Toxikos
Toxicology Consultants

REPORT

VISY PULP AND PAPER MILL HEALTH RISK ASSESSMENT

Visy Pulp and Paper Pty Ltd

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Pacific Environment Limited

Consulting • Technologies • Monitoring • Toxicology

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ES1 EXECUTIVE SUMMARY

Visy operates a Pulp & Paper Mill near Tumut, NSW that underwent expansion in September 2009 to increase operations from 300,000 tonnes per annum (tpa) to 700,000 tpa. The expansion constituted the installation of processing equipment and a second stack. The operation site is characterised by complex terrain and is located in an area of relatively close to potentially sensitive receptors.

Toxikos has been commissioned to conduct a health risk assessment (HRA) of the emissions resulting from the expanded mill.

The HRA was conducted using methodology consistent with enHealth guidance; it evaluates the possibility of toxicological effects on humans from inhalation exposure. Ground level concentrations (GLCs) of emission components were predicted at discrete locations around the mill by air dispersion modelling conducted by PAE Holmes and provided to Toxikos. In addition to the places where people may reside or congregate, Toxikos was supplied with GLCs at the locations within the air dispersion modelling domain.

For each of the above scenarios an assessment was conducted to estimate the potential acute and chronic health risks (non-carcinogenic) and carcinogenic risks for individuals in the area. The former assessments were conducted by comparing the predicted ground level concentrations to a health-based air guideline established by a competent regulatory authority. The ratio of these two values is termed the hazard quotient (HQ). In the context of this HRA, different chemical categories (criteria pollutants, metals, acids, TRS and PAHs and dioxins) were evaluated in terms of examining a hazard index (HI), which is simply the sum of all HQs for each of the emission components in each of the categories is the hazard index (HI). Using this approach is conservative and provides a high level of certainty in the outcome of the HRA. If the HQ and HI are less than unity then it is concluded that there are no unacceptable risks to human health for exposed individuals. The converse is not automatically true, however. That is, when levels of exposure result in an HQ or HI greater than unity, adverse health outcomes are not necessarily expected. Rather, there is erosion in the margin of safety between the level of exposure and the level of exposure known to cause adverse effects. Under such a situation, it is prudent to re-examine the basis of all of the assumptions used to generate the estimates of risk and exposure. The following table provides the components of mill emissions provided via modelled GLCs.

Criteria pollutants	Chlorine substances	Other components
Sulphur dioxide (SO_2)	Chlorine (Cl_2)	Dioxins (as TEQ)
Nitrogen dioxide (NO_2)	Chlorine dioxide (ClO_2)	VOCs (total)
Particulate matter (PM_{10})	Hydrogen chloride (HCl)	TRS
		Metals

At all of the discrete receptor locations the HQs and HIs were less then unity, indicating little likelihood for acute or chronic health effects. Lifetime cancer risks, calculated using the methodology of the US EPA, at all discrete receptor locations was less than one in a million, indicating that no unacceptable risks to human health are likely present.

In the HRA, a screening process was established to determine whether discharges of metals to air by the Visy factory required a detailed multi-media risk assessment for human health risks from exposure from soil and food. Trigger criteria were developed based on soil health investigation guideline levels and whether the predicted GLCs were greater than those recorded for remote or rural areas, and if the chronic HQ for direct inhalation effects was greater than 0.05. None of the metals evaluated passed the screening process to trigger the detailed risk assessment.

Toxikos also performed a preliminary evaluation for health risks for dioxins that might be associated with secondary exposure, via food, to mill emissions released to air. Discharge of dioxins was assessed by calculating total monthly intake of dioxin toxicity equivalents (TEQ) assuming current upper bound background intake for Australians and a steady state relationship of 1:99 for inhalation:food intake for mill emissions that was derived from review of the scientific literature. The calculations, using the highest predicted annual average GLC within the air dispersion modelling domain resulted in a monthly intake that was approximately 3 times less than the tolerable intake established by the National Health and Medical Research Council of Australia and the World Health Organisation. More than 99% of the dioxin TEQ intake was due to current background intakes (from food). It is concluded secondary exposure pathways for the dioxin emissions, even in the long term, do not represent a significant risk to the local populace.

