25	10	2	
Silver	6.5E-04	1.7E-04	2.5E-05	2.5	1	0.2	
Styrene	1.47	0.125	1.3E-02	1000	400	80	
Tetrachloroethylene	2.24	0.190	2.1E-02	900	360	72	
1,2,4-Trichlorobenzene	6.61	0.516	4.4E-02	1000	400	80	
Trichloroethylene	0.399	3.6E-02	6.7E-03	287.5	115	23	
1,2,4-Trimethylbenzene	2.6E-03	4.5E-04	4.2E-05	2500	1000	200	
Zinc	0.647	9.1E-02	1.6E-02	300	120	24	
Dioxins & Furans	3.0E-07	5.7E-08	5.8E-09	1.25E-02	5.0E-03	1.0E-03	
Mercury	5.7E-04	9.4E-05	1.7E-05	5	2	0.4	
CO	233	54.7	7.51	39250	15700	3140	
NOx	258	39.1	5.91	400	200	40	
PM _{2.5}	57.4	8.77	1.61	NV	25	5	
PM ₁₀	82.5	12.5	2.3	NV	50	10	
SO ₂	575	65.7	12.2	695	275	55	
TPM	94.3	14.4	2.65	NV	150	60	

Table 5-6 Maximum Modelled Concentration at Gridded Receptors and Health Based Criteria (exceedances – **bold)**

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			MOE Health Based AAQ Criteria ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Acetaldehyde	84.2	25.2	6.27	1250	500	100	
Acrolein	0.970	0.11	0.03	23.3	9.32	1.86	
Ammonia	96.8	16.9	1.06	250	100	20	
Antimony	1.8E-02	1.6E-03	2.2E-04	62.5	25	5	
Arsenic	4.8E-03	4.4E-04	6.1E-05	0.75	0.3	0.06	
Cadmium	5.9E-03	5.5E-04	9.0E-05	5	2	0.4	
Carbon Tetrachloride	0.49	0.11	0.02	6.25	2.4	0.48	
Chlorine	13.9	2.32	0.25	25	10	2	
Chlorine Dioxide	16	3.62	0.48	75	30	6	
Chloroform	1.4	0.3	0.05	2.5	1	0.2	
Chloromethane	0.276	4.0E-02	6.9E-03	17500	7000	1400	
Chromium	5.7E-03	8.2E-04	8.4E-05	3.75	1.5	0.3	
Cobalt	2.0E-02	1.9E-03	2.6E-04	0.25	0.1	0.02	
Copper	1.9E-02	3.6E-03	4.7E-04	125	50	10	
Cresol	28.2	8.47	2.20	187.5	75	15	
Dichloromethane	0.760	7.0E-02	1.6E-02	550	220	44	
Formaldehyde	4.70	0.821	0.197	162.5	65	13	
Chromium VI	1.2E-02	1.1E-03	1.7E-04	3.75	1.5	0.3	
Hydrochloric Acid	555	30.7	3.24	50	20	4	YES
Lead	1.7E-02	1.4E-03	2.3E-04	5	2	0.4	
Manganese	0.170	1.0E-02	1.4E-03	6.25	2.5	0.5	
Methanol	767	152	20.2	10000	4000	800	
Methyl Ethyl Ketone	7.34	1.99	0.576	2500	1000	200	
Naphthalene	3.22	0.384	6.9E-02	56.25	22.5	4.5	
Phenol	14	4.12	1.12	250	100	20	
Selenium	7.1E-03	4.9E-04	6.8E-05	25	10	2	
Silver	3.1E-03	2.9E-04	5.1E-05	2.5	1	0.2	
Styrene	1.67	0.240	8.0E-02	1000	400	80	
Tetrachloroethylene	2.68	0.604	0.172	900	360	72	
1,2,4-Trichlorobenzene	7.37	0.864	0.275	1000	400	80	
Trichloroethylene	1.29	0.425	0.109	287.5	115	23	
1,2,4-Trimethylbenzene	7.2E-03	1.2E-03	1.3E-04	2500	1000	200	
Zinc	1.88	0.180	2.0E-02	300	120	24	
Dioxins & Furans	1.4E-06	7.3E-08	7.4E-09	1.25E-02	5.0E-03	1.0E-03	
Mercury	1.8E-03	1.5E-04	2.2E-05	5	2	0.4	
CO	849	94.5	12.2	39250	15700	3140	
NOx	305	59.1	6.4	400	200	40	
PM _{2.5}	226	15.3	2.46	NV	25	5	
PM ₁₀	323	21.5	3.35	NV	50	10	
SO₂	1460	205	16.3	695	275	55	YES
TPM	109	23.1	2.9	NV	150	60	

Table 5-7 Maximum Modelled Fenceline Concentrations and WHO Criteria (exceedances – **bold)**

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			WHO Health Based AAQ Guidelines ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Arsenic	6.80E-03	3.50E-04	4.80E-05			6.7E-03	
Cadmium	6.30E-03	4.70E-04	6.90E-05			5.0E-03	
Dichloromethane	1.47	9.10E-02	8.20E-03		3000		
Chromium VI	2.40E-02	1.50E-03	1.50E-04			2.5E-04	
Lead	3.40E-02	2.10E-03	1.90E-04			0.5	
Manganese	0.33	1.70E-02	1.20E-03			0.15	
Tetrachloroethylene	1.35	0.255	4.10E-02			250	
Trichloroethylene	0.569	0.143	1.90E-02			23	
Mercury	3.20E-03	1.90E-04	2.00E-05			1	
CO	1430	102	10.4	3000			
NOx	1490	73.9	6.77	200		40	Yes
SO2	1250	105	14.1		125	50	
Benzene	1.17	7.10E-02	6.60E-03			1.7	
Carbon Disulphide	2.39	0.2	2.60E-02		100		
Hydrogen Sulphide	212	28.4	5.63		150		
Nickel	0.579	3.00E-02	3.90E-03			2.5E-02	

Table 5-8 Maximum Modelled Special Receptor Concentrations and WHO Criteria (exceedances – **bold)**

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			WHO Health Based AAQ Guidelines ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Arsenic	1.60E-03	2.30E-04	4.00E-05			6.7E-03	
Cadmium	1.70E-03	3.20E-04	6.00E-05			5.0E-03	
Dichloromethane	0.23	0.04	0.01		3000		
Chromium VI	4.10E-03	7.20E-04	1.20E-04			2.5E-04	
Lead	5.10E-03	9.80E-04	1.60E-04			0.5	
Manganese	4.10E-02	8.10E-03	1.00E-03			0.15	
Tetrachloroethylene	2.24	0.19	2.10E-02			250	
Trichloroethylene	0.399	3.60E-02	6.70E-03			23	
Mercury	5.70E-04	9.40E-05	1.70E-05			1	
CO	233	54.7	7.51	3000			
NOx	258	39.1	5.91	200		40	
SO2	575	65.7	12.2		125	50	
Benzene	0.291	3.50E-02	5.80E-03			1.7	
Carbon Disulphide	0.881	0.152	1.70E-02		100		
Hydrogen Sulphide	159	17.9	2.57		150		
Nickel	0.139	1.90E-02	3.30E-03			2.5E-02	

Table 5-9 Maximum Modelled Concentration at Gridded Receptors and WHO Criteria (exceedances – **bold)**

SoPC	Maximum Modelled Ground level Concentrations ($\mu\text{g}/\text{m}^3$)			WHO Health Based AAQ Guidelines ($\mu\text{g}/\text{m}^3$)			Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Hourly	24 Hour	Annual	
Arsenic	4.80E-03	4.40E-04	6.10E-05			6.7E-03	
Cadmium	5.90E-03	5.50E-04	9.00E-05			5.0E-03	
Dichloromethane	0.76	7.00E-02	1.60E-02		3000		
Chromium VI	1.20E-02	1.10E-03	1.70E-04			2.5E-04	
Lead	1.70E-02	1.40E-03	2.30E-04			0.5	
Manganese	0.17	1.00E-02	1.40E-03			0.15	
Tetrachloroethylene	2.68	0.604	0.172			250	
Trichloroethylene	1.29	0.425	0.109			23	
Mercury	1.80E-03	1.50E-04	2.20E-05			1	
CO	849	94.5	12.2	3000			
NOx	305	59.1	6.4	200		40	
SO2	1460	205	16.3		125	50	YES
Benzene	0.571	5.00E-02	8.40E-03			1.7	
Carbon Disulphide	14.5	4.22	1.04		100		
Hydrogen Sulphide	137	46.6	9.5		150		
Nickel	0.412	3.70E-02	5.10E-03			2.5E-02	

5.3.2 Modelled SoPCs with No Health Based AAQ Guidelines

Table 5-10 to Table 5-12 presents the maximum predicted hourly, 24-hour and annual average concentrations for all receptors and SoPCs that do not have OMOE or WHO health based criteria. The modelled ground level ambient air concentrations of these SoPCs were compared to toxicity reference values. The toxicity reference values are presented in Table 4-2 and discussed in detail in Appendix B.

Of the 16 substances of potential concern that do not have MOE Health Based AAQC only hydrogen sulphide (H₂S) exceeded for each of the receptor groups across all of the timelines. The remaining compounds had modeled concentrations below health based toxicity reference values from other jurisdictions and therefore are at concentrations below which health based effects would be expected, thus were not considered further in the risk assessment.

Therefore, hydrogen sulphide was carried forward in the risk assessment.

Table 5-10 Maximum Modelled Fenceline Concentration with Non Health Based Criteria (exceedances – bold)

SoPC	Maximum Modelled Ground level Concentrations (mg/m ³)			Toxicity Reference Values (mg/m ³)				Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Acute	Intermediate	chronic	carcinogenic	
Acetone	40.2	6.09	1.05	61800	30900	30900		
Benzene	1.17	7.1E-02	6.6E-03	160	12.8	30	3.03	
Biphenyl	18.5	0.967	9.5E-02			89		
Carbon Disulphide	2.39	0.200	2.6E-02			700		
Cumene	0.228	2.8E-02	3.7E-03			400		
Hydrogen Fluoride	0.152	7.7E-03	9.7E-04	16.4		14		
Hydrogen Sulphide	212	28.4	5.63	100	20	2		Yes
Isopropyl Alcohol	11.7	0.636	4.7E-02			7000		
Methyl Isobutyl Ketone	0.629	7.3E-02	1.5E-02			3000		
Nickel	0.579	3.0E-02	3.9E-03		0.2	0.09	0.042	
Propionaldehyde	4.86	0.366	6.2E-02					
Sulfuric Acid	12.5	0.926	0.144			1.0		
Toluene	0.164	4.7E-02	6.8E-03	3770		400		
1,1,2-Trichloroethane	0.505	2.8E-02	2.2E-03				0.6	
Xylene	0.646	7.3E-02	1.5E-02	4340	3040	180		
Hexachlorobenzene	2.0E-04	9.3E-06	4.8E-07				0.02	

Table 5-11 Maximum Modelled Concentration at Special Receptors with Non Health Based Criteria (exceedances – bold)

SoPC	Maximum Modelled Ground level Concentrations (mg/m ³)			Toxicity Reference Values (mg/m ³)				Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Acute	Intermediate	chronic	carcinogenic	
Acetone	35.9	3.29	0.558	61800	30900	30900		
Benzene	0.291	3.5E-02	5.8E-03	160	12.8	30	3.03	
Biphenyl	8.0133	0.8089	8.4E-02			89		
Carbon Disulphide	0.881	0.152	1.7E-02			700		
Cumene	0.197	2.2E-02	3.2E-03			400		
Hydrogen Fluoride	3.6E-02	4.8E-03	8.1E-04	16.4		14		
Hydrogen Sulphide	159	17.9	2.57	100	20	2		Yes
Isopropyl Alcohol	1.27	0.259	3.4E-02			7000		
Methyl Isobutyl Ketone	1.12	9.6E-02	9.9E-03			3000		
Nickel	0.139	1.9E-02	3.3E-03		0.2	0.09	0.042	
Propionaldehyde	2.09	0.281	4.1E-02					
Sulfuric Acid	3.81	0.685	0.125			1.0		
Toluene	0.121	0.01503	3.2E-03	3770		400		
1,1,2-Trichloroethane	5.6E-02	1.1E-02	1.5E-03				0.6	
Xylene	1.12	0.091275	8.8E-03	4340	3040	180		
Hexachlorobenzene	2.1E-05	4.2E-06	4.2E-07				0.02	

Table 5-12 Maximum Modelled Concentration at Gridded Receptors with Non Health Based Criteria (exceedances – bold)

SoPC	Maximum Modelled Ground level Concentrations (mg/m ³)			Toxicity Reference Values (mg/m ³)				Carried Forward in Risk Assessment
	Hourly	24-Hour	Annual	Acute	Intermediate	chronic	carcinogenic	
Acetone	53.8	11.6	2.88	61800	30900	30900		
Benzene	0.571	5.0E-02	8.4E-03	160	12.8	30	3.03	
Biphenyl	22.9	2.48	0.390			89		
Carbon Disulphide	14.5	4.22	1.04			700		
Cumene	0.94742	9.7E-02	1.4E-02			400		
Hydrogen Fluoride	0.10816	9.5E-03	1.3E-03	16.4		14		
Hydrogen Sulphide	137	46.6	9.5	100	20	2		Yes
Isopropyl Alcohol	5.94	0.751	0.197			7000		
Methyl Isobutyl Ketone	3.55	1.04	0.260			3000		
Nickel	0.412	3.7E-02	5.1E-03		0.2	0.09	0.042	
Propionaldehyde	5.78	0.440	0.07					
Sulfuric Acid	15.822	1.20	0.180			1.0		
Toluene	0.426	0.110	3.7E-02	3770		400		
1,1,2-Trichloroethane	0.257	3.3E-02	8.8E-03				0.6	
Xylene	1.24	0.149	5.4E-02	4340	3040	180		
Hexachlorobenzene	1.0E-04	5.3E-06	5.3E-07				0.02	

5.3.3 Polycyclic Aromatic Hydrocarbons

The toxic equivalents (TEQs) method estimates the potential carcinogenic risk from exposure to PAHs by determining the toxic equivalence of the individual carcinogenic PAHs to benzo(a)pyrene (B(a)P), the most carcinogenic, and best studied of these compounds.

Table 5-13 presents the results of PAH screening for the maximum concentrations of PAHs detected, which was at the Gridded Receptor. Given that PAHs are carcinogenic, then only the annual concentration is modeled using the TEQ methodology. It was determined that even at the highest concentrations of PAHs, there was no increased cancer risk above a benchmark of one person in one hundred thousand.

In addition, even if one assumes that all of the 17 PAHs modeled at the maximum gridded receptor ($2.78\text{E-}04 \mu\text{g}/\text{m}^3$) had a the same carcinogenic potential as benzo(a)pyrene (which is very conservative) then even still there is no increased incremental carcinogenic risk greater than 1 in 100,000 people, in comparison to the Health Canada derived Carcinogenic Exposure Value for B(a)P of $0.32 \mu\text{g}/\text{m}^3$. Therefore, there is no unacceptable carcinogenic health risk to the residents of the local air shed from exposure to PAH concentrations emitted from the Crofton mill.

Table 5-13 PAH Screening Using TEQ Method

PAH Compound	Annual Concentration (mg/m^3)	Toxic Equivalency Factors	Annual Toxicity Equivalent ($\text{mg TEQ}/\text{m}^3$)
Benzo(a)anthracene	1.83E-05	0.1	1.83E-06
Benzo(a)phenanthrene	1.83E-05	0.01	1.83E-07
Benzo(a)pyrene	1.82E-05	1	1.82E-05
Benzo(b)fluoranthene	1.81E-05	0.1	1.81E-06
Benzo(g,h,i)perylene	1.81E-05	0.01	1.81E-07
Benzo(k)fluoranthene	1.81E-05	0.1	1.81E-06
Dibenzo(a,h)anthracene	1.80E-05	1	1.80E-05
Fluoranthene	2.02E-05	0.001	2.02E-08
Indeno(1,2,3-c,d)pyrene	1.81E-05	0.1	1.81E-06
Phenanthrene	2.27E-05	0.001	2.27E-08
Pyrene	8.66E-05	0.001	8.66E-08
SUM Annual Toxicity Equivalent ($\text{mg TEQ}/\text{m}^3$)			4.40E-05
Carcinogenic Exposure Value for B(a)P (mg/m^3) 1 in 100,000 cancer risk			0.32
Carried Forward in Risk Assessment			NO

5.3.4 Evaluation of SoPC with No Toxicity Reference Value - Propionaldehyde

Propionaldehyde was the only SoPC for which no toxicity reference values could be found. In addition to the standard sources of TRVs (Health Canada, IRIS, etc.) a primary literature search was conducted using Biological Abstracts and PubMed databases. No scientific journal articles were found that dealt with the toxicity of propionaldehyde for either humans or animals. Therefore, propionaldehyde could not be evaluated as part of this assessment.

The OMOE does provide an odour based guideline for propionaldehyde of $2.5 \mu\text{g}/\text{m}^3$ for a 24 hour basis. The maximum gridded receptor concentration of propionaldehyde was $0.85 \mu\text{g}/\text{m}^3$ for 24 hours. Therefore, there would not be an odour based effect from exposure to propionaldehyde.

6.0 FURTHER RISK EVALUATION OF SELECTED SoPCs

This section elaborates on the potential risk as determined from a screening of receptors and SoPCs in Section 5.0. Additional information on the individual compounds and their toxicity is presented in this section. Of the numerous compounds assessed for their potential to be harmful to the health of local residents only four were carried forward for detailed assessment:

- HCl
- NO_x
- SO₂
- H₂S

This section deals with each of these compounds in turn.

6.1 HYDROCHLORIC ACID RISK CHARACTERIZATION

Hydrogen chloride exceed the OMOE generic guidelines for 24 hour exposure and using scaling factor at the 1 hour exposure level for all three scenarios. Therefore, further examination of the toxicity and potential health impacts of exposure to HCl is presented.

The US EPA inhalation Reference Concentration (RfC) for HCl is based on the results of two chronic (lifetime) studies of rats. These studies developed a lowest observed adverse effect level (LOAEL) of 15,000 µg/m³. Adjusting to a human equivalent, a LOAEL(HEC) of 6,100 µg/m³ was estimated by the US EPA. This LOAEL(HEC) was then adjusted by an uncertainty factor of 300 (10 for the use of a LOAEL; 10 for intraspecies extrapolation; 3 for interspecies differences) to arrive at a RfC of 20 µg/m³. This concentration should be applied to an annual exposure and not necessarily to a 24 hour exposure, such as used by the OMOE.

The California EPA Office of Environmental Health Hazard has published a reference level for acute exposure to HCl in the air of 2,100 µg/m³, which should be compared to the modeled 1 hour concentrations of HCl in the air shed (Cal EPA, 1999). This acute toxicity reference value is based on a study by Stevens *et. al.* (1992), where a No Observed Adverse Effect Level (NOAEL) of 2,700 µg/m³ was reported for humans exposed to HCl over a 45 minute period.