Overall, it is concluded that air emissions from the proposed expansion of the Visy mill at Tumut present little likelihood of causing adverse health effects to exposed individuals in the nearby areas.

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Acronyms and Abbreviations

ACGIH	American Conference of Governmental Industrial Hygienists
AGV	Air Guideline Value
Ah	Aryl hydrocarbon
AQM	Air Quality Monitoring
ATSDR	Agency for Toxic Substances and Disease Register
Av.	Averaging
Ave	Average
Avg	Average
B(a)P	Benzo (a) pyrene
BCF	Biological Concentration Factors
bkgd	Background
CBD	Central Business District
CCF	Chronic Cardiac Failure
Cl_2	Chlorine
ClO_2	Chlorine dioxide
CNS	Central Nervous System
CO	Carbon monoxide
COAD	Chronic Obstructive Airway Disease
CoC	Chemical of Concern
COHb	Carboxyhaemoglobin
CoI	Chemicals of Interest
COPD	Chronic Obstructive Pulmonary Disease
DPIWE	Department of Primary Industry, Water and Environment
DMSO	Dimethyl sulphoxide
DNA	Deoxyribonucleic acid
E-XX	10^{-xx}
EA	Environment Agency
ECF	Elemental Chlorine Free
EROD	Ethoxyresorufin-O-deethylase activity
G-6-PD	Glucose-6-phosphate dehydrogenase
GLC	Ground Level Concentration
Visy	Visy Ltd
HAP	Hazardous Air Pollutants
H_2S	Hydrogen sulphide
HCl	Hydrogen chloride
HEC	Human Equivalent Concentration
HI	Hazard index
HQ	Hazard Quotient
hr	hour
HRA	Human Health Risk Assessment

HRA	Health Risk Assessment
HWC	Hazardous Waste Combustors
IARC	International Agency for Research on Cancer
IDLH	Immediately Dangerous to Life or Health
IIS	Integrated Impact Statement
inhal	inhalation
IPCS	International Program on Chemical Safety
IQ	Intelligent Quotient
IRIS	Integrated Risk Information System
LC_{50}	Lethal Concentration (50%)
LOAEL	Low Observed Adverse Effect Level
$\mu g/m^3$	microgram per metre cubed
MBI	Monthly Background Intake
MfE	Ministry for the Environment
MI	Monthly Intake
MMD	Mass Medium Diameter
MoE	Margin of Exposure
MoHW	Ministry of Health and Welfare
mRNA	Messenger Ribonucleic Acid
N/A	Not applicable
NAAQS	National Ambient Air Quality Standard
NATO	Northern Atlantic Treaty Organisation
NCG	Non Condensable Gas
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
ng/m^3	nanograms per metre cubed
NHMRC	National Health and Medical Research Council
NIOSH	National Institute for Occupational Safety and Health
NO_2	Nitrogen Dioxide
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NPI	National Pollution Inventory
NS	Not specified
OEL	Occupational Exposure Limit
OEHHA	Office of Environmental Health Hazard Assessment
PAH	Polyaromatic Hydrocarbon
Pb	Lead
PCB	Polychlorinated biphenyls
PCDD	Polychlorinated dibenzodioxin
PCDF	Polychlorinated dibenzofuran
$PM_{2.5}$	Particulate Matter less than 2.5µm in size
PM_{10}	Particulate Matter less than 10µm in size
ppb	parts per billion
ppm	parts per million
REL	Reference Exposure Level

RIVM	National Institute for Public Health and the Environment (Dutch)
RPDC	Resource Planning and Development Commission
SO_2	Sulphur Dioxide
TCDD	2,3,7,8-Tetrachlorodibenzodioxin
TDI	Tolerable Daily Intake
TEF	Total Equivalency Factor
TEQ	Toxic Equivalent
TGA	Therapeutic Goods Administration
TEQ/kg bw/d	Toxic Equivalent per kilogram body weight per day
THI	Target Hazard Index
TMI	Tolerable Monthly Intake
TRS	Total Reduced Sulphur
TWA	Time Weighted Average
UF	Uncertainty Factor
UK DoE	United Kingdom Department of Environment
URF	Unit Risk Factor
URT	Upper Respiratory Tract
US EPA	United States Environment Protection Authority
VOC	Volatile Organic Compounds
WHO	World Health Organization

Units

Concentrations of dioxins are expressed as picograms (pg) (generally expressed as TEQ) per gram (g) or kilogram (kg) of the medium (fish or sediment), or per litre (L) if water, or per cubic metre (m^3) if in air.

1 pg is equal to 1/1,000,000,000,000 (1×10^{-12}) of a gram (or one million millionth of a gram).

Because a kilogram is 1,000 grams, 1 pg is one thousand million millionth of a kilogram (or 1/1,000,000,000,000,000 or 1×10^{-15} kg).

1 pg/m^3 is 1×10^{-12} g/m^3.

Units – Conversion from ppm to µg/m^3

ppm and µg/m^3 are terms used to define the concentration of a chemical gas in air. Conversion between ppm and µg/m^3 is compound specific as it requires the molecular weight of the compound and the volume of air occupied by one mole of the gas (the molar volume). For this assessment the molar volume has been calculated at 25°C and 101.7kPa, and equates to 24.45. While slightly higher conversion factors are obtained using lower temperatures any error is minor compared to other uncertainties in the assessment.

Conversion between units of concentration

Concentration (mg/m^3) = Concentration (ppm) × (molecular weight of the compound)/(24.45)

Conversion between concentrations

X 1,000	ppm	parts per million	**X 1,000**	mg/m^3	milligrams per cubic metre
	ppb	parts per billion		µg/m^3	micrograms per cubic metre

1 INTRODUCTION

1.1 General

Visy operates a Pulp & Paper Mill near Tumut, NSW that underwent expansion in September 2009 to increase operations from 300,000 tonnes per annum (tpa) to 700,000 tpa. The expansion constituted the installation of processing equipment and a second stack. The operation site is characterised by complex terrain and is located in an area of relatively close to potentially sensitive receptors.

Visy has contracted Toxikos Pty Ltd to perform an independent human health risk assessment (HRA) of the emissions to air from the expanded Visy mill. The methodology adopted in the conduct of this HRA is consistent with the protocols and guidelines recommended by the Australian enHealth Council (**enHealth, 2004**).

Only normal operations (normal design loads) have been evaluated in this assessment; start-up, shutdown and process upset conditions have not been addressed.

1.2 What is a health risk assessment?

Health is defined by the World Health Organization (WHO) as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (**WHO, 1948**). Well-being is broadly described as an individual's self-assessment of their state of happiness, healthiness and prosperity. It relates to the quality of life and one's ability to enjoy it. There are many social and economic factors that impinge upon well-being.

The following are examples of determinants of health well-being (**enHealth 2002**, **NHC 2004**):

- Social and cultural factors (e.g. social support, participation, access to cultural resources).
- Economic factors (e.g. income levels, access to employment).
- Environmental factors (e.g. land use, air quality).
- Population-based services (e.g. health and disability services, leisure services).
- Individual/behavioural factors (e.g. physical activity, smoking).
- Biological factors (e.g. biological age).

According to enHealth (**2002**) all developments have a potential impact on health. Some will have positive health impacts by providing jobs, attracting health services to an area, and improving overall economic well-being of a community etc. Other projects may have negative impacts such as increased risk of disease, social disruption, increased noise etc. Many developments will have both positive and negative aspects. It is understood that the potential influence of the mill on local area economic factors, social disruption through changed traffic patterns and other such factors are not addressed in this document.

Air quality is one of the many parameters influencing well-being. This HRA seeks to evaluate whether the expanded operations will significantly affect air quality of the inhabited area around the mill and what the likelihood is for health effects in association to exposure to the emissions from the mill.