This study involved a sensitive human sub-population group and thus no uncertainty factors were used, but the value was time adjusted to a one hour duration giving the proposed reference exposure level (REL) of 2,100 µg/m³.

The study that forms the basis of the CalEPA REL is for human populations, thus eliminating the need for the uncertainty factor of 3 applied by the US EPA for interspecies extrapolation. Additionally, the study uses a NOAEL rather than a LOAEL, thus eliminating the need for the uncertainty factor of 10 applied for the use of a LOAEL. The fact that a sensitive human sub-population is the basis of the CalEPA value, significantly reduces the need for an uncertainty factor to account for intraspecies variability.

In addition, the CalEPA has published a chronic toxicity REL for HCl of $9 \mu\text{g}/\text{m}^3$ (CalEPA, 2003). This REL is based on the same study as the US EPA; however, with a different interpretation of the toxicological results of the rat study.

Table 6-1 shows the CAL EPA REL value and US EPA RfC for HCl and all receptors where the concentration of HCl exceeded the hourly MOE Guideline. In all cases comparison of the CAL EPA derived REL to the impacted receptors reveals that none of the receptors exceed this concentration in modeled ambient air.

The OMOE has adopted the US EPA RfC as a 24 hour guideline rather than a annual or chronic level. Given the nature of the toxicity study that the guideline is based on it would be more appropriate to compare the toxicity reference value of $20 \mu\text{g}/\text{m}^3$ to the annual modeled concentration of HCl and the acute REL as

set by the CalEPA to the hourly concentrations to establish whether or not health effects could be expected as a result of exposure to HCl in the Crofton airshed.

Since the annual average concentrations (see Table 6-1) and the hourly concentrations are below the US EPA chronic RfC and CalEPA REL, no chronic or acute health effects to the local population are expected. In addition, Table 6-1 also shows the 99th and 95th percentile concentration of HCl at each of the receptors. All HCl concentrations did not exceed the toxicological standards derived by either US EPA or CalEPA.

Therefore, it is very unlikely that HCl is posing a potential health threat to local residents.

Table 6-1 HCl Air Quality Exceedances by Area Compared to Toxicity Reference Values

Receptor	Maximum ($\mu\text{g}/\text{m}^3$)			Percentiles ($\mu\text{g}/\text{m}^3$)			
	Hourly	24 Hour	Annual	99% Hourly	99% 24 Hour	95% Hourly	95% 24 Hour
US EPA RfC ($\mu\text{g}/\text{m}^3$)			20				
CAL EPA RfC ($\mu\text{g}/\text{m}^3$)	2100		9	2100		2100	
Fenceline	1084	53.1	1.81	35.6	22.3	6.83	8.46
Special Receptors							
Crofton Mobile Home Park	118	23.9	2.20	38.9	14.7	14.5	8.51
Crofton Motel	103	14.5	1.73	23.7	10.4	11.0	6.29
Crofton School/Comm. Centre	123	15.4	2.10	29.9	12.18	12.3	7.43
Crofton Church 1	65.7	14.7	2.51	31.0	12.0	15.0	8.40
Crofton Church 2	55.7	12.1	1.90	22.7	8.90	12.6	6.39
Crofton South	55.4	6.8	0.800	15.3	6.66	3.97	4.00
Crofton Substation **On-Property receptor	115.8	11.9	0.873	44.6	14.0	6.75	8.58
Max Gridded Receptor	555	30.6	3.24	5.61	1.98	1.46	1.18

6.2 NO_x ADJUSTMENT TO NO₂ RISK CHARACTERIZATION

The only receptor where NO_x exceed the Health Based Guidelines was at the fence line. Concentrations of NO_x were below the Health Based Guidelines at all special receptors and the maximum gridded receptor. The NO_x Health Based Guidelines are actually based on exposure to NO₂ and not NO.

Generally for combustion, NO₂ constitutes 5 to 10% of the initial total emissions of NO_x with the remaining 90-95% being NO. The conversion of the majority of NO occurs after emission to the atmosphere. The rate of conversion depends on the oxidizing potential of the atmosphere at the time of release. For example, if the ambient concentration of O₃ is high at the time of release, the conversion might be expected to be higher than if the ambient concentration of O₃ was low. Different methods are acceptable to regulatory authorities in the assessment of NO_x effects. The most conservative assumption to address NO to NO₂ conversion is to assume that 100% of the NO emitted is immediately converted to NO₂. Another very widely used assumption to account for the conversion of NO to NO₂ is to apply a factor of 0.75 to dispersion modelling predictions of the ground level concentrations of NO_x as NO₂ (US EPA, 1996). This implies a conversion of 75% and the predicted values are therefore reduced by 25%.

These concepts are described further by the US EPA as Tier 1 and Tier 2 approaches to the NO to NO₂ conversion. The United States Environmental Protection Agency (US EPA) Tier 1 assumption is that all NO is converted immediately after emission to NO₂ and if the value predicted from dispersion modelling exceeds the ambient criteria then proceed to Tier 2 (US EPA, 1996). The US EPA Tier 2 assumption is an empirically derived NO₂ / NO_x value of 0.75 (US EPA, 1996) meaning that the

predicted value from dispersion modelling for NO_x is multiplied by 0.75 to estimate the value for NO₂. The US EPA Tier 3 approach is the use of the Ozone Limiting Method. The Ozone Limiting Method (OLM) is usually more representative than the simple assumption that all NO is converted to NO₂. The OLM assumes that some NO₂ is emitted directly from the stack and that additional NO₂ is formed in the atmosphere by the direct mole for mole oxidation of NO by O₃ in the presence of organic radicals and sunlight.

If ozone is plentiful, and given enough time, all of the NO is converted by oxidation to NO₂. If, as is the norm, the ozone is limiting (i.e. present at low concentrations), the conversion of NO to NO₂ will be restricted by the amount of O₃. The ozone limiting methodology was applied to the CALPUFF predictions using O₃ data measured at the nearest monitoring station (Duncan Kosilah, Sept 2000-Dec 2001). At this station, the 99th percentile hourly average concentration was 83.8 ug/m³ (0.051 ppm) and the 99th percentile 24-hour average concentration was 75.9 ug/m³ (0.35 ppm).

The maximum predicted hourly and daily average NO₂ concentrations using the Tier 1, 2 and 3 methodologies are presented in Table 6-2. As can be seen, using the more advanced Tier 3 analysis for the conversion of NO to NO₂ produces much lower maximum hourly average ground-level NO₂ predictions than the conservative Tier 1 assumption.

The WHO Health Based guidelines for NO₂ are 200 µg/m³ for 1 hour exposure based on human clinical data (WHO, 2000) and a annual concentration of 40 µg/m³ based on the need to protect the public from exposure to nitrogen dioxide (WHO, 2000).

Table 6-2 Summary of Maximum Predicted NO₂ Ground-Level Concentrations Using Different NO-NO₂ Conversion Schemes

NO-NO ₂ Conversion Method	Averaging Period	Toxicity Reference Values from WHO (mg/m ³)	Maximum Fenceline (mg/m ³)	99 th Percentile (mg/m ³)	95 th Percentile (mg/m ³)
Tier 1 – 100% Conversion	Hourly	200	1494.60	193.27	117.0
	24 Hour		73.90	71.3	36.9
Tier 2 – 75% Conversion	Hourly	200	1121	145	88.0
	24 Hour		55.4	53.5	29.4
Tier 3 – Ozone Limiting Method	Hourly	200	245.0	115.0	107.0
	24 Hour		73.2	71.3	36.9

To allow for a more accurate representation of NO₂ concentrations in the Crofton area Jacques Whitford approached the Ministry of WLAP regarding the application of a Tier 3 approach. The Ministry approved of the proposed approach (described above) but specified that the Tier 1 approach results be presented as well (Warren McCormick, MWLAP, e-mail communication, 09/01/04 4:41 pm). If the NO₂ WHO toxicity values are applied to the Tier 3 results, only the maximum concentration would expect to result in potential health effect expected from exposure to NO₂. However, the 99th and 95th percentile concentrations are below the toxicologically based guideline.

6.3 SO₂ RISK CHARACTERIZATION

The SO₂ concentrations at the fenceline, as well as at the maximum off-site gridded receptor, exceeded the OMOE AAQC for acute and sub-chronic health effects and vegetative effects. An exceedance of an AAQC does not imply that an effect occurs, rather that it can not be ruled out entirely at this point.

Table 6-3 presents concentrations of SO₂ and corresponding WHO toxicity values used for the derivation of their guidelines (WHO, 2000). Although the 99th percentile concentration of SO₂ was greater than the TRV the 95th concentration was below. The annual concentrations of SO₂ were found to be below the annual toxicity reference value for the inhalation of SO₂.

Table 6-3 SO₂ Exceedances and Examination of Percentile Concentrations

Receptor	SO ₂ (mg/m ³)		
	Hourly	24 Hour	Annual
WHO toxicity based Guideline (mg/m ³)		125	50
Maximum Fenceline	1250	141	18.7
Maximum Gridded Receptor	1460	204	16.3
99 th Percentile at Max Gridded Receptor	543	159	NA
95 th Percentile at Max Gridded Receptor	345	109	NA

Given that there would be very limited time spent by any one person at the fence line, or gridded receptor located in close proximity to the fenceline, and given the conservative nature of the air model it is likely that SO₂ concentrations in the area do not pose a threat to human health on either an acute or chronic basis. However, this cannot be entirely ruled out at this point.

6.4 HYDROGEN SULPHIDE RISK CHARACTERIZATION

Hydrogen sulphide is a common issue surrounding the operation and maintenance of pulp and paper mills in Canada. H₂S is the primary component of total reduced sulphur, which is routinely monitored by NorskeCanada as part of their ongoing ambient air quality monitoring program.

As shown in Table 6-4, the air modeling exercise indicated that H₂S exceeded health based toxicity reference values at the fenceline approximately 60% of the locations modeled for hourly and 24 hour timeframes. In the case of annual modeled concentrations of H₂S, over 80% of the fenceline locations exceeded the US EPA RfC of 2.0 µg/m³. The modeled ambient concentration of H₂S at the Crofton Substation and the nearby Crofton Mobile Home Park also exceed the TRVs.

Given that there was less than a 0.2% exceedance (Table 5-2) of the H₂S TRVs measured at the Crofton Substation conservatively as TRS, Jacques Whitford believes that the air model is likely overestimating the H₂S concentrations in the near field area of the Crofton mill (Vol I).

It should also be noted that the 99th percentile of H₂S measured at the Crofton Mobile Home Park was less than either the hourly or 24 hour TRV, as was the hourly H₂S concentration at the Crofton Substation.

Therefore, it is unlikely that there is an actual acute health risk to local residents from exposure to H₂S living nearby the mill, however it can not be ruled out at this point.

Table 6-4 H₂S Exceedances in the Area Surrounding the Crofton Division

Receptor	Maximum			Percentiles			
	Hourly	24 Hour	Annual	99% Hourly	99% 24 Hour	95% Hourly	95% 24 Hour
Toxicity Reference Values (mg/m³)	100	20	2	100	20	100	20
Fenceline	212	17.3	3.74	35.1	14.5	18.2	10.7
Special Receptors							
Crofton Mobile Home Park	159	17.9	2.57	33.2	15.5	11.5	9.0
Crofton Substation **On-Property receptor	107	31.4	4.98	62.4	27.1	34.3	16.7
Max Gridded Receptor	138	46.6	9.5	115	91.8	57.4	42.4

7.0 UNCERTAINTY ANALYSIS

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

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

- Conditions where the problem is defined with a high level of certainty based on data and physical observations;
- An acceptable and reasonable level of conservatism in assumptions which will ensure that risks are not understated; and
- An appreciation of the bounds and limitations of the final solution.

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

- Available data to describe Crofton Division emissions;
- Sound conservative assumptions for certain parameters, as required; and
- Well-understood and generally accepted methods for risk prediction.

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

7.1 UNCERTAINTIES IN THE TOXICITY ASSESSMENT

7.1.1 Uncertainties in Toxicological Information

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

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

- The toxicological effect reported in animals is relevant and could occur in humans.
- The assumption that extrapolation from high-dose studies to low-dose environmental exposures adequately represents the shape of the dose-response curve in the low-dose exposure range.
- Short-term exposures used in animal studies can be extrapolated to chronic or long-term exposures in humans.

- The pharmacokinetic processes that occur in the test animals also occur in humans.

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

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

The magnitude of the uncertainty factors applied by the various regulatory agencies provides an indication of the level of confidence that should be placed on the reference value. Uncertainty factors typically range between 100 and 10,000, although some can be lower than 10. The latter values are found for a few substances where sound and substantial human toxicological information is available to enable the setting of toxicological end-point solely on the basis of human epidemiological information.

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

7.1.2 Selection of SoPCs

SoPC evaluated in this risk assessment were selected using available information from Crofton Division's NPRI data submission to Environment Canada based on 2003 emissions.

Although many of the emission estimates are based on emission factors derived using NCASI data, Jacques Whitford believes that the number of SoPCs is appropriate for a Canadian pulp and paper mill of this type.

7.1.3 Use of Surrogates

PAHs were assessed using the US EPA toxic equivalency factor approach as described in Section 5.3.3. Benzo(a)pyrene, which has the highest toxic potency was then chosen to represent the group and was compared to the sum of the individual emissions and to the estimate PAH TEQs. This approach is inherently conservative and is likely to overestimate the risks. It also permits a surrogate evaluation of those substances for which no toxicological information was available.

7.2 UNCERTAINTIES IN THE EXPOSURE ASSESSMENT

7.2.1 Estimation of Air Concentrations

SoPC concentrations in air were predicted based on the average rates of emissions of particles and gasses from sources at the Gofton mill. Annual emissions were calculated based upon site specific emissions data and NCASI emissions factors. Non-industry specific emissions factors for some sources and substances are available from the US EPA (AP-42). The use of NCASI emissions factors over AP-42 is both reasonable and preferred owing to their superior portrayal of pulp mill emissions.

Receptors were placed at the maximum concentration for their entire exposure duration (entire lifetime for carcinogenic exposures). However, other receptor locations would experience lower air concentrations and human receptors are unlikely to spend all of their time at the area where the maximum concentration occurs. These assumptions are likely to result in some overestimation of the potential risks.

7.2.2 Receptor Characteristics

For each receptor scenario, all receptors were assumed to live in the air shed (land use specific or maximum) for the entire exposure duration. For carcinogenic substances, this equates to 24 hours/day, 365 days/year for 70 years. These assumptions will combine to overestimate the potential risks.

7.3 UNCERTAINTIES IN THE RISK CHARACTERIZATION

7.3.1 Substance Interactions

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

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

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

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

In this risk assessment, the SoPC-specific toxicities have not been summed within each exposure scenario.

7.3.2 Sensitive Populations

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

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

7.4 SUMMARY OF UNCERTAINTY ANALYSIS

Jacques Whitford believes that the risk assessment did not underestimate potential risks to human receptors, given the conservative nature of the approach undertaken.

8.0 SUMMARY AND RECOMMENDATIONS

Jacques Whitford Limited (Jacques Whitford) was retained by NorskeCanada Crofton Division to conduct a Human Health Risk Assessment (HHRA) relating to its aerial emissions from its Crofton Division operations in Crofton, British Columbia.

Emissions modeling was completed using the CALPUFF model to predict ground level air concentrations and deposition rates across a 44 km by 54 km grid around Crofton Division.

Modelling results indicated that the maximum point of impingement (MPOI) for Crofton Division were primarily located on the mill property (Vol I).

Both ambient air quality data collected at three monitoring stations and modeled concentrations of SoPCs in ambient air were undertaken.

For each scenario, potential risks were predicted for a resident of the air shed that would live in the area from birth to death at the age of 75 years. It was assumed that they would be breathing the air 24 hours a day, 7 days a week, 365 days per year.

8.1 SUMMARY OF RESULTS

Results of the risk assessment are summarized below:

- Concentrations of PM₁₀ and TRS routinely monitored at the three monitoring stations located on a transect from north to south of Crofton Division indicated that no chronic or acute adverse health effects are expected as a result of exposure to measured concentrations.

- Of the numerous substances modeled the majority had concentrations that were either below OMOE and/or WHO health based guidelines or toxicity reference values applicable to individual substances.
- HCl, NO_x, and SO₂ had concentrations at some receptors that exceeded health based guidelines. Further examination of each of these compounds revealed that it is unlikely that exposure to any of these substances in the local air shed would result in adverse health effects. However, at this point it can not be entirely ruled out for SO₂.
- Modelled concentrations of H₂S near field to the Crofton Division exceeded health based toxicity reference values. Further examination of the areas of exceedance indicates that the modeled is likely overestimating H₂S concentrations (e.g. Crofton Substation). However, adverse acute and chronic health effects from exposure to H₂S for residence living close to the mill could not be ruled out at this point.

Results of the risk assessment study were in agreement with a 1990s study conducted by John Hopkins University (Matanoski *et. al.*, 1995) that indicated workers in the pulp and paper industry, who are exposed to higher concentrations of substances than general population, do not have significantly higher rates of mortality from all causes or from any specific cause of death compared to the general United States population.

8.2 RECOMMENDATIONS

In light of the findings of the air quality modelling and human health risk assessment, it is recommended that:

1. To validate the findings respecting concentrations of H₂S, NO_x and SO₂ at locations on and just outside the Crofton fenceline, ambient air quality monitoring for these parameters should be conducted for a minimum of one year at suitable locations near the fenceline.
2. If this monitoring determines that modeled concentrations in the near-field are not conservative then a similar monitoring program for HCl should be conducted at suitable locations.
3. Through initial comparison of air modelling results for H₂S and ambient measured concentrations of TRS, Jacques Whitford believes that the current position of the ambient air monitoring stations are in suitable locations. However, given the extensive air modelling exercise undertaken the ambient air quality and meteorological monitoring program associated with the Crofton mill should be reviewed with respect to the substances measured, frequency and method collection.

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APPENDIX A

Ontario Ministry of the Environment

**Summary of Point of Impingement Standards, Point of Impingement Guidelines
and Ambient Air Quality Criteria (AAQCs).**

**SUMMARY of
POINT OF IMPINGEMENT STANDARDS, POINT OF IMPINGEMENT GUIDELINES, and
AMBIENT AIR QUALITY CRITERIA (AAQCs)**

**STANDARDS DEVELOPMENT BRANCH
ONTARIO MINISTRY OF THE ENVIRONMENT**

September 2001

INTRODUCTION:

In Ontario, the enabling legislation for the Point of Impingement Standards is Regulation 346 (formerly Regulation 308) of the *Environmental Protection Act*. Regulation 346 should be consulted for application of the Point of Impingement Standards. Desirable Ambient Air Quality Criteria are defined in Regulation 337 (formerly Regulation 296) under the *Environmental Protection Act*. This document contains three tables:

- Table 1 - Sample Calculation for Toxicity Equivalent Values for Chlorinated Dioxin and Furan compounds
- Table 2 - Point of Impingement (POI) Limits and Ambient Air Quality Criteria (AAQC)
- Table 3 - Future Effects-based POI limits with current interim values subject to Risk Management Framework for Air Standards (currently under development)

NOTES TO TABLE USERS:

- 1) When an entry in the 'Status' column is given as 'CARC' (ie. CARCINOGEN), it is implied that there is no assigned standard or guideline. Emissions to the environment are to be prevented or limited to the greatest extent possible.
- 2) In the 'Status' column (ie. Table 2) when entries include the "#" symbol then the status of the Standard/Guideline is interim, pending the development of a Risk Management (RM) Framework for Air Standards that will address implementation issues such as time, technology and/or economics. Table 3 provides the future effects-based POI limits which will replace the current standard/guideline with interim status, subject to the RM framework.
- 3) In the 'AAQC Limiting Effect' column (ie. Table 2) when entries are separated by a semi-colon (eg. odour;health;odour for the contaminant butanol, n-) then these apply consecutively to the numbers in that row (ie. 770, 15000 and 3100 respectively); entries separated by 'and' generally apply to a single number which protects against both effects listed.
- 4) There are several regulations pertaining to ozone depleting substances. Ozone depleting substances are those substances governed by Part VI of the Environmental Protection Act (EPA) (1992) and regulations under the Act (ie. Regulations 851/93; Regulation 189/94). The chlorofluorocarbons (CFCs) in Part VI of the EPA are referenced in the list of AAQCs as "Part VI EPA" and are included for information purposes. The refrigerant regulation (Regulation 189/94) deals with all CFCs, HCFCs, and HFCs.
- 5) **Calculation of TEQ (Toxicity Equivalent)**

International toxicity equivalency factors (I-TEFs) are applied to 17 dioxin and furan isomers of concern to convert them into 2,3,7,8-TCDD (tetrachlorodibenzo-p-dioxin) toxicity equivalents. The conversion involves multiplying the concentration of the isomer by the appropriate I-TEF to yield the TEQ for this isomer. Summing the individual TEQ values for each of the isomers of concern provides the total toxicity equivalent level for the sample mixture.

A table listing the 17 isomers of concern and their I-TEFs can be found in the MOEE publication titled: Environment Information - Dioxins & Furans; PIBS 681b, revised 08/91 or in the example provided in Table 1.

Table 1 - Sample Calculation for Toxicity Equivalent Values for Chlorinated Dioxin and Furan compounds

Dioxin/Furan Isomers of Concern	International Toxicity Equivalency Factors (I-TEFs)	Concentration pg/m ³ (Analytically measured)	Toxicity Equivalent (TEQ) pg TEQ/m ³
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1	0.01	0.01
1,2,3,7,8-Pentachlorodibenzo-p-dioxin	0.5	0.011	0.0055
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	0.1	0.006	0.0006
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	0.1	0.01	0.001
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	0.1	0.019	0.0019
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	0.01	0.15	0.0015
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	0.001	-	-
2,3,7,8-Tetrachlorodibenzofuran	0.1	0.11	0.011
2,3,4,7,8-Pentachlorodibenzofuran	0.5	0.033	0.0165
1,2,3,7,8-Pentachlorodibenzofuran	0.05	0.024	0.0012
1,2,3,4,7,8-Hexachlorodibenzofuran	0.1	0.03	0.003
1,2,3,6,7,8-Hexachlorodibenzofuran	0.1	0.016	0.0016
1,2,3,7,8,9-Hexachlorodibenzofuran	0.1	0.016	0.0016
2,3,4,6,7,8-Hexachlorodibenzofuran	0.1	0.007	0.0007
1,2,3,4,6,7,8-Heptachlorodibenzofuran	0.01	0.047	0.00047
1,2,3,4,7,8,9-Heptachlorodibenzofuran	0.01	0.008	0.00008
1,2,3,4,6,7,8,9-Octachlorodibenzofuran	0.001	-	-
TOTAL TOXICITY EQUIVALENT			0.05665*

* Sum of toxicity equivalents of individual isomers.

The I-TEF scheme is intended to be used with isomer specific analytical results.

Table 2 - Point of Impingement (POI) Limits and Ambient Air Quality Criteria (AAQC)

Contaminant Name	Contaminant Code or CAS No.	Point of Impingement (POI) Limit			Ambient Air Quality Criteria (AAQC)				
		Half-hour POI Limit ($\mu\text{g}/\text{m}^3$)	POI Limiting Effect	Status	Annual ($\mu\text{g}/\text{m}^3$)	24-Hour ($\mu\text{g}/\text{m}^3$)	1-Hour ($\mu\text{g}/\text{m}^3$)	10-Minute ($\mu\text{g}/\text{m}^3$)	AAQC Limiting Effect
Acetaldehyde	75-07-0	500	Health	G		500			Health
Acetic acid	64-19-7	2500	Odour	S		2500			Odour
Acetone	67-64-1	48000	Odour	S		48000			Odour
Acetophenone	98-86-2	625	Odour	G			1167	850	Health and Odour
Acetylene	74-86-2	56000	Odour	S		56000			Odour
Acrolein	107-02-8	28	Health	G			23.3		Health
Acrylamide	79-06-1	45	Health	S		15			Health
Acrylonitrile	107-13-1	180	Interim #	S#	0.12	0.6			Health
Adipic acid	124-04-9	3500	Health	G		1167			Health
Alkyltoluene sulphonamide, N-	N/A	100		G		120			Particulate
Allyl glycidyl ether	106-92-3	180	Health	G		60			Health
Aluminum distearate	300-92-5	100	Particulate	G		2180			Health
Aluminum oxide	1344-28-1	100	Particulate	G		120			Particulate
Aluminum stearate	7047-84-9	100	Particulate	G		2180			Health
Aluminum tristearate	637-12-7	100	Particulate	G		2180			Health
Ammonia	7664-41-7	3600	Odour #	S#		100			Health
Ammonium chloride	12125-02-9	100	Particulate	G		120			Particulate
Amyl acetate, iso-	123-92-2					53200			Health and Odour
Amyl acetate, n-	628-63-7					53200			Health and Odour
Amyl acetate, secondary	626-38-0					66500			Health and Odour
Antimony and compounds	7440-36-0	75	Health	S		25			Health
Arsenic and compounds	7440-38-2	1	Health	G		0.3			(A) Health
Arsine	7784-42-1	10	Health	S		5			Health
Asbestos (fibres > 5 μm in length)	1332-21-4					0.04 fibres/cm ³			Health
Asbestos (total)	1332-21-4	5	Health	G					
Barium - total water soluble	7440-39-3	30	Health	G		10			Health
Benzene	71-43-2			CARC					Health
Benzo(a)pyrene - single source	50-32-8	0.0033	Health	G	0.00022	0.0011			Health
Benzo(a)pyrene, all sources	50-32-8				0.0003				Health
Benzoic acid	68-85-0	2100	Health	G		700			Health
Benzothiazole	95-16-9	200	Health	G		70			Health
Benzoyl chloride	98-88-4	350	Health	G		125			Corrosion and Health
Benzyl alcohol	100-51-6	2640	Health	G		880			Health
Beryllium and compounds	7440-41-7	0.03	Health	S		0.01			Health
Biphenyl	92-52-4	60	Odour	G			60		Odour
Borax	1303-96-4	100	Health	G		33			Health
Boric acid	10043-35-3	100	Health	G		33			Health
Boron	7440-42-8	100	Particulate	S		120			Particulate
Boron tribromide	10294-33-4	100	Corrosion	S		35			Corrosion
Boron trichloride	10294-34-5	100	Corrosion	S		35			Corrosion
Boron trifluoride	7637-07-2	5		S		2			Vegetation
Bromacil	314-40-9	30	Health	G		10			Health
Bromine	7726-95-6	70	Health	S		20			Health
Bromochlorodifluoromethane (Halon 1211)	N/A	see	"Part VI/EPA"						Ozone depleting

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Bromoform	75-25-2	165	Health	G		55			Health
Bromotrifluoromethane (Halon 1301)	75-63-8	see	"Part VI/EPA"						Ozone depleting
Butanol, iso-	78-83-1	1940	Odour	G		655	15000	2640	Odour; Health; Odour
Butanol, n-	71-36-3	2278	Odour	G		770	15000	3100	Odour; Health; Odour
Butanol, tertiary	75-65-0			UD		30300			Health
Butoxy-2-propanol, 1-	5131-66-8	9900	Health	G		3300			Health
Butyl acetate, n-	123-86-4	735	Odour	G		248	15000	1000	Odour; Health; Odour
Butyl acrylate	141-32-2	100	Particulate	G		120			Particulate
Butyl benzene sulphonamide, N-	3622-84-2	105	Health	G		35			Health
Butyl benzyl phthalate	85-68-7	450	Health	G		150			Health
Butyl stearate	123-95-5	100	Particulate	G		120			Particulate
Cadmium and compounds	7440-43-9	5	Health	S		2			(A) Health
Calcium carbide	75-20-7	20	Corrosion	G		10			Corrosion
Calcium cyanide (as total salt)	592-01-8	100	Particulate	G		120			Particulate
Calcium hydroxide	1305-62-0	27	Corrosion	S		13.5			Corrosion
Calcium oxide	1305-78-8	20	Corrosion	S		10			Corrosion
Calcium stearate	1592-23-0	100	Particulate	G		35			Health
Captan	133-06-2	75	Health	G		25			Health
Carbon black	1333-86-4	25	Soiling	S		10			Soiling
Carbon disulphide	75-15-0	330	Odour	S		330			Odour
Carbon monoxide ¹	630-08-0	6000	Health	S		15700 (8 hr average)	36200		(A) see note below
Carbon tetrachloride	56-23-5	7.2	Health	G		2.4			Health
Chloramben	133-90-4	100	Particulate	G		120			Particulate
Chlordane	57-74-9	15	Health	G		5			Health
Chlorinated dibenzo-p-dioxins (CDDs) (See Table 1)	N/A	15 pgTEQ/m ³	Health	G		5			Health
Chlorine	7782-50-5	300	Interim #	S#		10		230	Health; Odour
Chlorine dioxide	10049-04-4	85	Health	S		30			Health
Chlorodifluoromethane (Freon 22)	75-45-6	1050000	Health	G		350000			Health
Chloroform	67-66-3	300	Interim #	S#	0.2	1			Health
Chloropentafluoroethane (CFC-115)	76-15-3	see	"Part VI/EPA"						Ozone depleting
Chromium -di-, tri- and hexavalent forms	7440-47-3	5	Health	G		1.5			Health
Citric acid	77-92-9	100	Particulate	G		120	300		Health and Particulate
Coal tar pitch volatiles - soluble fraction	8007-45-2	3	Health	G	0.2	1			Health
Cobalt	7440-48-4	0.3	Health	G		0.1			Health
Copper	7440-50-8	100	Health	S		50			Health
Cresols	1319-77-3	230	Health	S		75			Health
Cyanogen chloride	506-77-4	15	Health	G		12			Health
Cyclohexane	110-82-7	300000	Health	G		100000			Health
Dalapon sodium salt	127-20-8	100	Health	G		50			Health
Decaborane	17702-41-9	50	Health	S		25			Health
Decane, n	124-18-5			UD			60000		Health and Odour
Decene, 1-	872-05-9	180000	Health	G		60000			Health
Detergent enzyme (Subtilisin)	1395-21-7	0.2	Health	G		0.06			Health
Diacetone alcohol	123-42-2	990	Odour	G		335		1350	Odour
Diazinon	333-41-5	9	Health	G		3			Health

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Diborane	19287-45-7	20	Health	S		10			Health
Dibromotetrafluoroethane (Halon 2402)	124-73-2	see	"Part VI/EPA"						Ozone depleting
Dibutyl amine	111-92-2			UD			2645		Health
Dibutyl phthalate (DBP, di-n-butyl phthalate)	84-74-2	100	Health	G		50			Health
Dibutyltin dilaurate	77-58-7	100	Health	G		30			Health
Dicapryl phthalate	131-15-7	100		S		120			Particulate
Dichloro-1,1,2,2, - tetrafluoroethane, 1,2 (Freon 114)	76-14-2	2100000	Health	G		700000	see "Part VI/EPA"		Health
Dichlorobenzene, ortho-	95-50-1	37000	Health	G			30500		Health
Dichlorobenzene, para-	106-46-7	285	Health	G		95			Health
Dichlorobenzidine, 3,3-	91-94-1			CARC					Health
Dichloroethane, 1,1-	75-34-3	600	Health	G		200			Health
Dichloroethylene, cis-1,2-	156-59-2	315	Health	G		105			Health
Dichloroethylene, sym-1,2-	540-59-0	315	Health	G		105			Health
Dichloroethylene, trans-1,2-	156-60-5	315	Health	G		105			Health
Diethyl amine	109-89-7			UD			2910		Health
Diethyl phthalate (DEP)	84-66-2	100	Health	G		125			Health
Diethylene glycol monobutyl ether	112-34-5					65			Health
Diethylene glycol monobutyl ether acetate	124-17-4					85			Health
Diethylene glycol monoethyl ether	111-90-0	800	Odour	G		273		1100	Odour
Diethylene glycol monoethyl ether acetate	112-15-2					1800			Health
Diethylene glycol monomethyl ether	111-77-3	800	Odour	G		1200			Health
Diethylhexyl phthalate (DEHP)	117-81-7	100	Health	G		50			Health
Difluorodichloromethane (Freon 12)	75-71-8	1500000	Health	G		500000	see "Part VI/EPA"		Health
Dihexyl phthalate (DHP)	84-75-3	100	Health	G		50			Health
Diisobutyl ketone	108-83-8	470	Odour	G		3500		649	Health; Odour
Dimethyl acetamide, N,N-	127-19-5	900	Health	G		300			Health
Dimethyl amine	124-40-3			UD			1840		Health and Odour
Dimethyl disulphide	624-92-0	40	Odour	S			40		Odour
Dimethyl ether	115-10-6	2100	Odour	G		2100			Odour
Dimethyl methylphosphonate	756-79-6					875			Health
Dimethyl phthalate (DMP)	131-11-3	100	Health	G		125			Health
Dimethyl sulfoxide	67-68-5	6300	Health	G		2100			Health
Dimethyl sulphide	75-18-3	30	Odour	S			30		Odour
Dimethyl-1,3-diamino propane, N,N-	109-55-7	60	Health	G		20			Health
Diocetyl phthalate	117-84-0	100	Particulate	S		120			Particulate
Dioxane	123-91-1			UD		3500			Health
Dioxolane-1,3	646-06-0	30	Health	G		10			Health
Diphenylamine	122-39-4	50	Health	G		17.5			Health
Diquat dibromide -respirable	85-00-7	0.096	Health	G		0.032			Health
Diquat dibromide -total in ambient air	85-00-7	0.48	Health	G		0.16			Health
Dodecyl benzene sulphononic acid	1886-81-3	100	Particulate	G		120			Particulate
Dodine	2439-10-3	30	Health	G		10			Health
Droperidol	548-73-2	3	Health	G		1			Health

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Dustfall	N/A	8000 ($\mu\text{g}/\text{m}^2$)	Soiling	S	4.6 g/m^2 + (annual)	7 g/m^2 (30 day)			(A) Soiling
Ethanol (Ethyl alcohol)	64-17-5	19000	Odour	G			19000		Odour
Ethyl acetate	141-78-6	19000	Odour	S			19000		Odour
Ethyl acrylate	140-88-5	4.5	Odour	S			4.5		Odour
Ethyl benzene	100-41-4	3000	Health [#]	S [#]		1000		1900	Health; Odour
Ethyl ether	60-29-7	7000	Interim [#]	S [#]		8000		950	Health; Odour
Ethyl hexanol, 2-	104-76-7	600	Odour	G			600		Odour
Ethyl-3-ethoxy propionate	763-69-9	147	Odour	G		50		200	Odour
Ethylanthraquinone, 2-	84-51-5	30	Health	G		10			Health
Ethylene	74-85-1			UD		40			Vegetation
Ethylene dibromide	106-93-4	9	Health	G		3			Health
Ethylene dichloride	107-06-2	6	Health	G	0.4	2			Health
Ethylene glycol	107-21-1					12700			Health
Ethylene glycol butyl ether (Butyl cellosolve)	111-76-2	350	Odour	G		2400		500	Health;Odour
Ethylene glycol butyl ether acetate (But.cell.ace)	112-07-2	500	Odour	G		3250		700	Health;Odour
Ethylene glycol dinitrate	628-96-6	10	Health	G		3			Health
Ethylene glycol ethyl ether (Cellosolve)	110-80-5	800	Odour	G		380		1100	Health;Odour
Ethylene glycol ethyl ether acetate (Cell.ace.)	111-15-9	220	Odour	G		540		300	Health;Odour
Ethylene glycol monoethyl ether	112-25-4					2500			Health
Ethylene oxide	75-21-8	15	Health	G		5			Health
Ethylenediaminetetra acetic acid	60-00-4	100		G		120			Particulate
Fentanyl citrate	990-73-8	0.06	Health	G		0.02			Health
Ferric oxide	1309-37-1	75	Soiling	S		25			Soiling
Fluoridation -as total fluorides, total GS	7664-39-3					40 $\mu\text{g}/100$ cm^2 /30 day			(A) Vegetation
Fluoridation -as total fluorides, total NGS	7664-39-3					80 $\mu\text{g}/100$ cm^2 /30 day			(A) Vegetation
Fluorides (as HF) - gaseous -growing season GS	7664-39-3					0.34 $\mu\text{g}/\text{m}^3$ /30 day			(A) Vegetation
Fluorides (as HF) - gaseous -growing season GS	7664-39-3	4.3	Vegetation	S		0.86			(A) Vegetation
Fluorides (as HF) - total, growing season GS	7664-39-3	8.6	Vegetation	S		1.72			(A) Vegetation
Fluorides (as HF) - total, growing season GS	7664-39-3					0.69 $\mu\text{g}/\text{m}^3/30$ day			(A) Vegetation
Fluorides (as HF)- total, non growing season NGS	7664-39-3	17.2	Vegetation	S		3.44			(A) Vegetation
Fluorides (as HF)- total non-growing season NGS	7664-39-3					1.38 $\mu\text{g}/\text{m}^3/30$ day			(A) Vegetation