An assessment of the holistic nature of health as per the WHO definition is usually termed a 'Health Impact Assessment'. These are most often done once an impact has occurred and there is a need to identify the possible causes for mal-health. On the other hand, a health risk assessment is an analysis that uses information about substances to estimate a theoretical level of risk of an adverse health impact for people who might be exposed to defined levels of these substances. The information comes from established regulatory limits, scientific studies and measurement data of emissions.

Risk assessments are often conducted by considering possible or theoretical exposures predicted from air dispersion modelling of 'known' concentrations of emissions from a specific point of release at the industrial facility. As a conservative measure the risk assessment analysis, carefully considers the most vulnerable people (e.g. children, the sick and elderly) in order to ensure that all members of the public will be adequately protected.

The risk assessment helps answer common questions for people who might be exposed to hazardous compounds in the environment. In this case the following questions related to the emissions from the Visy mill were carefully considered:

- Under what circumstances might I and my family and neighbours be exposed to hazardous substances from this expansion?
- Is it possible we might be exposed to hazardous substances at levels higher than those determined to be safe?
- If the levels of hazardous substances are higher than regulatory standards, what are the health effects that might occur?

Additional information on the nature of the risk assessment performed on the mill emissions can be found in **Section 3**. Commensurate with common international practice, an exposure level at which adverse health effects are unlikely to be observed (e.g., acceptable risk) for the substance is taken to be the ambient air guideline established by a competent authority.

1.3 Scope of the risk assessment

The HRA is a useful tool for estimating the likelihood and severity of risks to human health, safety and the environment and for informing decisions about how to manage those risks. It is a document that assembles and synthesizes scientific information to determine whether a potential hazard exists and/or the extent of possible risk to human health.

Although this report describes certain technical aspects of the risk assessment, it does not address the important processes of risk management and risk communication.

This HRA addresses likely impacts on community health from exposure to emissions from the expanded operation of the mill. Due to the absence of other industries emitting similar substances as the Visy mill, background ambient air concentrations for substances emitted by the expanded mill do not need to be considered in the risk assessment.

The risk assessment considers potential health effects from acute (i.e. short-term) and chronic (i.e. long-term) exposures to substances in the emissions. Included in the latter are considerations for the potential increased cancer risks, as these risks are defined as an increased risk of developing cancer over a lifetime. An evaluation for odour and the potential impact of odour on health is not included in the scope of the HRA. The HRA therefore evaluates the potential of Visy mill emissions to cause adverse health effects in individuals who may be exposed either on a short-term, infrequent basis or on a long-term basis. Exposure of individuals to the Visy mill emissions is assumed to occur 24 hours per day for each day of the year for 70 years. Thus, from the aspect of chronic exposure assumptions the HRA is conservative (i.e. errs on the side of caution).

The risk assessment has been facilitated by the provision of spread sheet results for the emission dispersion modelling undertaken by PAEHolmes. These spread sheets contain predicted ground level concentrations (GLCs) of individual substances in the emissions at twenty discreet locations where people reside or gather.

2 THE VISY PULP AND PAPER MILL SETTING

2.1 General geography, demography and land use

The Visy Pulp and Paper Mill is located approximately 7 km west and east of the nearest townships of Tumut and Adelong respectively. The populations of Tumut and Adelong are approximately 6000 and 830 respectively. Occasional rural residences occur within the surrounding area, typically within the valley in which the site lies. The nearest residence, 'Woomera', is located approximately 1 km north-west of the Mill (refer to **Figure 4-2**)

The land in the vicinity of the site is zoned Zone No.1 (a) (Rural Zone), apart from the land to the north of the site, which is zoned Zone 1(f), Rural (State Forest Zone). The major economic activities in the area are timber production, manufacturing, retail, recreational tourism and activities associated with Snowy Mountains Hydro Electric Scheme. Land is also used for dry land farming including cropping and sheep and cattle grazing. Some dairy and stone fruit orchards are also present.