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Fluorides in dry forage-dry weight	7664-39-3					35 ppm/30 day ave.*			(A) Effects on animals
						80 ppm/30 day ave.**			(A) Effects on animals
						60 ppm/60 day ave.***			(A) Effects on animals
Fluorinert 3M-FC-70	N/A	100	Particulate	G		120			Particulate
Formaldehyde	50-00-0	65	Odour	S		65			Health
Formic acid	64-18-6	1500	Health	S		500			Health
Furfural	98-01-1	1000	Odour	S			1000		Odour
Furfuryl alcohol	98-00-0	3000	Health	S		1000			Health
Glutaraldehyde	111-30-8	42	Health	G		14	35		Health
Haloperidol	52-86-8	0.3	Health	G		0.1			Health
n-Heptane	142-82-5	33000	Health	S		11000			Health
Hexachlorocyclopentadiene	77-47-4	6	Health	G		2			Health
Hexamethyl disilazane	999-97-3	5	Health	G		2			Health
Hexamethylene diisocyanate monomer	822-06-0	1.5	Health	G		0.5			Health
Hexamethylene diisocyanate trimer	4035-89-6	3	Health	G		1			Health
Hexamethylenediamine	124-09-4	48	Health	G		16			Health
Hexamethyleneimine	111-49-9	945	Health	G		315			Health
Hexane	110-54-3	35000	Health	G		12000			Health
Hexylene glycol	107-41-5	14400	Health	G			12000		Health
Hydrogen bromide	10035-10-6	800	Health	G			668		Health
Hydrogen chloride	7647-01-0	100	Corrosion [#]	S [#]		20			Health
Hydrogen cyanide	74-90-8	1150	Health	S		575			Health
Hydrogen peroxide	7722-84-1	90	Health	G		30			Health
Hydrogen sulphide	7783-06-4	30	Odour	S			30		(A) Odour
Iron - metallic	15438-31-0	10	Soiling	S		4			Soiling
Isobutyl acetate	110-19-0	1220	Odour	G		412		1660	Odour; Odour
Isopropyl ether	108-20-3	220	Odour	G		110000			Health
Isopropyl acetate	108-21-4	1470	Odour	G		500		2000	Odour; Odour
Isopropyl benzene	98-82-8	100	Odour	S		400			Health
Lead	7439-92-1	6	Health	S		2			(A) Health
						0.7			(A) Health
						$\mu\text{g}/\text{m}^3/30$			
						day +			
Lead - in dustfall	7439-92-1					0.1 g/m ² /30 day			Health
Lindane (Hexachlorocyclohexane)	58-89-9	15	Health	G		5			Health
Lithium -other than hydrides	7439-93-2	60	Health	S		20			Health
Lithium hydrides	7580-67-8	7.5	Health	S		2.5			Health
Magnesium oxide	1309-48-4	100	Particulate	S		120			Particulate
Magnesium stearate	557-04-0	100	Particulate	G		35			Health
Malathion	121-75-5	100		G		120			Particulate
Maleic anhydride	108-31-6	100	Health	G		30			Health
Manganese compounds (including permanganates)	7439-96-5	7.5	Health	G		2.5			Health

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Mercaptans (as Methyl mercaptan) -total	74-93-1	20	Odour	S			20		(A) Odour
Mercaptobenzothiazole disulphide	120-78-5	100	Particulate	G		120			Particulate
Mercury	7439-97-6	5	Health	S		2			(A) Health
Mercury (as Hg) - alkyl compounds	7439-97-6	1.5	Health	S		0.5			Health
Metaldehyde (Acetaldehyde tetramer)	108-62-3	100	Particulate	G		120			Particulate
Methacrylic acid	79-41-4	2000	Odour	G		2000			Odour
Methane diphenyl diisocyanate (MDI)	101-68-8	3	Health	G		1			Health
Methanol (Methyl alcohol, Wood alcohol)	67-56-1	12000	Health	S		4000			Health
Methoxy-1-propyl acetate,2-	70657-70-4	4600	Health	G		1530			Health
Methoxychlor	72-43-5	100	Particulate	G		120			Particulate
Methyl acrylate	96-33-3	4	Odour	S			4		Odour
Methyl bromide	74-83-9	4000	Health	G		1350			Health
Methyl chloride	74-87-3	20000	Health	G		7000			Health
Methyl ethyl ketone (2-Butanone)	78-93-3	30000	Interim #	S#		1000			Health
Methyl ethyl ketone peroxide	1338-23-4	250	Health	G		80	200		Health; Health
Methyl isobutyl ketone	108-10-1	1200	Odour	S		1200			Odour
Methyl mercapto aniline	2987-53-3			UD					Odour
Methyl methacrylate	80-62-6	860	Odour	S		860			Odour
Methyl salicylate	119-36-8	300	Health	G		100			Health
Methyl styrene, alpha	98-83-9			UD			24000		Health
Methyl tert-butyl ether	1634-04-4	2200	Odour	G		7000			Health
Methyl-2-hexanone, 5-	110-12-3	460	Odour			160		630	Odour
Methyl-2-pyrrolidone, N-	872-50-4						40000		Health
Methyl-n-amyl ketone	110-43-0			UD		4600			Health
Methylal	109-87-5	18000	Health	G		6200			Health
Methylcyclopentadienyl manganese tricarbonyl (MMT)	12108-13-3	30	Health	G		10			Health
Methylene chloride	75-09-2	5300	Interim #	G#	44	220			Health;Health
Methylene dianiline	101-77-9	30	Health	G		10			Health
Methylene iodide	75-11-6	195	Health	G		65			Health
Methylene-bis-2-chloroaniline, 4,4-	101-14-4	30	Health	G		10			Health
Miconazole nitrate	22832-87-7	15	Health	G		5			Health
Milk powder	N/A	20	Soiling	S		20			Soiling and Odour
Mineral Spirits ²	N/A	7800	Health#	S#		2600			Health
Molybdenum	7439-98-7	100	Particulate	G		120			Particulate
Monochlorobenzene	108-90-7	4200	Health	G			3500	4500	Health; Odour
Monomethyl amine	74-89-5	25	Odour	S		25			Odour
Naphthalene	91-20-3	36	Odour	G		22.5		50	Health; Odour
Naphthol, alpha-	90-15-3	100	Health	G		100			Health
Nickel	7440-02-0	5	Vegetation	S		2			(A) Vegetation
Nickel carbonyl	13463-39-3	1.5	Health	S		0.5			Health
Nitric acid	7697-37-2	100	Corrosion	S		35			Corrosion
Nitrilotriacetic acid	139-13-9	100	Health	S		120			Particulate
Nitrogen oxides ³	10102-44-0	500	Health	S		200	400		(A) Health; Health
Nitroglycerin	55-63-0	10	Health	G		3			Health
Nitrosodiethylamine, N-	55-18-5			CARC					Health

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Nitrosodimethylamine, N-	62-75-9			CARC					Health
Nitrous oxide	10024-97-2	27000	Health	G		9000			Health
Octane	111-65-9	45400	Odour	G		15300		61800	Odour; Odour
Octene, 1-	25377-83-7	150000	Health	G		50000			Health
Oleic acid	112-80-1	6	Health	G			5		Health
Oxalic acid	144-62-7	75	Health	G		25			Health
Oxo-heptyl acetate	90438-79-2	255	Health	G		85			Health
Oxo-hexyl acetate	88230-35-7	255	Health	G		85			Health
Ozone	10028-15-6	200	Health	S			165		(A) Health and Vegetation
Palladium - water soluble compounds	7657-10-1	30	Health	G		10			Health
Paraquat dichloride - respirable	1910-42-5	0.009	Health	G		0.003			Health
Paraquat dichloride - total in ambient air	1910-42-5	0.045	Health	G		0.015			Health
Penicillin	1406-05-9	0.3	Health	G		0.1			Health
Pentaborane	19624-22-7	3	Health	S		1			Health
Pentachlorophenol	87-86-5	60	Health	G		20			Health
Perchloroethylene	127-18-4	10000	Interim #	G#		360			Health
Phenol	108-95-2	100	Health	S		100			Health
Phosgene	75-44-5	130	Health	S		45			Health
Phosphine	7803-51-2	30	Health	G		10			Health
Phosphoric acid (as P2O5)	7664-38-2	100	Particulate	S		120			Particulate
Phosphorus oxychloride	10025-87-3	40	Health	G		12			Health
Phosphorus pentachloride	10026-13-8	30	Health	G		10			Health
Phthalic anhydride	85-44-9	100	Particulate	S		120			Particulate
Pimozide	2062-78-4	3	Health	G		1			Health
Platinum - water soluble compounds	7440-06-4	0.6	Health	G		0.2			Health
Polybutene -1-sulphone	N/A	100	Particulate	G		120			Particulate
Polychlorinated biphenyls (PCBs)	1336-36-3	0.45	Health	G	0.035	0.15			Health
Polychloroprene	25267-15-6	100		G		500			Particulate
Potassium cyanide	151-50-8	100		G		120			Particulate
Potassium hydroxide	1310-58-3	28	Corrosion	G		14			Corrosion
Potassium nitrate	7757-79-1	100		G		120			Particulate
Propanol, iso- (Isopropyl alcohol, Isopropanol)	67-63-0	24000	Odour	G		24000			Odour
Propanol, n- (Propyl alcohol)	71-23-8	48000	Health	G		16000			Health
Propionaldehyde	123-38-6	7	Odour	G		2.5		10	Odour; Odour
Propionic acid	79-09-04	100	Odour	G				100	Odour
Propionic anhydride (as Propionic acid)	123-62-6	100	Odour	G				100	Odour
Propyl acetate, n-	109-60-4	900	Odour	G		6600			Health
Propylene dichloride	78-87-5	2400	Odour	S		2400			Odour
Propylene glycol	57-55-6	100	Health	G		120			Health
Propylene glycol methyl ether	107-98-2	89000	Odour	G		30000		121000	Odour; Odour
Propylene glycol monomethyl ether acetate	108-65-6	5000	Odour	G		5000			Odour
Propylene oxide	75-56-9	450	Interim #	S#	0.3	1.5			Health; Health
Pyridine	110-86-1	60	Odour	G		150		80	Health; Odour
Quinone	106-51-4	45	Health	G		15			Health
Selenium	7782-49-2	20	Health	G		10			Health

Contaminant Name	Contaminant Code or CAS No.	Point of Impingement (POI) Limit			Ambient Air Quality Criteria (AAQC)				
		Half-hour POI Limit ($\mu\text{g}/\text{m}^3$)	POI Limiting Effect	Status	Annual ($\mu\text{g}/\text{m}^3$)	24-Hour ($\mu\text{g}/\text{m}^3$)	1-Hour ($\mu\text{g}/\text{m}^3$)	10-Minute ($\mu\text{g}/\text{m}^3$)	AAQC Limiting Effect
Silane	7803-62-5	450	Health	G		150			Health
Silica -respirable (<10 um diameter), cristabolite	14464-46-1	15	Health	G		5			Health
Silica -respirable (<10 um diameter), quartz	14808-60-7	15	Health	G		5			Health
Silica -respirable (<10 um diameter), tridymite	15468-32-3	15	Health	G		5			Health
Silver	7440-22-4	3	Health	S		1			Health
Sodium bisulphite	7631-90-5	100	Particulate	G		120			Particulate; Health
Sodium chlorate	7775-09-9	18	Health	G		6			Health
Sodium chlorite	7758-19-2	60	Health	G		20			Health
Sodium cyanide	143-33-9	100	Particulate	G		120			Particulate
Sodium hydroxide	1310-73-2	20	Corrosion	G		10			Corrosion
Sodium nitrate	7631-99-4	100	Particulate	G		7000			Health
Stannous Chloride (as Sn)	7772-99-8	30	Health	G		10			Health
Strontium	7440-24-6	100	Particulate	G		120			Particulate
Strontium carbonate	1633-05-2	100	Particulate	G		120			Particulate
Strontium hydroxide	18480-07-4	100	Particulate	G		120			Particulate
Strontium oxide	1314-11-0	100	Particulate	G		120			Particulate
Styrene	100-42-5	400	Odour	S		400			Health
Sulfamic acid	5329-14-6	100	Particulate	G		120			Particulate
Sulphur dioxide	7446-09-5	830	Health	S	55	275	690		(A) Health and Vegetation
Sulphur hexafluoride	2551-62-4	1800000	Health	G		600000			Health
Sulphuric acid	7664-93-9	100	Corrosion	S		35			Corrosion
Suspended particulate matter < 44 μm aero. dia.	N/A	100	Visibility	S	60++	120			(A) Visibility
Talc - fibrous	14807-96-6	5	Health	G		2			Health
Tellurium - excluding hydrogen telluride	13494-80-9	30	Health	S		10			Health
Tetrabutylurea	4559-86-8	30	Health	G		10			Health
Tetrahydrofuran	109-99-9	93000	Odour	S		93000			Odour
Tetramethyl thiuram disulphide	137-26-8	30	Health	G		10			Health
Thiourea	62-56-6	60	Health	G		20			Health
Tin	7440-31-5	30	Health	S		10			Health
Titanium	7440-32-6	100	Particulate	S		120			Particulate
Titanium dioxide	13463-67-7	100	Health	G		34			Health
Tolmetin sodium	35711-34-3	15	Health	G		5			Health
Toluene	108-88-3	2000	Odour	S		2000			Odour
Toluene diisocyanate	584-84-9	1	Health	S		0.5			Health
Total reduced sulphur (as hydrogen sulphide)	N/A	40	Odour	G			40		Odour
Tributyltin oxide	56-35-9	0.42	Health	G		0.14			Health
Trichlorobenzene, 1,2,4-	120-82-1	100		G		400			Health
Trichloroethane, 1,1,1,- (Methyl chloroform)	71-55-6	350000	Health	S		115000			Health
Trichloroethylene	79-01-6	3500	Interim #	S#	23	115			Health
Trichlorofluoromethane	75-69-4	18000	Health	G		6000	see "Part VI/EPA"		Health
Trifluoroacetic acid	76-05-1	45	Health	G		15			Health

Contaminant Name	Contaminant Code or CAS No.	Point of Impingement (POI) Limit			Ambient Air Quality Criteria (AAQC)				
		Half-hour POI Limit ($\mu\text{g}/\text{m}^3$)	POI Limiting Effect	Status	Annual ($\mu\text{g}/\text{m}^3$)	24-Hour ($\mu\text{g}/\text{m}^3$)	1-Hour ($\mu\text{g}/\text{m}^3$)	10-Minute ($\mu\text{g}/\text{m}^3$)	AAQC Limiting Effect
Trifluorotrichloroethane	76-13-1	2400000	Health	S		800000	see "Part VI/EPA"		Health
Trimethyl amine	75-50-3	0.5	Odour	G			0.5		Odour
Trimethylbenzene, 1,2,4-	95-63-6	500	Odour	G		1000			Odour and Health
Trimethylol propane	77-99-6	100	Health	G		1250			Health
Tripolytin methacrylate	N/A	3	Health	G		1			Health
Vanadium	7440-62-2	5	Health	G		2			(A) Health
Vinyl chloride	75-01-4	3	Health	G	0.2	1			Health
Vinylidene chloride (1,1-Dichloroethene)	75-35-4	30	Health	S		10			Health
Warfarin	81-81-2	30	Health	G		10			Health
Whey powder	N/A	100	Particulate	G		120			Particulate
Xylenes	1330-20-7	2300	Odour	S		2300			Odour
Zinc	7440-66-6	100		S		120			Particulate
Zinc chloride	7646-85-7	12	Health	G			10		Health
Zinc stearate	557-05-1	100	Particulate	G		35			Health

TERMS:

¹ = Carbon monoxide AAQC is for an 8-hour average based on high background levels from automobiles

² = Mineral spirits are petroleum distillate mixtures of C₇-C₁₂ hydrocarbons, with boiling points ranging from 130-220 °C and flash points ranging from 21-60 °C. Please see Rationale document: "Ontario Air Standards for Mineral Spirits" for further detail.

³ = NOx (Nitrogen Oxides) are assumed to be the sum of nitrogen dioxide and nitrogen monoxide. AAQCs are based on nitrogen dioxide.

S = Air Quality Standard, **G** = Guideline, **CARC** = Carcinogen, **UD** = Under Development, or odour threshold review.

A = AAQC Chemicals listed in Regulation 337 (formerly Regulation 296) under the Environmental Protection Act.

Part VI/EPA = "Part VI/EPA" refers to Part VI of the Ontario Environmental Protection Act R.S.O. 1990, C. E-19, which addresses the manufacture, use, storage, disposal, etc., of ozone depleting substances.

N/A = Not Available

GS = Growing Season May 1 - September 30- Northern Ontario, Mid-Ontario & N Regions

April 1 - October 31 - Southern Ontario, SW, WC, E & C Regions

NGS = Non Growing Season October 1 - April 30 - Northern Ontario, Mid Ontario & N Regions

November 1 - March 31 - Southern Ontario, SW, WC, E & C Regions.

* average monthly results for growing season.

** average results for any single month.

*** average of 2 consecutive months.

+ = arithmetic mean, ++ = geometric mean

[#] = Status of Standard/Guideline is interim, pending the outcome of the Risk Management (RM) Framework for Air Standards (currently under development). See Table 3 for list of pending future Effects-based limits.

Table 3[#] - Future Effects-based POI limits with current interim values subject to RM Framework for Air Standards (currently under development)

Contaminant Name ($\mu\text{g}/\text{m}^3$)	Contaminant CAS No.	Future Effects-based POI Limit	Limiting Effect
Acrylonitrile	107-13-1	1.8	Health
Ammonia	7664-41-7	300	Health
Chlorine	7782-50-5	30	Health
Chloroform	67-66-3	3	Health
Ethyl benzene	100-41-4	1400	Odour
Ethyl ether	60-29-7	700	Odour
Hydrogen chloride	7647-01-0	60	Health
Methyl ethyl ketone (2-Butanone)	78-93-3	3000	Health
Methylene chloride	75-09-2	660	Health
Mineral spirits	N/A	3000	Odour
Perchloroethylene	127-18-4	1080	Odour
Propylene oxide	75-56-9	4.5	Health
Trichloroethylene	79-01-6	350	Health

APPENDIX B

TOXICITY PROFILES

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Glossary and Acronyms

AAQC	Ambient Air Quality Criteria
Absolute bioavailability	Absolute bioavailability is the fraction or percentage of an administered dose that reaches systemic circulation (blood) irrespective of via the gastrointestinal tract, skin or lungs
Ah	Aryl hydrocarbon
ATSDR	Agency for Toxic Substances and Disease Registry
Bioavailability	The degree to which a substance becomes available to the target tissue after administration or exposure.
Cal-EPA	California Environmental Protection Agency
CEPA	Canadian Environmental Protection Act
COPC	Contaminants of Potential Concern
ESOD	Erythrocyte Superoxide Dismutase
FAO	Food and Agriculture Organization. An organization of the United Nations.
IARC	International Agency for Research on Cancer. An organization of the WHO.
IOC	Intake of concern
IOM	Institute of Medicine
IPCS	International Programme on Chemical Safety
IRIS	Integrated Risk Information System. A database maintained by the US EPA.
LOAEL	Lowest-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the lowest dose results in observed adverse health effects. May be used in place of a NOAEL where a NOAEL cannot be determined.

MAC	Maximum Allowable Concentration
MADEP	Massachusetts Department of Environmental Protection
MOE	Ontario Ministry of the Environment
MRL	Minimal Risk Level. A term used by the ATSDR to describe an estimate of daily human exposure to a hazardous substance that is likely to be without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure.
NATO	North Atlantic Treaty Organization
NCEA	National Center for Environmental Assessment
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No-observed-effects-level. A term that describes the benchmark on a threshold dose-response curve at which the highest dose does not result in adverse effects.
NRC	National Research Council
OEHHA	Office of Environmental Health Hazard Assessment
ORD	Office of Research and Development
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzo-p-dioxins
PCDF	Polychlorinated dibenzofurans
POI	Point of Impingement
PTWI	Provisional Tolerable Weekly Intake
RAF	Relative absorption factor
RDA	Recommended Dietary Allowance

REL	Reference Exposure Level is a NIOSH time-weighted average concentration for up to a 10-hour workday during a 40-hour work week.
Relative bioavailability	A comparative fraction which predicts bioavailability in one medium or form in relation to the medium for which the TRV was derived.
RfC	Reference Concentration. The RfC is an estimate of lifetime daily exposure to a non-carcinogen in air for the general human population that appears to be without appreciable risk of deleterious effects expressed in mg chemical/kg body weight-day.
RfD	Reference Dose. The RfD is an estimate of lifetime daily exposure to a non-carcinogen for the general human population that appears to be without appreciable risk of deleterious effects expressed in mg chemical/kg body weight-day.
SF	Slope factor. The SF is a plausible upper bound estimate of the probability of a response per unit intake of a chemical over a lifetime expressed as (mg chemical/kg body weight-day) ⁻¹ and is used to express carcinogenic effects.
STSC	Superfund Health Risk Technical Support Center
TC	Tolerable Concentration. A term used by Health Canada to describe concentrations in air that a person may be continuously exposed to over a lifetime without adverse effects. The TC is used to derive the TDI.
TC ₀₅	Tumorigenic concentration that will induce a 5% increase in the incidence of tumors or deaths due to tumors following exposure to that chemical in air.
TD	Tumorigenic Dose. A term used to describe a dose that will induce an increase in the incidence of tumors or deaths due to tumours as calculated from a non-threshold dose-response curve.
TD ₀₅	Tumorigenic Dose that will induce a 5% increase in the incidence of tumors or deaths due to tumors.
TDI	Tolerable Daily Intake. A term used by Health Canada in place of RfD.
TEF	Toxic Equivalency Factor

TEQ	Toxic Equivalent
TRV	Toxicity Reference Value
UF	Uncertainty Factor. A factor that is applied to NOAELs or LOAELs to yield a RfC or RfD. For example, the UF can be used to account for intra-species and inter-species extrapolations.
UL	Tolerable upper intake level. A term used by the IOM to describe the highest daily nutrient intake that will not result in adverse health effects.
Unit Risk	Units risks estimate the upper bound probability of an individual developing cancer following exposure to a particular level (usually as 1 µg/L in water or 1 µg/m ³) of a potential carcinogen. For example, if the unit risk is 1.2 x 10 ⁻⁶ µg/L then it is expected that 1.2 excess tumours are expected to occur per 1,000,000 people exposed to 1 µg of that chemical in 1 L of drinking water.
US EPA	United States Environmental Protection Agency
WHO	World Health Organization

1.0 INTRODUCTION

For the purpose of this assessment, toxicity reference values (TRVs) were obtained for each of the identified substances of potential concern (SoPC). Toxicological information was obtained, as necessary, from various sources including Health Canada, the US EPA Integrated Risk Information System (IRIS) database, the World Health Organization (WHO), Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Level (MRL), and the California Environmental Protection Agency (CAL EPA).

TRVs are values used to describe maximum acceptable doses of chemicals that will not result in the development of adverse health effects. TRVs can be used to describe non-carcinogenic and carcinogenic effects and can express effects in different terms based on magnitude of the dose, length of exposure and route of exposure.

1.1 Non-Carcinogenic TRVs

Non-carcinogenic chemicals exhibit threshold effects following exposure. Threshold effects are defined by the observation of adverse effects at a given dose or concentration. Given these threshold effects, two measures of interest can describe the dose-response curve: the no-adverse-effects-level (NOAEL) and lowest-adverse-effects-level (LOAEL). The NOAEL is the benchmark at which the highest dose does not result in observed adverse effects. The LOAEL may be used when a NOAEL is not available and is the lowest dose at which adverse effects are observed.

The reference concentration (RfC) is used as a non-carcinogenic endpoint specific to inhalation exposure. It is expressed as $\mu\text{g chemical} / \text{m}^3 \text{ air inhaled}$ (e.g. $\mu\text{g} / \text{m}^3$). The RfC is the estimate of lifetime daily exposure to a non-carcinogenic substance for the general human population that appears to be without appreciable risk of deleterious effects. The RfC is derived from either the NOAEL or the LOAEL, typically determined in a laboratory study. Uncertainty factors (UF) are applied to the NOAEL or LOAEL to account for interspecies variability and intraspecies variability (e.g. sensitive sub-populations). Additionally, uncertainty factors are applied to extrapolate from subchronic exposure to chronic exposure or where there is a paucity of data available for a chemical (e.g. no data regarding effects on reproduction). For the purposes of this study, RfC's were chosen for chemical evaluation. If an RfC did not exist an Oral RfD was converted to a RfC by multiplying the RfD by the receptor's body weight and dividing by the air inhalation rate.

1.2 Carcinogenic TRVs

Carcinogenic chemicals exhibit non-threshold effects following exposure. Non-threshold effects are defined by the observation of adverse effects regardless of concentration and length of exposure. Primarily, two TRVs are used to describe carcinogenic effects: the slope factor and unit risk.

A slope factor (SF) is used for assessment of carcinogenic effects of a chemical. The SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime, expressed as $(\text{mg}/\text{kg body weight}/\text{day})^{-1}$. It is used to estimate an upper bound probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen.

Unit risks are used to estimate an upper bound probability of an individual developing cancer as a result of exposure to a particular level (usually as $1 \mu\text{g}/\text{m}^3$ in air) of a potential carcinogen. Unit risks are calculated by dividing the SF by body weight and multiplying that product by the inhalation or drinking rate as applicable.

Health Canada uses tumorigenic doses and concentrations for substances that are considered to have non-threshold or carcinogenic effects. The potency is expressed as a dose or concentration that will induce a 5% increase in the incidence of tumours or deaths due to tumours as calculated from a dose-response curve. The TRVs that defined the 5% increased are tumorigenic concentration 05 (TC_{05}) primarily used as a benchmark for exposure to a certain chemical in air or tumorigenic dose 05 (TD_{05}).

If an inhalation unit risk was not available an Oral Slope Factor was converted to a unit risk by multiplying the slope factor by the air inhalation rate and dividing by the receptor's body weight.

For the purposes of this report, given that only the inhalation pathway was being examined, UR and TC_{05} concentrations were converted to their exposure values representing the airborne concentration that would result in a one in one hundred thousand cancer risk as shown:

$$\text{Carcinogenic Exposure values } (\text{mg}/\text{m}^3) = \frac{0.00001}{\text{Unit Risk } (\text{mg}/\text{m}^3)^{-1}}$$

2.0 ACETONE

2.1 General Health Effects

There have been several studies located regarding health effects in humans following the inhalation of acetone. However, it has been generally reported that the health effects in humans are neurological in nature (ATSDR, 1994).

2.2 Assessment of Carcinogenicity

The US EPA indicates that data are inadequate for an assessment of the human carcinogenic potential of acetone, as there is a lack of data concerning carcinogenicity in humans or animals (US EPA, 2003).

2.3 Susceptible Populations

Animal studies suggest that sex differences play a role in the susceptibility to the effects caused by acetone. Male rats were more susceptible to hematological, hepatic, and renal effects as well as effects to reproductive organs (American Biogenics Corp, 1986; NTP, 1991), while pregnant female rats exhibited decreased body weight (NTP 1991). Diabetics may also be more susceptible to the effects of acetone, as acetone induced insulin resistance may result in greater hyperglycemia in diabetics (Skutches et al., 1991). Furthermore, patients with diabetic ketoacidosis have higher plasma levels of endogenous acetone and exposure to exogenous acetone may increase the levels further (Reichard et al., 1986).

2.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

2.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

2.4.2 Non-Cancer Inhalation Toxicity Reference Values

The ATSDR has published a chronic inhalation MRL of 30,900 $\mu\text{g}/\text{m}^3$ (ATSDR, 1994). The MRL was derived from a LOAEL of 1,250 ppm (2,970,000 $\mu\text{g}/\text{m}^3$) in humans for neurological effects (Stewart et al., 1975). The ATSDR intermediate MRL is equivalent to the chronic MRL. The ATSDR also provides an acute inhalation MRL of 61,800 $\mu\text{g}/\text{m}^3$, derived from a LOAEL of 237 ppm (503,000 $\mu\text{g}/\text{m}^3$) for 4 hours for neurobehavioral effects in humans in an experimental study by Dick et al. (1989).

Table 1 summarizes the selected toxicity reference values for Acetone.

Table 2-1: Selected Toxicity Reference Values for Acetone

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	61,800 $\mu\text{g}/\text{m}^3$	Neurological	ATSDR
Inhalation - intermediate	30,900 $\mu\text{g}/\text{m}^3$	Neurological	ATSDR
Inhalation - chronic	30,900 $\mu\text{g}/\text{m}^3$	Neurological	ATSDR
Cancer Effects			
Inhalation	NA	NA	NA

Notes:

NA: Not Applicable

2.5 References

American Biogenics Corp. 1986. Ninety day gavage study in albino rats using acetone.

American Biogenics Corp, Decatur, IL. [Unpublished study to be peer reviewed]

ATSDR (Agency for Toxic Substances and Disease Registry). 1994. Toxicological Profile for Acetone. Available on-line at: <http://www.atsdr.cdc.gov>

Dick, RB, Setzer, JV, Taylor, BJ, et al. 1989. Neurobehavioral effects of short duration exposures to acetone and methyl ethyl ketone. Br J Ind Med 46: 111-121.

NTP. 1991. National Toxicology Program - technical report no. 3. Toxicity studies of acetone in F344/N rats and B6C3F, mice (drinking water studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institute of Health. NIH publication No. 91-3122.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

Reichard, Jr GA, Skutches, CL, Hoeldtke, RD, et al. 1986. Acetone metabolism in humans during diabetic ketoacidosis. *Diabetes* 35:668-674.

Skutches, CL, Owen, OE, Reichard, Jr GA. 1990. Acetone and acetol inhibition of insulin-stimulated glucose oxidation in adipose tissue and isolated adipocytes. *Diabetes* 39:450-455.

Stewart, RD, Hake, CL, Wu, A, et al. 1975. Acetone: Development of a biologic standard for the industrial worker by breath analysis. Cincinnati, OH: National Institute for Occupational Safety and Health. NTIS PB82-172917.

U.S. EPA (Environmental Protection Agency). 2003. Integrated Risk Information System (IRIS) Database – Acetone. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

3.0 BENZENE

3.1 General Health Effects

Chronic inhalation of certain levels of benzene causes disorders in the blood in humans (ATSDR, 1997; Environment Canada and Health Canada, 1993). Benzene specifically affects bone marrow (the tissues that produce blood cells) (ATSDR, 1997). Aplastic anemia, excessive bleeding, and damage to the immune system (by changes in blood levels of antibodies and loss of white blood cells) may develop (ATSDR, 1997). In animals, chronic inhalation and oral exposure to benzene produces the same effects as seen in humans (ATSDR, 1997). Benzene causes both structural and numerical chromosomal aberrations in humans (ATSDR, 1997).

3.2 Assessment of Carcinogenicity

Increased incidence of leukemia (cancer of the tissues that form white blood cells) has been observed in humans occupationally exposed to benzene (ATSDR, 1997, US EPA, 2003). The US EPA has classified benzene in Group A - known human carcinogen (US EPA, 2003). Health Canada has classified benzene as Group 1 – carcinogenic to man, under the classification scheme developed by the Bureau of Chemical Hazards (Environment Canada and Health Canada, 1993).

3.3 Susceptible Populations

Ethanol is reported to increase the severity of chronic non-cancer benzene effects (Baarson et al., 1982). Although there are possible indications of a genetic susceptibility to benzene, it is believed that all humans are susceptible to the pancytopenic effects of benzene (ATSDR, 1997). Evidence from human and animal studies suggests that increases in childhood leukemia may be associated with in-utero exposures and maternal and paternal exposure prior to conception (US EPA, 2002). Children and fetuses may be at increased risk of aplastic anemia effects as dividing cells are at a greater risk (Aksoy, 1989). Also, studies suggest that “unhealthy lifestyles,” including smoking, excessive alcohol consumption, inadequate physical exercise and sleep, excessive stress, and inadequate nutritional balance could make cells more sensitive to the production of chromosomal aberrations after exposure to benzene (ATSDR, 1997).

3.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

3.4.1 Cancer Inhalation Toxicity Reference Values

In humans, chronic inhalation exposure to benzene in the workplace resulted in an increased incidence of leukemia (US EPA, 2003). The inhalation unit risk of $3.3 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$ (Health Canada, 2003), based on the TC_{05} recommended by Health Canada (1996) was used to calculate a TRV of $3.03 \mu\text{g}/\text{m}^3$ for a 1 in 100,000 risk level.

3.4.2 Non-Cancer Inhalation Toxicity Reference Values

The ATSDR has published an acute-duration inhalation MRL of $160 \mu\text{g}/\text{m}^3$, derived from a LOAEL value of 10 ppm ($31,900 \mu\text{g}/\text{m}^3$) for reduced lymphocyte proliferation following mitogen stimulation in mice (Rozen et al., 1984).

ATSDR's intermediate-duration inhalation MRL of $12.8 \mu\text{g}/\text{m}^3$ was derived from a LOAEL value of 0.78 ppm ($2,490 \mu\text{g}/\text{m}^3$) for neurological effects of intermediate-duration inhalation exposure of mice to benzene (Li et al., 1992).

The US EPA (2003) established a reference concentration (RfC) of $30 \mu\text{g}/\text{m}^3$ for benzene based on BMD of $8.2 \text{ mg}/\text{m}^3$ modeling of ALC data from an occupational epidemiologic study by Rothman et al. (1996), in which workers were exposed to benzene by inhalation and showed decreased lymphocyte counts. An uncertainty factor (UF) of 300 was applied to the BMD, 3 for conversion of LOAEL, 10 for intraspecies differences, 3 for subchronic to chronic, and 3 for database deficiencies.

The US EPA (2003) states that overall confidence in this RfC is medium.

Table 2 summarizes the selected toxicity reference values for benzene.

Table 3-1: Selected Toxicity Reference Values for Benzene

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	160 $\mu\text{g}/\text{m}^3$	Immunologic	ATSDR
Inhalation - intermediate	12.8 $\mu\text{g}/\text{m}^3$	Neurologic	ATSDR
Inhalation - chronic	30 $\mu\text{g}/\text{m}^3$	Decreased lymphocyte count	US EPA
Cancer Effects			
Inhalation - chronic	3.03 $\mu\text{g}/\text{m}^3$	Leukaemia	Health Canada

Notes:

NA: Not Applicable

3.5 References

Aksoy, M. 1989. Hematotoxicity and carcinogenicity of benzene. *Environ Health Perspect* 82:193-197. Cited In: ATSDR, 1997.

ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Toxicological Profile for Benzene. Available on-line at: <http://www.atsdr.cdc.gov>

Baarson, K, Snyder CA, Green J, et al. 1982. The hematotoxic effects of inhaled benzene on peripheral blood, bone marrow and spleen cells are increased by ingested ethanol. *Toxicol Apl Pharmacol* 64: 393-404.

Environment Canada and Health Canada. 1993. Canadian Environmental Protection Act, Priority Substances List Assessment Report, Benzene. Government of Canada, Environment Canada, Health Canada.

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumorigenic Doses/Concentrations for Priority Substances. ISBN 0-662-24858-9.

Health Canada, 2003. Federal Contaminated Site Risk Assessment In Canada Part II: Health Canada Toxicological Reference Values (TRVs), Version 1.0, October 3, 2003.

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- US EPA (Environmental Protection Agency). 2002. Toxicological Review of Benzene (noncancer) Effects. CAS No. 71-43-2. In Support of Summary information on the Integrated Risk Information System. October 2002. Available on-line at: <http://www.epa.gov/iris/>
- U.S. EPA (Environmental Protection Agency). 2003. Integrated Risk Information System (IRIS) Database - Benzene. Confirmed current as of May 2003. Available on-line at: <http://www.epa.gov/iris/>

4.0 BIPHENYL, 1,1-

4.1 General Health Effects

There have been no studies located regarding health effects in humans following the inhalation of 1,1 –biphenyl.

4.2 Assessment of Carcinogenicity

The US EPA classifies biphenyl as a Class D chemical, that is, not classifiable as to human carcinogenicity on the basis that no human data and inadequate studies in mice and rats (US EPA, 2004). The results of genotoxicity tests are generally negative.

4.3 Susceptible Populations

No data regarding susceptible populations were found.

4.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

4.4.1 Cancer Inhalation Toxicity Reference Values

There are no published non-cancer TRVs for 1,1-biphenyl via an inhalation route of exposure.

4.4.2 Non-Cancer Inhalation Toxicity Reference Values

No inhalation TRVs were found for 1,1-byphenyl. A chronic RfC was therefore calculated from the US EPA chronic oral RfD of 50 µg/kg-d. The US EPA oral RfD for 1,1-biphenyl is based on a study of weanling albino rats that were fed varying percentages of biphenyl in their diet. Dietary levels of 0.5% biphenyl and greater were associated with kidney damage, reduced hemoglobin levels, decreased food intake, and decreased longevity (Ambrose, et al., 1960). Uncertainty factors applied to the 50

mg/kg-d NOAEL were a total of 1000., 10 for interspecies comparison, 10 for sensitive human population and and Modifying factor of 10 for intraspecies variability.

The US EPA (1992) states that overall confidence in this RfD is medium.

Although the health effects data for biphenyl were reviewed by the U.S. EPA RfD/RfC Work Group, and determined to be inadequate for derivation of an inhalation RfC, the lack of inhalation TRVs necessitate the conversion of the RfD into an RfC for use in this assessment.

The RfD was converted to a to an RfC by multiplying the RfD by the receptor's body weight and dividing by the air inhalation rate. Conversions were made for adult and toddler receptors. The relevant receptor characteristics and resultant TRVs are presented in Table 3.

Table 4-1: Receptor Characteristics and Calculated RfCs for 1-1-biphenyl

Receptor Characteristic	Source	Toddler	Adult
Body weight (kg)	Health Canada , 2003	16.5	70.7
Inhalation rate (m ³ /d)	Health Canada , 2003	9.3	15.8
Calculated RfC (µg/m³)		89 µg/m ³	224 µg/m ³

Table 4 summarizes the selected toxicity reference values and Table 11 summarizes the selected relative bioavailabilities.

Table 4-2: Selected Toxicity Reference Values for 1,1-biphenyl

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	NA	NA	NA
Inhalation – chronic – Toddler	89 µg/m ³	Kidney damage	Calculated based on US EPA RfD
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:NA: Not Applicable

4.5 References

Ambrose, A.M., A.N. Booth, F. DeEds and A.J. Cox, Jr. 1960. A toxicological study of biphenyl, a citrus fungistat. Food Res. 25: 328-336. Cited in US EPA, 2004

Environment Canada and Health Canada. 1993. Canadian Environmental Protection Act, Priority Substances List Assessment Report, Benzene. Government of Canada, Environment Canada, Health Canada.