The site lies within a north-west to south-east aligned valley surrounded by steep slopes that rise approximately 400 m above the intervening valley. The mountain ranges lie between Adelong to the west and Tumut to the east. The valley floor is occupied by Sandy Creek which drains toward the south-east into Gilmore Creek and ultimately the Tumut River; the confluence of Sandy Creek and Gilmore Creek is located approximately 3 km south-west of Tumut. Sandy Creek is located approximately 900 m south-west of the site. Surface waters are abstracted for stock watering purposes in the Tumut region. Surface waters may also be abstracted for irrigation. Approximately 110 hectares of land at 'Gadara Park' is irrigated with treated wastewater from the plant to grow crops for grazing and harvesting for hay and silage.

2.2 Meteorology

The climate in the vicinity of the site is characterised by cool winters and warm summers. At the Bureau of Meteorology weather station at Adelong, the average minimum and maximum temperatures measured between 1883 and 2004 were 12.9°C and 30.8°C in January (the warmest month) and 0.9°C and 12.5°C in July (the coolest month). Average rainfall at Adelong is 791.3 mm, with June being the wettest month (monthly average rainfall 84.6 mm) and February being the driest (monthly average rainfall 42.2 mm). There are between 100 and 150 frosts per year and occasional snow.

Wind conditions at the site are mostly calm, with winds mostly occurring at night time, associated with cold drainage from the surrounding mountains. At Tumut between 1974 and 1975, calm conditions prevailed on average 65% of the time in the morning and 43% of the time in the afternoon. On a small scale winds would be largely affected by the local topography. At larger scales, winds are affected by synoptic scale winds, which are modified by a complex pattern of regional drainage flows that develop overnight.

Average annual 9 am and 3 pm wind speeds measured at Adelong are 4.3 km/hr and 8.8 km/hr respectively. Wind strength is lowest in the winter months when the average monthly 9 am and 3 pm wind speeds are 1.4 km/hr and 5.5 km/hr respectively (June and May respectively). In summer, the average monthly 9 am and 3 pm wind speeds peak at 7.8 km/hr and 12.4 km/hr respectively (December and November respectively).

3 RISK ASSESSMENT METHODOLOGY

3.1 General overview

The overall methodology employed in this risk assessment is consistent with that of the Australian enHealth Council (**enHealth 2002**), the US Environmental Protection Agency (**US EPA 1989, 2000**) and the US Agency for Toxic Substances and Disease Registry (**ATSDR 1992a**).

The generic steps of a risk assessment are described in more detail in Sections 4, 5, and 6. The essential steps are:

- Toxicity assessment (hazard identification).
- Exposure assessment.
- Risk characterisation.

Although this risk assessment is quantitative, there are aspects that are primarily of a screening nature due to the fact that it deals with risks for a person who is hypothetically exposed to the highest atmospheric emission concentration that is reasonably expected to occur at nominated locations around the Visy mill.

The purpose of a screening risk assessment is to efficiently determine if, at the predicted exposures, health impacts are possible and if so discover the likely causative agents. Thus the risk assessment herein uses a number of procedures to decide which of the emission components either on their own or as a mixture are potential threats to public health and hence important for further detailed assessment.

The "screening procedure" is used to determine, using conservative (or worst-case) assumptions, whether a risk could exist, and whether the risk could be sufficiently serious to justify action. If the screening-level assessment indicates that a potential hazard is not of concern, a more comprehensive assessment is usually not required. However it may be undertaken in some circumstances for reasons other than health impact assessment. Conversely, if the screening-level assessment indicates that the potential hazard may be of concern, then the assessor may proceed to undertake a more comprehensive assessment to refine the potential estimates of the risk.

By necessity, to ensure protection of public health this risk assessment is conservative; that is, it errs on the side of caution by attempting to over predict the likelihood for health risk through the assumptions used relating to exposure. However to provide reality and contextual information in the assessment, a qualitative analysis has been undertaken for the uncertainty inherent in the assessment. Although aspects of uncertainty are discussed within the section where a particular topic is discussed, they are drawn together in **Section 10**.

This HRA evaluates the likelihood of non-cancer and cancer health effects arising from short- (i.e. acute) or long-term (i.e. chronic) exposures to emissions from the Visy mill.