U.S. EPA (Environmental Protection Agency). 2004. Integrated Risk Information System (IRIS) Database – 1,1-Biphenyl. Confirmed current as of July 2004. Available on-line at: <http://www.epa.gov/iris/>

5.0 CARBON DISULPHIDE

5.1 General Health Effects

At very high concentrations, inhaled carbon disulphide may be life threatening because of its effects on the central nervous system. At lower concentrations, as well as over longer durations, studies have shown inhaled carbon disulphide may cause breathing and chest pain, headaches, tiredness, and trouble sleeping (ATSDR, 1996). Psychiatric symptoms, such as irritability, anger, mood changes, manic delirium and hallucinations, paranoid ideas, loss of appetite, gastrointestinal disturbances and sexual disorders have also been noted as a result of acute exposure (WHO, 2000).

5.2 Assessment of Carcinogenicity

The US EPA has not undertaken a complete evaluation and determination under its IRIS program for evidence of human carcinogenic potential (US EPA, 1995), nor has the International Agency for Research on Cancer (IARC).

5.3 Susceptible Populations

ATSDR states that individuals at risk for arteriosclerosis or those with early arteriosclerosis would probably be at increased risk for health effects following exposure to carbon disulfide (NIOSH 1978).

Three other groups are recognized as being unusually susceptible to carbon disulfide: alcoholics (including those treated with Antabuse), those with neuropsychic disorders, and those with vitamin B6 deficiency (Djuric et al. 1973; Lefaux 1968; Peters et al. 1982).

5.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

5.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

5.4.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (1995) has published a Reference Concentration for Chronic Inhalation Exposure (RfC) of 700 $\mu\text{g}/\text{m}^3$ based on the results of a study by Johnson et al. (1983), which showed peripheral nervous system dysfunction in viscose rayon workers after longterm inhalation exposure. The RfC is based on a benchmark concentration of 19.7 mg/m^3 and a total uncertainty factor of 30 applied, 3 for sensitive members of the population, and 10 for information deficiencies in the database. The confidence in the RfC is medium.

Table 12 summarizes the selected toxicity reference values and Table 13 summarizes the selected relative bioavailabilities.

Table 5-1: Selected Toxicity Reference Values for Carbon Disulphide

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	NA	NA	NA
Inhalation – int.	NA	NA	NA
Inhalation - chronic	700 $\mu\text{g}/\text{m}^3$	Nervous system	US EPA, IRIS
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

5.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 1996. Toxicological Profile for Carbon Disulphide. Available on-line at: <http://www.atsdr.cdc.gov>

Djuric, D, Postic-Grujin, A, Graovac-Leposavic, L, et al. 1973. Antabuse as an indicator of human susceptibility to carbon disulfide: Excretion of diethyldithiocarbamate sodium in the urine of workers exposed to CS₂ after oral administration of disulfiram. Arch Environ Health 26:287-289.

- Lefaux, R. 1968. Practical toxicology of plastics. Cleveland: CRC Press, Inc., 117-119.
- NIOSH. 1978. Occupational health guideline for carbon disulfide. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, 1-3.
- OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.
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- U.S. EPA (Environmental Protection Agency). 1995. Integrated Risk Information System (IRIS) Database – Carbon Disulfide. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>
- WHO (World Health Organization). 2000. Air Quality Guidelines for Europe.

6.0 CUMENE (ISOPROPYL BENZENE)

6.1 General Health Effects

There have been no studies quantifying the toxicity of cumene in humans. The most prominent effects observed in rodents exposed repeatedly to cumene by inhalation were increases in organ weights (most notably kidney) (Cushman et al., 1995).

6.2 Assessment of Carcinogenicity

The International Agency for Research on Cancer (IARC) has not evaluated cumene in terms of its carcinogenic potential. The US EPA (1997) has concluded that the carcinogenic potential of cumene cannot be determined because no adequate data, such as well-conducted long-term animal studies or reliable human epidemiological studies, are available to perform any assessment. Cumene is therefore assigned carcinogen category D (not classifiable), indicating inadequate or no human or animal data.

6.3 Susceptible Populations

The only toxicity information on cumene of possible relevance to increased childhood susceptibility is that from developmental studies, one study in rats (Bushy Run Research Center, 1989a) and another in rabbits (Bushy Run Research Center, 1989b), in which no adverse fetal effects were observed. There is too little information to make any further statements about how children may be differentially affected by cumene, as there are no data regarding cumene exposure prior to mating, from conception through implantation, or during late gestation, parturition, or lactation (US EPA, 1997).

There were no studies located regarding other susceptible populations and exposure to cumene.

6.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

6.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

6.4.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (1995) has published a Reference Concentration for Chronic Inhalation Exposure (RfC) of 400 $\mu\text{g}/\text{m}^3$ based on the NOEL (435 mg/m^3) for significant increases (>10%) in renal and adrenal weights in rats exposed to cumene in the subchronic inhalation study of Cushman et al. (1995). Overall the US EPA applied a 1000 for uncertainty factor into the derivation of the RfC, 10 subchronic to chronic, 10 intraspecies differences, 3 for database deficiencies, and 3 for interspecies comparisons.

The US EPA (1999) states that overall confidence in this RfC is medium.

There were no toxicity values published for acute or intermediate exposures to cumene.

Table 6 summarizes the selected toxicity reference values for cumene.

Table 6-1: Selected Toxicity Reference Values for Cumene

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	NA	NA	NA
Inhalation – int.	NA	NA	NA
Inhalation - chronic	400 $\mu\text{g}/\text{m}^3$	Increased renal and adrenal weights	US EPA - IRIS
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

6.5 References

- Bushy Run Research Center. 1989a. Cumene fourteen-week vapor inhalation study in rats with neurotoxicity evaluation (part 1-2) with attached studies and cover letter dated December 7, 1989. TSCATS/0522881; EPA/OTS Doc. No. 40-8992172.
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- U.S. EPA (Environmental Protection Agency). 1997. Integrated Risk Information System (IRIS) Database – Cumene. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

7.0 HEXACHLOROBENZENE

7.1 General Health Effects

There is no information on the human health effects of exposure to hexachlorobenzene in air. Studies conducted with rats indicate that breathing hexachlorobenzene may harm the immune system (ATSDR, 2002).

7.2 Assessment of Carcinogenicity

The International Agency for Research on Cancer (IARC) has determined that hexachlorobenzene is possibly carcinogenic to humans. The US EPA has determined that hexachlorobenzene is a probable human carcinogen (US EPA, 1996), while CEPA considers hexachlorobenzene to be a toxic substance to Human Life or Health (CEPA, 1993).

7.3 Susceptible Populations

There have not been any studies located regarding susceptibility to hexachlorobenzene via inhalation; however, case studies indicate that young children are sensitive to hexachlorobenzene intoxication (ATSDR, 2002). Children (average age 7) who ingested the chemical between 1955-1959 in Turkey through contaminated bread developed short stature, pinched faces, osteoporosis of bones of the hand and painless arthritic changes. Some children in this study were presumed to have been exposed in utero, via transplacental transfer and postnatally, via lactational transfer (ATSDR, 2002).

7.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

7.4.1 Cancer Inhalation Toxicity Reference Values

The US EPA (1994) has developed an inhalation unit risk value of $0.00046 (\mu\text{g}/\text{m}^3)^{-1}$, which was based on extrapolations from oral exposure data showing hepatocellular

carcinoma in female rats (Erturk et al., 1986). This translates into a carcinogenic exposure value of $0.02 \mu\text{g}/\text{m}^3$ for a 1 in 100,000 risk level.

Table 7 summarizes the selected toxicity reference values for hexachlorobenzene.

Table 7-1: Selected Toxicity Reference Values for Hexachlorobenzene

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	NA	NA	NA
Inhalation – chronic	NA	NA	NA
Cancer Effects			
Inhalation - chronic	$0.02 \mu\text{g}/\text{m}^3$	Hepatocellular carcinoma	US EPA

Notes:

NA: Not Applicable

7.5 References

ATSDR 2002 Agency for Toxic Substances and Disease Registry. Toxicological Profile for hexachlorobenzene. Available on-line at: <http://www.atsdr.cdc.gov/tfacts90.html>

Erturk, E., R.W. Lambrecht, H.A. Peters, D.J. Cripps, A. Gocmen, C.R. Morris and G.T. Bryan. 1986. Oncogenicity of hexachlorobenzene. In: Hexachlorobenzene: Proc. Int. Symp., C.R. Morris and J.R.P. Cabral, Ed. IARC Scientific Publ. No. 77, Oxford University Press, Oxford. p. 417-423.

U.S. EPA (Environmental Protection Agency). 1991. Integrated Risk Information System (IRIS) Database – Hexachlorobenzene. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

8.0 HYDROGEN CHLORIDE (HYDROCHLORIC ACID, HCL)

8.1 General Health Effects

Hydrochloric acid is corrosive to the eyes, skin, and mucous membranes. Acute inhalation exposure may cause coughing, hoarseness, inflammation and ulceration of the respiratory tract, chest pain, and pulmonary edema in humans (US EPA, 1999).

8.2 Assessment of Carcinogenicity

There is no information available on the carcinogenic effects of hydrogen chloride (HCl) in humans. In one study, no carcinogenic response was observed in rats exposed to HCl via inhalation (US Dept. of Health and Human Services, 1993). The US EPA has not classified HCl with respect to potential carcinogenicity (US EPA, 1999).

8.3 Susceptible Populations

Persons suffering from skin, respiratory, or digestive diseases may be more susceptible to the toxic effects of exposure to HCl (US Dept. of Health and Human Services, 2002).

8.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

8.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

8.4.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (1999) has published a Reference Concentration for Chronic Inhalation Exposure (RfC) of 20 $\mu\text{g}/\text{m}^3$ based on hyperplasia of the nasal mucosa, larynx, and trachea in rats. The RfC is based on the LOAEL of 6.1 mg/m^3 and a total uncertainty factor of 300; 10 for intraspecies variability, 10 to convert the LOAEL to NOAEL, and 3

for interspecies comparison. The US EPA (1999) states that overall confidence in this RfC is low.

The California EPA Office of Environmental Health Hazard has published a reference level for acute exposure to HCl in the air of 2,100 $\mu\text{g}/\text{m}^3$, which should be compared to the modeled 1 hour concentrations of HCl in the air shed. This acute toxicity reference value is based on a study by Stevens *et. al.* (1992), where a No Observed Adverse Effect Level (NOAEL) of 2,700 $\mu\text{g}/\text{m}^3$ was reported for humans exposed to HCl over a 45 minute period.

This study involved a sensitive human sub-population group and thus no uncertainty factors were used, but the value was time adjusted to a one hour duration giving the proposed reference exposure level (REL) of 2,100 $\mu\text{g}/\text{m}^3$.

The study that forms the basis of the CalEPA REL is for human populations, thus eliminating the need for the uncertainty factor of 3 applied by the US EPA for interspecies extrapolation. Additionally, the study uses a NOAEL rather than a LOAEL, thus eliminating the need for the uncertainty factor of 10 applied for the use of a LOAEL. The fact that a sensitive human sub-population is the basis of the CalEPA value, significantly reduces the need for an uncertainty factor to account for intraspecies variability.

Table 8 summarizes the selected toxicity reference values for hydrogen chloride.

Table 8-1: Selected Toxicity Reference Values for Hydrogen Chloride

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	2,100	irritation	Cal EPA
Inhalation – intermediate	NA	NA	NA
Inhalation - chronic	20 $\mu\text{g}/\text{m}^3$	Nasal hyperplasia	US EPA - IRIS
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

8.5 References

CAL EPA (California Environmental Protection Agency) 2003. Chronic Toxicity Summary Hydrogen Chloride. Office of Environmental Health Hazard Assessment.

CAL EPA (California Environmental Protection Agency) 1999. Acute Toxicity Summary Hydrogen Chloride. Office of Environmental Health Hazard Assessment.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

US Department of Health and Human Services. 2002. Hazardous Substances Data Bank (HSDB). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. Available on-line at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/>

US EPA (Environmental Protection Agency). 1993. Integrated Risk Information System (IRIS) on Hydrogen Chloride. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris>

9.0 HYDROGEN FLUORIDE

9.1 General Health Effects

Chronic inhalation exposure of humans to hydrogen fluoride has resulted in irritation and congestion of the nose, throat, and bronchi at low levels (US EPA, 1989; Ca-EPA, 1997). Increased bone density has also been reported among workers chronically exposed to hydrogen fluoride by inhalation (Ca-EPA, 1997).

9.2 Assessment of Carcinogenicity

Epidemiological studies have not demonstrated an association between fluoride in drinking water and an increased risk of cancer (US EPA, 1989). Increased rates of cancer have been observed in workers involving possible fluoride exposure; however, these situations involved mixed exposures to several chemicals and hydrogen fluoride could not be specifically implicated as the cause of the cancers (ATSDR, 2001). The US EPA has not classified hydrogen fluoride with respect to potential carcinogenicity.

9.3 Susceptible Populations

Existing data indicate that the elderly, people with deficiencies of calcium, magnesium, and/or vitamin C, and people with cardiovascular and kidney problems may be unusually susceptible to the toxic effects of fluoride and its compounds; however, these effects would not be expected at typical exposure levels (i.e., 1 ppm) (ATSDR, 2001).

9.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

9.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

9.4.2 Non-Cancer Inhalation Toxicity Reference Values

The ATSDR has published an acute inhalation MRL of $16.4 \mu\text{g}/\text{m}^3$, based on the minimal LOAEL of 0.5 ppm ($409 \mu\text{g}/\text{m}^3$) fluoride for upper respiratory tract irritation from a study by Lund et al. (1997, 1999). This is a final MRL.

The Cal-EPA has published a chronic inhalation reference exposure level of $14 \mu\text{g}/\text{m}^3$ for humans based on a LOAEL of $1,890 \mu\text{g}/\text{m}^3$ presented in study by Derryberry et al. (1963), indicating effects on bone density in humans (Cal-EPA, 1997). The Cal-EPA reference exposure level is a concentration below which adverse human health effects are not likely to occur.

Table 9 summarizes the selected toxicity reference values for hydrogen fluoride.

Table 9-1: Selected Toxicity Reference Values for Hydrogen Fluoride

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	$16.4 \mu\text{g}/\text{m}^3$	Respiratory	ATSDR
Inhalation - intermediate	NA	NA	NA
Inhalation - chronic	$14 \mu\text{g}/\text{m}^3$	Bone density effects	Cal-EPA
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

9.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 2001. Toxicological Profile for Fluorides, Hydrogen Fluoride and Fluorine. US Public Health Service, US Department of Health and Human Services, Atlanta, GA. Available on-line at: <http://www.atsdr.cdc.gov>

Cal-EPA (California Environmental Protection Agency). 1997. Technical Support Document for the Determination of Noncancer Chronic Reference Exposure

- Levels. Draft for Public Comment. Office of Environmental Health Hazard Assessment, Berkeley, CA.
- Derryberry OM, Bartholomew MD, and Fleming RBL. 1963. Fluoride exposure and worker health - The health status of workers in a fertilizer manufacturing plant in relation to fluoride exposure. *Arch. Environ. Health* 6:503-514.
- Lund K, Refsnes M, Sandstrom T, et al. 1999. Increased CD3 positive cells in bronchoalveolar lavage fluid after hydrogen fluoride inhalation. *Scand J Work Environ Health* 25(4):326-334.
- Lund, K., J. Ekstrand, J. Boe, P. Sostrand, and J. Kongerud. 1997. Exposure to hydrogen fluoride: an experimental study in human of concentrations of fluoride in plasma, symptoms, and lung function. *Occup. Environ Med* 1977; 54:32-37. Cited in CalEPA, 1999.
- Morris, J.B., and F.A. Smith. 1982. Regional deposition and absorption of inhaled hydrogen fluoride in the rat. *Toxicol Appl Pharmacol* 62:81-89. Cited in ATSDR, 2001.
- OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

10.0 HYDROGEN SULPHIDE

10.1 General Health Effects

The US EPA (2004) states that numerous case-reports that identify hydrogen sulphide as a component of the chemical of exposure demonstrate that high levels of inhaled hydrogen sulphide for even very brief periods are life-threatening and can cause immediate unconsciousness followed by serious and debilitating neurologic and respiratory effects and death (Allyn, 1931; Milby 1962). Exposure to lower concentrations can result in eye irritation, a sore throat and cough, shortness of breath, and fluid in the lungs. These symptoms usually go away in a few weeks. Long-term, low-level exposure may result in fatigue, loss of appetite, headaches, irritability, poor memory, and dizziness (ATSDR, 1999).

10.2 Assessment of Carcinogenicity

Neither the IARC nor the US EPA has evaluated hydrogen sulphide for carcinogenicity. The US EPA states that there is not adequate data for an assessment of the carcinogenic potential of hydrogen sulphide (US EPA, 2004).

10.3 Susceptible Populations

At least one study has suggested that persons with neuropsychiatric disorders might be more susceptible (Poda, 1966). Furthermore, additional studies suggest that some asthmatics may be more sensitive to hydrogen sulfide than the general population (Jappinen et al., 1990; Shim and Williams, 1986). It also seems likely that individuals already suffering from hydrogen sulfide-associated toxicity will be at higher risk from further hydrogen sulfide exposures (ATSDR, 1999).

10.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

10.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

10.4.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (1999) has published a chronic RfC of $2.0 \mu\text{g}/\text{m}^3$ based on NOAELs for nasal lesions of the olfactory mucosa in rat studies. The RfC is based on a $\text{NOAEL}_{(\text{HEC})}$ of $0.64 \text{ mg}/\text{m}^3$ with uncertainty factors of 300 applied; 3 for interspecies variability, 10 for sensitive populations, and 10 for conversion from subchronic to chronic dose. The US EPA (1999) states that overall confidence in this RfC is medium to high.

The WHO provides an intermediate tolerable concentration of $20 \mu\text{g}/\text{m}^3$, based on a NOAEL of $14,000 \mu\text{g}/\text{m}^3$ for nasal lesions in the olfactory mucosa in a study by Brenneman et al. (2000), in which male rats were exposed to hydrogen sulphide. This TRV was derived in a similar manner to the US EPA value, however, with only an uncertainty factor of 30 employed, as subchronic to chronic dose was not considered.

Finally, the ATSDR and WHO provide an acute inhalation MRL for hydrogen sulphide of $97.6 \mu\text{g}/\text{m}^3$, based on respiratory effects in human population (ATSDR, 1999).

Table 10 summarizes the selected toxicity reference values and Table 26 summarizes the selected relative bioavailabilities.

Table 10-1: Selected Toxicity Reference Values for Hydrogen Sulphide

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	97.6 µg/m ³	Respiratory effects	ATSDR/WHO
Inhalation - intermediate	20 µg/m ³	Nasal lesions	WHO
Inhalation - chronic	2.0 µg/m ³	Nasal lesions	US EPA
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

10.5 References

Allyn, LB. (1931) Notes on hydrogen sulfide poisoning. *Ind. and Eng. Chem.* 23:234. Cited in U.S. EPA, 2004.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Hydrogen Sulphide. Available on-line at: <http://www.atsdr.cdc.gov>

Brenneman KA, James A, Gross EA, Dorman DC (2000) Olfactory neuron loss in adult male CD rats following subchronic inhalation exposure to hydrogen sulfide. *Toxicologic Pathology*, 28:326–333. Cited in: WHO, 2003.

Jappinen P, Tenhunen R. 1990. Hydrogen sulphide poisoning: Blood sulphide concentration and changes in haem metabolism. *Br J Ind Med* 47:283-285. Cited in ARSDR, 1999.

Milby, TH. (1962) Hydrogen sulfide intoxication; review of the literature and report of an unusual accident resulting in two cases of nonfatal poisoning. *J. Occup. Med.* 4: 431-437. Cited in U.S. EPA, 2004.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria

- (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.
- Poda GA. 1966. Hydrogen sulfide can be handled safely. Arch Environ Health 12:795-800. Cited in ATSDR, 1999.
- Shim C, Williams MH. 1986. Effects of odor on asthma. Am J Med 80:18-22. Cited in ATSDR, 1999.
- U.S. EPA (Environmental Protection Agency). 2004. Integrated Risk Information System (IRIS) Database – Hydrogen Sulphide. Confirmed current as of July 2004. Available on-line at: <http://www.epa.gov/iris/>.
- WHO (World Health Organization). 2003. Hydrogen Sulfide: Human Health Aspects, Concise International Chemical Assessment Document 53. Geneva. ISBN 92 4 153053 7.

11.0 ISOPROPYL ALCOHOL

11.1 General Health Effects

No adequate chronic exposure studies located for health effects in humans following the inhalation of isopropyl alcohol (Ca-EPA, 2003); however, studies of animals have shown kidney lesions in rats and mice, as well as fetal growth retardation and developmental anomalies in rats (Burleigh-Flayer et al, 1997; Nelson et al, 1988).

11.2 Assessment of Carcinogenicity

The IARC indicates that isopropyl alcohol is a Group 3 chemical, and therefore is not classifiable as to its carcinogenicity to humans. The US EPA has not assessed isopropyl alcohol with regard to its carcinogenicity.

11.3 Susceptible Populations

No studies indicating susceptible populations were found.

11.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

11.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

11.4.2 Non-Cancer Inhalation Toxicity Reference Values

The Ca-EPA (2003) has published a chronic inhalation reference exposure level of 7000 $\mu\text{g}/\text{m}^3$ based on kidney lesions in mice and rats, and fetal growth retardation and developmental anomalies in rats in studies undertaken by Burleigh-Flayer et al (1997) and by Nelson et al (1988).

Table 11 summarizes the selected toxicity reference values and Table 28 summarizes the selected relative bioavailabilities.

Table 11-1: Selected Toxicity Reference Values for Isopropyl Alcohol

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	NA	NA	NA
Inhalation – intermediate	NA	NA	NA
Inhalation - chronic	7000 µg/m ³	Kidney; developmental	Cal-EPA
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

11.5 References

Burleigh-Flayer H, Gill M, Hurley J, Bevan C, Gardiner T, Kapp R, Tyler T, Wright G. 1998. Motor activity effects in female Fischer 344 rats exposed to isopropanol for 90 days. *J. Appl. Toxicol.* 18:373-381. Cited in Cal-EPA, 2003.

Brugnone F, Perbellini L, Apostoli P, Bellomi M, and Caretta D. 1983. Isopropanol exposure: environmental and biological monitoring in a printing works. *Br. J. Ind. Med.*, 40:160-168. Cited in Cal-EPA, 2003.

Cal-EPA (California Environmental Protection Agency). 2003. Chronic Toxicity Summary: Isopropanol. Office of Environmental Health Hazard Assessment. Available on-line at: http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html

Nelson, BK, Brightwell, WS, MacKenzie-Taylor, DR, Khan, A, Burg, JR, Weigel, WW, and Goad, PT. 1988. Teratogenicity of *n*-propanol and isopropanol administered at high inhalation concentrations to rats. *Food Chem. Toxicol.* 26(3):247-254. Cited in Cal-EPA, 2003.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

12.0 METHYL ISOBUTYL KETONE

12.1 General Health Effects

Occupation exposure of workers to solutions containing MIBK have indicated transient sensory irritation, neurological effects and odour sensitization, in addition to reports of eye, nose, and throat irritation (US EPA, 2003a).

12.2 Assessment of Carcinogenicity

The US EPA has concluded that the data for MIBK are inadequate for an assessment of human carcinogenic potential (US EPA, 2003a). This characterization is based on the absence of both cancer epidemiology studies in humans and carcinogenicity assays in animals.

12.3 Susceptible Populations

There were no human studies located to indicate the relative sensitivity of children and adults to the toxic effects of MIBK (US EPA, 2003a).

12.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

12.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

12.4.2 Non-Cancer Inhalation Toxicity Reference Values

The US EPA (2003b) has published an RfC of 3,000 $\mu\text{g}/\text{m}^3$ based on reduced fetal body weight, skeletal variations, and increased fetal death in mice, and increased fetal death in rats, presented in a study by Tyl et al. (1987). The $\text{NOAEL}_{\text{HEC}}$ of 1026 mg/m^3 was divided by an uncertainty factor of 300; 3 for interspecies comparison, 10 intraspecies variability and 10 for database deficiencies.

The US EPA (1999) states that overall confidence in this RfC is low to medium.

Table 12 summarizes the selected toxicity reference values for MIBK.

Table 12-1: Selected Toxicity Reference Values for MIBK

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation – acute	NA	NA	NA
Inhalation – intermediate	NA	NA	NA
Inhalation – chronic	3000 µg/m ³	Fetal, skeletal	US EPA
Cancer Effects			
Inhalation – chronic	NA	NA	NA

Notes:

NA: Not Applicable

12.5 References

Hjelm, E.W., Hagberg M, Iregren A, and A. Lof . 1990. Exposure to methyl isobutyl ketone: toxicokinetics and occurrence of irritative CNS symptoms in man. *Int Arch Occup Environ Health* 62:19-26. Cited in: US EPA, 2003b.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

Tyl, R.W., K.A. France, L.C. Fisher, I.M. Pritts, T.R. Tyler, R.D. Phillips, and E.J. Moran. 1987. Developmental toxicity evaluation of inhaled methyl isobutyl ketone in Fischer 344 rats and CD-1 mice. *Fundam Appl Toxicol* 8:310-327.

U.S. EPA (Environmental Protection Agency). 2003a. Toxicological review of Methyl Isobutyl Ketone (CAS No. 108-10-1). In support of summary on the integrated risk information system. National Center for Environmental Assessment, Washington, DC. Available online at: <http://www.epa.gov/iris>

U.S. EPA (Environmental Protection Agency). 2003b. Integrated Risk Information System (IRIS) Database – Methyl Isobutyl Ketone. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

13.0 NICKEL

13.1 General Health Effects

Nickel is essential to maintaining good health. It is thought that a small amount of nickel is essential for humans (ATSDR, 1997). In sensitized individuals, dermal contact may cause dermatitis, while inhalation may cause asthma (ATSDR, 1997). Inhalation of nickel can effect the respiratory tract. Acute exposure may lead to the inflammation of the respiratory tract while chronic exposure to high levels of nickel may lead to cancer of the nasal cavity and lungs (ATSDR, 1997).

13.2 Assessment of Carcinogenicity

Nickel is considered to be carcinogenic to humans and is listed as a Group 1 carcinogen by IARC. The US EPA (1991) considers nickel refinery dust to be a human carcinogen via inhalation exposure. The carcinogenic activity of nickel is dependent upon the specific species of nickel present. Compounds such as nickel sulphide and nickel subsulphide, both present in nickel refinery dusts have been shown to be carcinogenic in humans (CEPA, 1994; US EPA, 1991).

13.3 Susceptible Populations

Sensitized individuals may be unusually susceptible because exposure to nickel by any route may trigger an allergic response (ATSDR, 1997). Persons with kidney dysfunction are also likely to be more susceptible to nickel as the primary route of nickel elimination is via the urine. Increased nickel serum concentrations have been observed in dialysis patients (Hopfer et al. 1989).

13.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

13.4.1 Cancer Inhalation Toxicity Reference Values

The US EPA (1991) has developed a unit risk value of $0.24 \text{ (mg/m}^3\text{)}^{-1}$, which was based on extrapolations from human epidemiological data showing lung and nasal tumours. This translates into a Benchmark Concentration of $0.042 \text{ }\mu\text{g/m}^3$ for a 1 in 100,000 risk level.

13.4.2 Non-Cancer Inhalation Toxicity Reference Values

The ATSDR derives a draft intermediate-duration MRL of $0.2 \text{ }\mu\text{g/m}^3$ for the inhalation of nickel, based the LOAEL from a study of nickel sulfate (NTP, 1996), where alveolar macrophage hyperplasia was observed in rats. Deriving the MRL from the NTP (1996) study is also protective of toxicity of other nickel compounds. Because data were insufficient to derive an acute-duration MRL, for the purposes of this assessment, the intermediate MRL has been adopted to assess subchronic exposure.

The ATSDR also presents a draft chronic MRL of $0.09 \text{ }\mu\text{g/m}^3$, based on the NOAEL from the NTP (1996) study.

Table 13 summarizes the selected toxicity reference values for nickel.

Table 13-1: Selected Toxicity Reference Values for Nickel

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	$0.2 \text{ }\mu\text{g/m}^3$	Respiratory	ATSDR
Inhalation - chronic	$0.09 \text{ }\mu\text{g/m}^3$	Respiratory	ATSDR
Cancer Effects			
Inhalation - chronic	$0.042 \text{ }\mu\text{g/m}^3$	Lung and nasal tumours	US EPA

Notes:

NA: Not Applicable

13.5 References

- ATSDR (Agency for Toxic Substances and Disease Registry). 1997. Draft Toxicological Profile for Nickel. Available on-line at: <http://www.atsdr.cdc.gov/toxprofiles/>
- CEPA. 1994. Nickel and its Compounds. Canadian Environmental Protection Act. Priority Substances List Assessment Report.
- NTP. 1996. NTP technical report on the toxicology and carcinogenesis studies of nickel sulfate hexahydrate (CAS No. 10101-97-0) in F344/N rats and B6C3F1 mice (inhalation studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP-TRS No. 454.
- OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.
- US EPA (Environmental Protection Agency). 1991. Integrated Risk Information System (IRIS) Database – Nickel – refinery dust. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

14.0 NITROGEN OXIDES(NO_x)

14.1 General Health Effects

Key health effects associated with exposure to NO_x (NO₂) include increased susceptibility to respiratory symptoms and disease in children as well as pulmonary function decrements, symptoms and increased airway resistance in asthmatic subjects and in patients with chronic pulmonary obstructive disease (US EPA, 1995). Exposure to NO₂ has also increased the airway responsiveness in asthmatics (US EPA, 1995).

14.2 Assessment of Carcinogenicity

The US EPA (IRIS, 1997) and Health Canada have not classified NO_x with respect to carcinogenicity. NO_x causes a wide variety of health and environmental impacts because of various compounds and derivatives in the family of nitrogen oxides, including nitrogen dioxide, nitric acid, nitrous oxide, nitrates, and nitric oxide. Ambient air quality guidelines/objectives are generally specific to nitrogen dioxide (NO₂). Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infections such as influenza. The effects of short-term exposure are still unclear, but continued or frequent exposure to concentrations that are typically much higher than those normally found in the ambient air may cause increased incidence of acute respiratory illness in children.

14.3 Susceptible Populations

Two general groups in the population may be more susceptible to the effects of NO₂ exposure than other individuals: persons with pre-existing respiratory disease and children 5 to 12 years old (US EPA, 1995). Individuals in these groups appear to be affected by lower levels of NO₂ than individuals in the rest of the population. Asthmatics are considered to be one of the groups most responsive to NO₂ exposure (US EPA, 1995). Patients with chronic obstructive pulmonary disease (COPD) constitute another subpopulation that is potentially susceptible to NO₂ exposure, as are immunocompromised individuals (e.g., individuals suffering from the human immunodeficiency virus and cancer patients being treated with chemotherapy) (US EPA, 1995).

14.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

14.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

14.4.2 Non-Cancer Inhalation Toxicity Reference Values

The Canadian Environmental Protection Agency (CEPA, 1998) has established a series of Maximum Acceptable Levels for ground level NO₂ as part of their National Ambient Air Quality Objectives (NAAQOs). They present 1-hour guideline of 400 µg/m³, a 24-hour guideline of 200 µg/m³, and an annual guideline of 100 µg/m³, which were used in this assessment to evaluate acute-, intermediate-, and chronic-duration exposures, respectively. These guidelines are health-based and rely on controlled studies of the most sensitive population to NO₂ (i.e., asthmatics).

Table 14 summarizes the selected toxicity reference values

Table 14-1: Selected Toxicity Reference Values for NO_x

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	400 µg/m ³	Respiratory	CEPA
Inhalation – intermediate	200 µg/m ³	Respiratory	CEPA
Inhalation - chronic	100 µg/m ³	Respiratory	CEPA
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

14.5 References

- CEPA (Canadian Environmental Protection Agency). 1998. National Ambient Air Quality Objectives.
- OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.
- US EPA (United States Environmental Protection Agency). 1995. Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Technical Information. Office of Air Quality Planning and Standards, United States Environmental Protection Agency. September 1995. EPA-452/R-95-005. Available on-line at: http://www.epa.gov/ttn/naaqs/standards/nox/s_nox_pr_sp.html

15.0 POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

15.1 General Health Effects

Polycyclic aromatic hydrocarbons present a unique problem in the assessment of risk. The PAH family contains a large number of substances with a range of biological and chemical activities. PAHs are divided into a group of compounds with no apparent carcinogenic activity and a group of compounds that are carcinogenic. Reliable toxicity studies have been performed for relatively few PAH compounds; however, similar structures and chemical properties have led to a surrogate approach for estimating toxicity values (OMOE, 1997; HC, 1994d).

15.2 Assessment of Carcinogenicity

The Department of Health and Human Services (DHHS) has determined that some PAHs may reasonably be expected to be carcinogens. Likewise, the IARC (1983) has rated benzo(a)pyrene a Group 2A chemical, indicating that the agent (mixture) is probably carcinogenic to humans. The IARC also suggests that the exposure circumstance entails exposures that are probably carcinogenic to humans. Similarly, the US EPA attributes a rating of B2 to benzo(a)pyrene, indicating that it is considered a probable human carcinogen based on sufficient evidence of carcinogenicity in animals (USEPA 2004).

15.3 Susceptible Populations

No studies were located regarding unusual susceptibility of any human subpopulation to PAHs. However, mice that were fed high levels of one PAH during pregnancy had difficulty reproducing and so did their offspring. These offspring also had higher rates of birth defects and lower body weights (ATSDR, 1996). It is not known whether these effects occur in people.

15.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

15.4.1 Cancer Inhalation Toxicity Reference Values

Although there is strong evidence of carcinogenicity for several PAH compounds, only benzo(a)pyrene has reliable carcinogenic toxicity values. The most common method for estimating carcinogenic toxicity values for the other PAH compounds is the Toxicity Equivalency Factor (TEF) approach. It is assumed that the carcinogenic PAH compounds each have the same biological mechanism of action and biological end-point, but differ in their relative potencies or degrees of carcinogenicity. The US EPA (1993) used this approach to derive TEF values of 0.1, 0.01, 1.0, 0.1, 0.01, 0.1, 1.0, 0.001, 0.1, 0.001 and 0.001 for benz(a)anthracene, benzo(a)phenanthrene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, dibenzo(a,h)anthracene, Fluoranthene, indeno(1,2,3-c,d)pyrene, phenanthrene and pyrene, respectively.

It was necessary to conduct the TEQ methodology for assessing PAH toxicity as the OMOE only provides health based criteria for benzo(a)pyrene. The Health Canada inhalation unit risk value was used to derive a carcinogenic exposure value of $0.32 \mu\text{g}/\text{m}^3$ for risk of cancer of 1 in 100,000. The total annual toxicity equivalents were then summed and the total compared to the Health Canada-derived carcinogenic exposure value.

Health Canada has not developed toxicity values for the non-carcinogenic PAH.

Carcinogenic PAHs Toxic Equivalence Values

Substance	Toxic Equivalence	Toxicological Basis	Source Agency
Carcinogenic Effects			
Benzo(a)anthracene	0.1	TEF	Note 1
Benzo(a)phenanthrene	0.01	TEF	Note 1
Benzo(a)pyrene	1	Numerous Cancers	Health Canada (2003b)
Benzo(b)fluoranthene	0.1	TEF	Note 1

Substance	Toxic Equivalence	Toxicological Basis	Source Agency
Benzo(g,h,i)perylene	0.01		
Benzo(k)fluoranthene	0.1	TEF	Note 1
Dibenz(a,h)anthracene	1	TEF	Note 1
Fluoranthene	0.001	TEF	Note 1
Indeno(1,2,3-c,d) pyrene	0.1	TEF	Note 1
Phenanthrene	0.001	TEF	Note 1
Pyrene	0.001	TEF	Note 1

Note 1. Values are based on the relative potency scale from the US EPA (1993) as discussed in text.

Table 15-1: Selected Toxicity Reference Values for PAHs - Based on B(a)P

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	NA	NA	NA
Inhalation - chronic	NA	NA	NA
Cancer Effects			
Inhalation - chronic	0.32 $\mu\text{g}/\text{m}^3$	NA	Health Canada 2003

Notes:

NA: Not Applicable

15.5 References

ATSDR 1996 Agency for Toxic Substances and Disease Registry. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Available on-line at: <http://www.atsdr.cdc.gov/tfacts90.html>

IARC 1983. International Agency for Research on Cancer (IARC) Monographs Programme on the Evaluation of Carcinogenic Risks to Humans, Polycyclic Aromatic Hydrocarbons, Volume 32(1983) Suppl. 7 (1987), p.58.

U.S. EPA (Environmental Protection Agency). 2004. Integrated Risk Information System (IRIS) Database – Benzo(a)pyrene. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

16.0 SULPHUR DIOXIDE

16.1 General Health Effects

Exposure to low levels of sulphur dioxide can lead to sore throat and an increase in airway resistance (ATSDR, 1997).

16.2 Assessment of Carcinogenicity

There are no studies that clearly show carcinogenic effects of sulphur dioxide in people (ASTDR, 1998). Sulphur dioxide was tested for carcinogenicity in mice by inhalation exposure, and results indicated a significant incidence of lung tumors in females (IARC, 1997). The International Agency for Research on Cancer (IARC) has classified SO₂ as Group 3, not classifiable to human carcinogenicity.