International and Australian regulatory agencies consider an exposure level at which adverse health effects are unlikely to observed or present a negligible risk (e.g., acceptable risk) to be the same as, or less than, the relevant regulatory standard (i.e. an ambient air guideline value, AGV). Hence, by definition an unacceptable health risk potentially occurs when the predicted ground level concentration is greater than the regulatory standard. The process of characterising the health risk by comparing predicted ground level concentration to an AGV is common practice in risk assessments for air pollutants (**Morello-Frosh et al. 2000, Pratt et al. 2000, Tam and Neumann 2004**). It is a pragmatic approach used to identify important chemicals in polluted air or industrial emissions. The ratio of the GLC to AGV is called the 'hazard quotient' (HQ). By adding hazard quotients together to yield a hazard index (HI), an appreciation of the likelihood of an adverse health outcome from exposure to the emissions as a mixture can be obtained. The mechanics and interpretation of this method is described in **Sections 6.1 – 6.3**.

A schematic overview of the basics of the risk assessment process is in **Figure 3-1**.

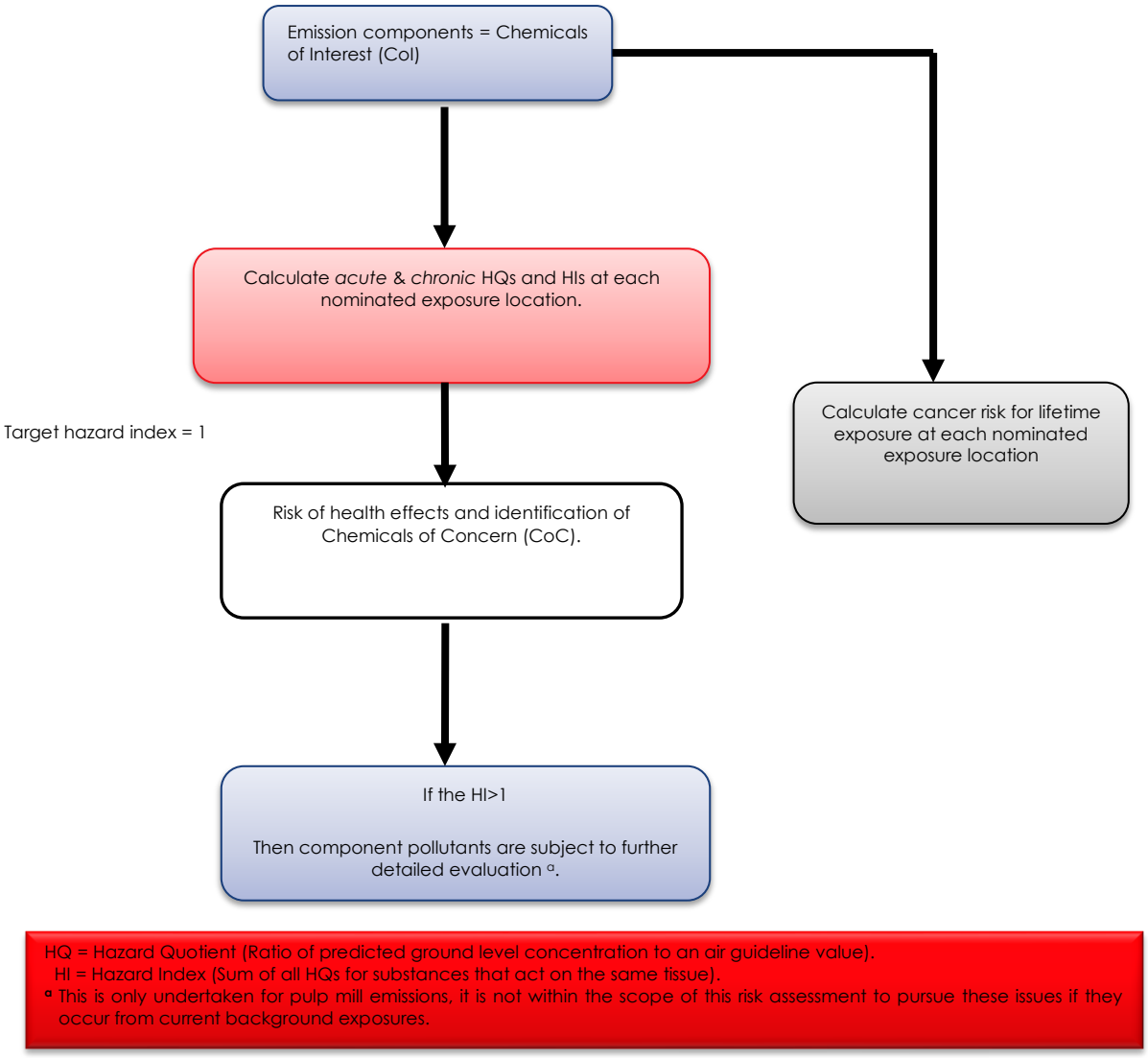

Figure 3-1: Overview of the basics of the screening risk assessment

3.2 Issue identification

With respect to the potential for health impacts, the basic issues associated with air emissions from the Visy mill have commonality with almost any new or proposed expansion of an industrial facility, which revolve around the following.

1 There may be community concerns that there may be new substances, currently not released into the air, which emitted in sufficient quantity may cause health effects not previously experienced by residents in the area.

2 People may be worried that the expansion may have increased emissions and cause, or exacerbate health effects.

4 EXPOSURE ASSESSMENT

4.1 Exposure pathways and exposure estimations