16.3 Susceptible Populations

Exercising asthmatics are recognized as the most susceptible group to SO₂ inhalation (ASTDR, 1998). Elderly adults with pre-existing respiratory or cardiovascular disease may be susceptible to the increased risk of mortality associated with acute-duration exposure to SO₂ (ASTDR, 1998). Children may be particularly susceptible to increased frequencies of respiratory illness following chronic-duration exposure to SO₂ (ASTDR, 1998).

16.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

16.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

16.4.2 Non-Cancer Acute Inhalation Toxicity Reference Values

The Canadian Environmental Protection Agency (CEPA, 1998) has established a series of Maximum Acceptable Levels for ground level SO₂ as part of their National Ambient Air Quality Objectives (NAAQOs). They present 1-hour guideline of 875 µg/m³, a 24-hour guideline of 301 µg/m³, and an annual guideline of 60 µg/m³, which were used in this assessment to evaluate acute-, intermediate-, and chronic-duration exposures, respectively.

Table 15 summarizes the selected toxicity reference values for Sulphur Dioxide.

Table 16-1: Selected Toxicity Reference Values for Sulphur Dioxide

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	875 µg/m ³	Respiratory	CEPA
Inhalation – intermediate	301 µg/m ³	Respiratory	CEPA
Inhalation - chronic	60 µg/m ³	Respiratory	CEPA
Cancer Effects			
Inhalation	NA	NA	NA

Notes:

NA: Not Applicable

16.5 References

ASTDR (Agency for Toxic Substances and Disease Registry). 1998. Toxicological Profile for Sulfur Dioxide. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. December 1998. 185 pages + Appendices. Available on-line at: <http://www.atsdr.cdc.gov/>

CEPA (Canadian Environmental Protection Agency). 1998. National Ambient Air Quality Objectives.

IARC (International Agency for Research on Cancer). 1997. Search of the IARC Agents and Summary Evaluations, Monographs Programme on the Evaluation of Carcinogenic Risks to Humans. Available on-line at: <http://www-cie.iarc.fr/htdocs/monographs/vol54/02-sulfur-dioxide.htm>

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

17.0 SULPHURIC ACID

17.1 General Health Effects

According to the ATSDR (1999), sulphuric acid can cause burns to the skin, eyes, lungs, and digestive tract, while severe exposure can result in death. Breathing sulphuric acid can result in tooth erosion and respiratory tract irritation (ATSDR, 1999).

17.2 Assessment of Carcinogenicity

The International Agency for Research on Cancer (IARC) has determined that occupational exposure to strong inorganic acid mists containing sulphuric acid is carcinogenic to humans. The IARC has not classified pure sulphuric acid for its carcinogenic effects (ATSDR, 1999).

17.3 Susceptible Populations

Persons with asthma, smokers, and persons exposed to second-hand smoke may be more sensitive to sulphuric acid exposure (Holma 1985; Holma 1989; Albert et al. 1971). Other persons with compromised lung function would also be more sensitive to inhalation exposure to sulphuric acid aerosols (Albert et al. 1971). Children may also have increased sensitivity to sulphuric acid in air, due to their smaller airway diameters and limited buffering capacity, resulting from incomplete development of mucous and mucosa (Holma et al. 1985).

17.4 Selection of Toxicity Values

As the POI Limit and AAQC established by the Ontario MOE (OMOE, 2001) are based on corrosion concerns, health-based TRVs were determined by searching the following sources, in order of preference: Health Canada, US EPA's IRIS, ATSDR, and WHO. The following section details the toxicity reference values selected, their basis, and source.

17.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

17.4.2 Non-Cancer Inhalation Toxicity Reference Values

The Cal-EPA (2004) has published a chronic inhalation reference exposure level of $1.0 \mu\text{g}/\text{m}^3$, based on a study by Alarie et al. (1973), which identified a LOAEL for chronic exposure to sulphuric acid of $380 \mu\text{g}/\text{m}^3$. Alarie et al. (1973) observed bronchiolar epithelial hyperplasia, and thickening of the bronchial walls in monkeys.

Table 16 summarizes the selected toxicity reference values for sulphuric acid.

Table 17-1: Selected Toxicity Reference Values for Sulphuric Acid

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	NA	NA	NA
Inhalation - chronic	$1.0 \mu\text{g}/\text{m}^3$	Respiratory	Cal-EPA
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

17.5 References

Alarie Y, Busey WM, Krumm AA, Ulrich CE, and Va V. 1973. Long-term continuous exposure to sulfuric acid mist in cynomolgus monkeys and guinea pigs. Arch. Environ. Health 27:16-24.

Albert RE, Alessandro D, Lippmann M, et al. 197 1. Long-term smoking in the donkey. Arch Environ Health 22:12-19. Cited in: ATSDR, 1999.

ATSDR (Agency for Toxic Substances and Disease Registry). 1999. Toxicological Profile for Sufuric Acid. Available on-line at: <http://www.atsdr.cdc.gov>

Cal-EPA (California Environmental Protection Agency). 2001. Chronic Toxicity Summary: Sulfuric Acid. Office of Environmental Health and Hazard Assessment. Available online at:

http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html

Holma B. 1989. Effects of inhaled acids on airway mucus and its consequences for health. *Environ Health Perspect* 79:109-113. Cited in: ATSDR, 1999.

Holma B. 1985. Influence of buffer capacity and pH-dependent rheological properties of respiratory mucus on health effects due to acidic pollution. *Sci Total Environ* 41: 101-123. Cited in: ATSDR, 1999.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

18.0 TOLUENE

18.1 General Health Effects

Inhalation exposure of humans to toluene, has resulted primarily in neurological effects such as headache, fatigue, decreased manual dexterity. Chronic exposure to toluene can lead to subtle changes in neurological functions including cognitive and neuromuscular performance, hearing and colour discrimination (ATSDR, 2000).

18.2 Assessment of Carcinogenicity

The IARC classifies toluene as a Group 3 carcinogen (the agent is not classifiable as to its carcinogenicity to humans) based on inadequate evidence in humans for carcinogenicity of toluene and evidence suggesting lack of carcinogenicity of toluene in experimental animals. The US EPA has classified toluene in Group D - not classifiable as to human carcinogenicity (US EPA, 1992). Toluene has been classified as Group IV-C – probably not carcinogenic to man under the classification scheme developed by the Bureau of Chemical Hazards (Environment Canada and Health Canada, 1992).

18.3 Susceptible Populations

Environmental or genetic factors that decrease the capacity for metabolic detoxification of toluene are likely to increase susceptibility (ASTDR, 2000). Nutritional status may also affect susceptibility to toluene (ASTDR, 2000). Individuals with pre-existing medical conditions, such as defects in heart rhythm, asthma, or other respiratory difficulties, may be more susceptible to the effects of toluene (ASTDR, 2000).

18.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

18.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

18.4.2 Non-Cancer Inhalation Toxicity Reference Values

ATSDR has derived an acute-duration inhalation MRL of 3,770 $\mu\text{g}/\text{m}^3$ for toluene based on a NOAEL of 40 ppm (151,000 $\mu\text{g}/\text{m}^3$) for neurological effects in humans (Andersen et al., 1983). This is considered a final MRL.

The US EPA (1994) has published an RfC of 400 $\mu\text{g}/\text{m}^3$ based on neurological effects reported in an occupational study by Foo et al. (1990). The RfC is based on a $\text{LOAEL}_{\text{HEC}}$ of 119 mg/m^3 and an uncertainty factor of 300; 10 intraspecies variability, 10 for conversion of LOAEL to NOAEL, 3 for database deficiencies.

The US EPA (1994) states that overall confidence in this RfC is medium. Although Health Canada (1996) has developed a Tolerable Concentration of 3,800 $\mu\text{g}/\text{m}^3$ for chronic exposure to toluene based upon the results of animal studies that indicated a decreased body weight in mice (Huff, 1990), the US EPA RfC was selected for use in this assessment as it is significantly more conservative and appears to be better supported by the rationale provided.

18.5 Conclusion

Table 17 summarizes the selected toxicity reference values for Toluene.

Table 18-1: Selected Toxicity Reference Values for Toluene

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	3,770 $\mu\text{g}/\text{m}^3$	Neurological	ATSDR
Inhalation - intermediate	NA	NA	NA
Inhalation - chronic	400 $\mu\text{g}/\text{m}^3$	Respiratory	Health Canada
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

18.6 References

- Andersen, I, Lundqvist, GR, Molhave, L, et al. 1983. Human response to controlled levels of toluene in six-hour exposures. *Scand J Work Environ Health* 9:405-418.
- ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Toluene. ATSDR/U.S. Public Health Service Public Health Service Available on-line at: <http://www.atsdr.cdc.gov>
- Environment Canada and Health Canada. 1992. Canadian Environmental Protection Act, Priority Substances List Assessment Report, Toluene. Government of Canada, Environment Canada, Health Canada.
- Foo, S.C., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. *Br. J. Ind. Med.* 47(7): 480-484
- Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances. Canadian Environmental Protection Act. Priority Substances Supporting Documentation. August 1996.
- Huff, J. 1990. NTP (National Toxicology Program). Toxicology and Carcinogenesis Studies of Toluene in F344/N rats and B6C3F1 mice. Technical Report Series No. 371, National Toxicology Program, US Department of Health and Human Services, Public Health Service, National Institutes of Health, Research Triangle Park, NC. Cited In: Environment Canada and Health Canada, 1992.
- OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.
- U.S. EPA (Environmental Protection Agency). 1994. Integrated Risk Information System (IRIS) Database - Toluene. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

19.0 TRICHLOROETHANE-1,1,2

19.1 General Health Effects

There have been no studies located regarding health effects in humans following the inhalation of 1,1,2-Trichloroethane (ATSDR, 1989).

19.2 Assessment of Carcinogenicity

Increased incidence of hepatocellular carcinomas and pheochromocytomas in one strain of mice (US EPA, 1994). 1,1,2-Trichloroethane is structurally related to 1,2-Dichloroethane, a probable human carcinogen. The US EPA has classified 1,1,2-Trichloroethane in Group C, a possible human carcinogen (US EPA, 1994).

19.3 Susceptible Populations

Persons with diabetes (Hanasono et al. 1975) or with prior exposure to polybrominated biphenyls (Kluwe et al. 1978) or with prior exposure to isopropyl or ethyl alcohol or acetone (Traiger and Plaa, 1974) may be more susceptible to the hepatotoxic effects of 1,1,2-Trichloroethane. Prior exposure to other enzyme inducing drugs or chemicals could potentially have the same effect (ATSDR, 1989).

19.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

19.4.1 Cancer Inhalation Toxicity Reference Values

The US EPA (1994) has developed a unit risk value of $0.000016 (\mu\text{g}/\text{m}^3)^{-1}$, which was based on extrapolations from oral exposure data showing hepatocellular carcinoma in mice. This translates into a carcinogenic exposure value of $0.6 \mu\text{g}/\text{m}^3$ for a 1 in 100,000 risk level.

19.4.2 Non-Cancer Inhalation Toxicity Reference Values

There are no published non-cancer TRVs for 1,1,2-Trichloroethane via an inhalation route of exposure.

Table 18 summarizes the selected toxicity reference values for 1,1,2-Trichloroethane.

Table 19-1: Selected Toxicity Reference Values for 1,1,2-Trichloroethane

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	NA	NA	NA
Inhalation - intermediate	NA	NA	NA
Inhalation - chronic	NA	NA	NA
Cancer Effects			
Inhalation - chronic	0.6 µg/m ³	Hepatocellular carcinoma	US EPA

Notes:

NA: Not Applicable

19.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. Toxicological Profile for 1,1,2-Trichloroethane. Available on-line at: <http://www.atsdr.cdc.gov>

Hanasono GK, Witschi H, Plaa GL. 1975. Potentiation of the hepatotoxic responses to chemicals in alloxan-diabetic rats. Proc Soc Exp Biol Med 149: 903-907. Cited In: ATSDR, 1989.

Kluwe WM, McCormack KM, Hook JB. 1978. Potentiation of hepatic and renal toxicity of various compounds by prior exposure to polybrominated biphenyls. Environ Health Perspect. 23: 241-246. Cited In: ATSDR, 1989.

Traiger GJ, Plaa GL. 1974. Chlorinated hydrocarbon toxicity: Potentiation by isopropyl alcohol and acetone. Arch Environ Health. 28:276-278. Cited In: ATSDR, 1989.

U.S. EPA (Environmental Protection Agency). 1994. Integrated Risk Information System (IRIS) Database – 1,1,2-Trichloroethane. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

20.0 XYLENES

20.1 General Health Effects

Chronic exposure of humans to mixed xylenes, as seen in occupational settings, has resulted primarily in neurological effects such as headache, dizziness, fatigue, tremors, incoordination, anxiety, impaired short-term memory, and inability to concentrate (ATSDR, 1995). Labored breathing, impaired pulmonary function, increased heart palpitation, severe chest pain, abnormal EKG, and possible effects on the kidneys have also been reported (ATSDR, 1995). Mixed xylenes have not been extensively tested for chronic effects, although animal studies show effects on the liver and central nervous system from inhalation and oral exposures (ATSDR, 1995).

20.2 Assessment of Carcinogenicity

No information is available on the carcinogenic effects of mixed xylenes in humans (ATSDR, 1995). The IARC has classified mixed xylenes in Group 3 – cannot be classified as to its carcinogenicity to humans. The US EPA has classified mixed xylenes as a Group D, not classifiable as to human carcinogenicity (US EPA, 2003a).

20.3 Susceptible Populations

Available data indicate that pregnant women, fetuses, and very young children may be at greater risk of adverse health effects from xylenes than the general population (ATSDR, 1995). People with subclinical and clinical epilepsy are at increased risk of seizures if exposed to xylene because of its excitatory effects on the central nervous system (ATSDR, 1995). People with renal, hepatic, or cardiac disease may also be more susceptible to the toxic effects of xylene (ATSDR, 1995).

20.4 Selection of Toxicity Values

The following section details the toxicity reference values selected, their basis, and source.

20.4.1 Cancer Inhalation Toxicity Reference Values

There are no published TRVs for cancer via an inhalation route of exposure.

20.4.2 Non-Cancer Inhalation Toxicity Reference Values

The ATSDR has published an acute-duration MRL of 4,340 $\mu\text{g}/\text{m}^3$, derived for mixed xylenes, and based on an LOAEL of 100 ppm (434,000 $\mu\text{g}/\text{m}^3$) for prolonged reaction times (Dudek et al. 1990). This is considered a final MRL.

An intermediate MRL of 3,040 $\mu\text{g}/\text{m}^3$ is also presented by ATSDR, based on a LOAEL of 200 ppm (868,000 $\mu\text{g}/\text{m}^3$) for decreased rotarod performance (Hass and Jakobsen 1993). This is considered a final MRL.

Health Canada (1996) has developed a Tolerable Concentration of 180 $\mu\text{g}/\text{m}^3$ for chronic exposure to xylene isomers, based upon the results of studies of rats exposed to xylenes via inhalation in a teratogenicity study.

Table 19 summarizes the selected toxicity reference values for xylenes.

Table 20-1: Selected Toxicity Reference Values for Xylenes

Route of Exposure	TRV	Toxicological Basis	Source Agency
Non-Cancer Effects			
Inhalation - acute	4,340 $\mu\text{g}/\text{m}^3$	Neurological	ATSDR
Inhalation - intermediate	3,040 $\mu\text{g}/\text{m}^3$	Developmental	ATSDR
Inhalation - chronic	180 $\mu\text{g}/\text{m}^3$		Health Canada
Cancer Effects			
Inhalation - chronic	NA	NA	NA

Notes:

NA: Not Applicable

20.5 References

ATSDR (Agency for Toxic Substances and Disease Registry). 1995. Toxicological Profile for Total Xylenes. ATSDR/U.S. Public Health Service Available on-line at: <http://www.atsdr.cdc.gov>

Health Canada. 1996. Health-Based Tolerable Daily Intakes/Concentrations and Tumourigenic Doses/Concentrations for Priority Substances. Canadian Environmental Protection Act. Priority Substances Supporting Documentation. August 1996.

OMOE (Ontario Ministry of the Environment). 2001. Summary of Impingement Standards, Point of Impingement Guidelines, and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of Environment, September 2001.

U.S. EPA (Environmental Protection Agency). 2003a. Integrated Risk Information System (IRIS) Database - Xylenes. Confirmed current as of August 2004. Available on-line at: <http://www.epa.gov/iris/>

U.S. EPA (Environmental Protection Agency). 2003b. Toxicological review of xylenes (CAS No. 1330-20-7). In support of summary on the integrated risk information system. National Center for Environmental Assessment, Washington, DC. Available online at: <http://www.epa.gov/iris>

21.0 PARTICULATE MATTER – PM₁₀, PM_{2.5}

21.1 General Health Effects

PM₁₀ and PM_{2.5} refer to particulate matter that is 10 microns or less in diameter, and particulate matter that is 2.5 microns or less in diameter, respectively. Environmental contaminants in air exist primarily as fine suspended particulate matter. When inhaled, some fraction of the larger particles (i.e., greater than 10 microns in diameter) is deposited in the airways or lungs, and the rest is exhaled. Finer particles tend to penetrate into the alveoli. While some soluble compounds may be absorbed from the airways or lungs, the major site of absorption is the alveoli.

Particulate matter can produce immediate, short term effects from direct exposure from air. Typical symptoms can include eye, mucous membranes in the nose, skin, and respiratory tract irritation. For this reason, the short term concentration in air to which a person is exposed is considered to be the most important and generally accepted method of evaluating potential health effects (USEPA, 1996).

The WHO (2000) has published a summary of estimates of relative increase in daily mortality, respiratory hospital admissions, reporting of bronchiodialator use, cough and lower respiratory symptoms, and changes in peak expiratory flow associated with a 10 µg/m³ increase in PM₁₀ and PM_{2.5}, as reported in studies in which PM₁₀ and PM_{2.5} were actually measured and not inferred from other measures. For illustrative purposes, the WHO also presents estimates of the effects of a 3-day episode with daily PM₁₀ concentrations averaging 50µg/m³ and 100 µg/m³ on a population of 100 000.

21.2 Assessment of Carcinogenicity

No reference to the carcinogenicity of PM_{2.5} or PM₁₀ was found.

21.3 Susceptible Populations

Epidemiological studies indicate that the elderly, children, and people with chronic lung disease, influenza, or asthma, are especially sensitive to the effects of particulate matter (CEPA/FAC, 1999).

21.4 Selection of Toxicity Reference Values (TRV)

The following section details the toxicity reference values selected, their basis, and source.

21.4.1 Non- Cancer Inhalation Toxicity Reference Values

The WHO states that although the weight of evidence from numerous epidemiological studies points clearly and consistently to associations between concentrations of particulate matter and adverse health effects at low levels of exposure. The database does not enable the derivation of specific guidelines at present. However the CCME (2000) has published a Canada-Wide Standards criterion for particulate matter, based on PM_{2.5}, which is recognized as having the greatest effect on human health. The CCME CWS criterion for PM_{2.5} is 30 µg/m³, 24 hour averaging time. In addition, the B.C. Ambient Air Quality Objective for PM₁₀ of 50 µg/m³, 24 hour averaging time was used.

21.5 References

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