Exposure to substances in the emissions from the Visy mill may occur through a number of different environmental media (**Figure 4-1**). However, exposure to the emissions via inhalation is the primary subject of this HRA.

Exposure to components in the emissions may also theoretically occur via deposition directly onto food plants or soil which may be ingested by humans and/or food producing animals. Additionally, there is the potential for root uptake by plants. Some contaminants in soil are taken up by plants via root uptake or deposited onto plants can theoretically be bioaccumulated by the plants, or animals that feed upon them and are used as food sources for humans. However, over the last few decades it has been appreciated that only relatively few chemicals have the potential to present significant health risks to humans as a result of accumulation through the food chain. Such chemicals have a common set of chemical and biological properties: they are poorly degraded in the environment and hence have long environmental half-lives, they are only slowly metabolised and excreted by animals and humans and hence have long biological half lives in the body, and they are highly soluble in fat which together with the poor metabolism means they can accumulate and be stored in fatty tissue. If this storage in the body is significant, body burdens of the chemical may reach a level where toxicity might occur. Organic chemicals in emissions from the Visy mill that have these properties are dioxins/furans, but these will be released in very small amounts and do not pose a risk to human health (see **Section 8**).

Estimation of exposure to emissions at any given receptor location relies upon:

1 Determination of what is in the emissions.

2 Determination of the concentration of emission components at the point of release to the atmosphere (performed by the mill design engineers).

3 Dispersion modelling to predict the 'ground level concentration' of the contaminant at locations where people may live or spend appreciable amounts of time

These aspects are discussed in more detail in the following sections.

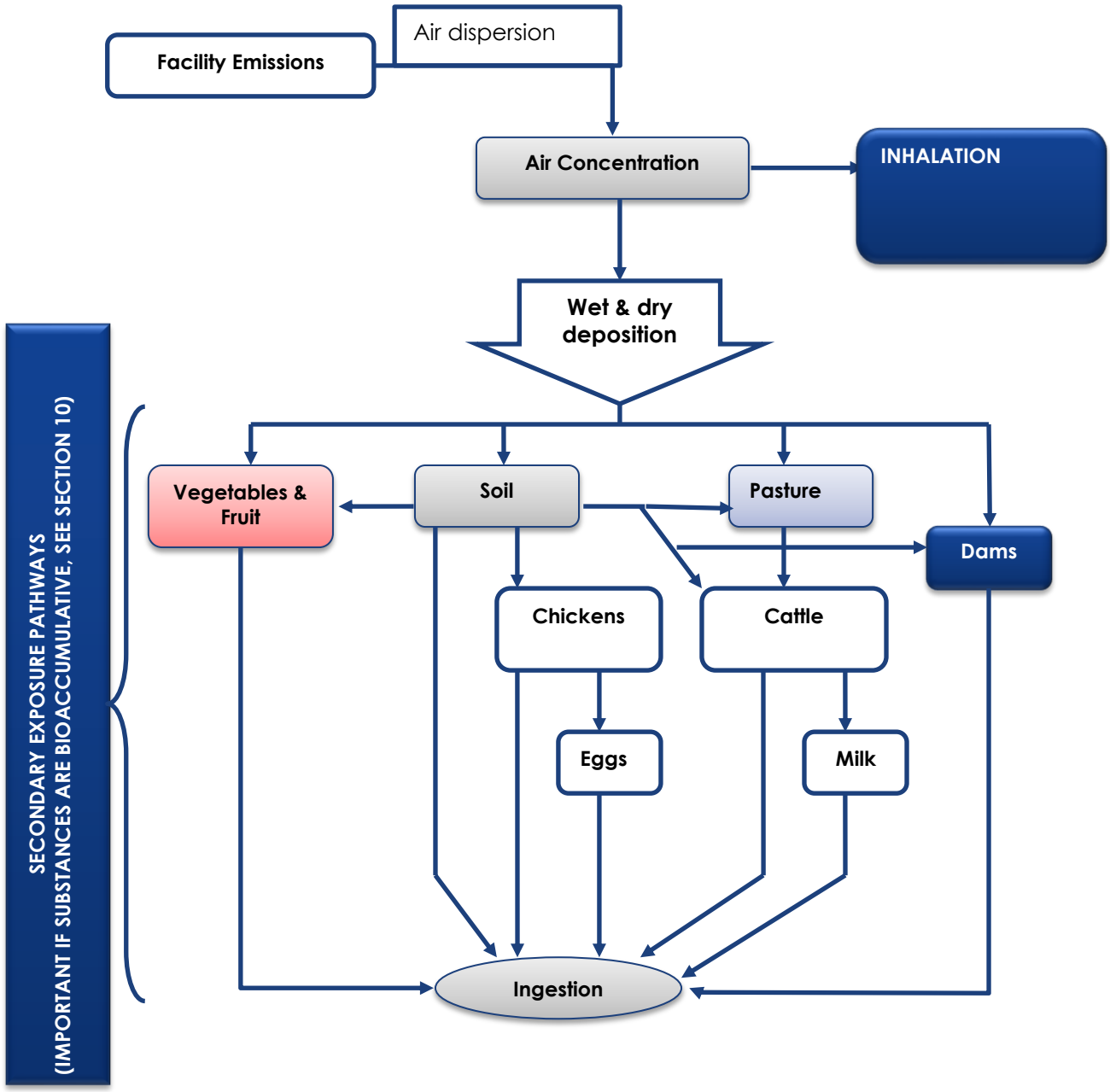

Figure 4-1: Theoretical exposure pathways to air emissions

4.2 What is in the emissions?

Table 4-1 provides the components of mill emissions provided via modelled GLCs.

Table 4-1: Components of mill emissions modelled for prediction of ground level concentrations[a]

Criteria pollutants	Chlorine substances	Other components
Sulphur dioxide (SO_2)	Chlorine (Cl_2)	Dioxins (as TEQ)
Nitrogen dioxide (NO_2)	Chlorine dioxide (ClO_2)	VOCs (total)
Particulate matter (PM_{10})	Hydrogen chloride (HCl)	TRS
		Metals

[a] Depending on the substance, GLC were provided for the maximum, 99.9th, 99.5th and 99th percentile for averaging times that mostly matched the health guidelines for the compounds. Annual average GLCs were also supplied.

4.2.1 Where are people exposed?

The air dispersion modelling conducted by PAEHolmes (**2012**) provided probability estimates for ground level concentration frequencies (percentiles) at discrete locations. In risk assessment terminology the locations are called receptors. The choice of locations was determined by Visy and PAEHolmes on the basis they were either within a radius of 5 km of the mill.

The post-expansion monitoring data utilised included ambient air quality monitoring, periodic stack monitoring, continuous emissions monitoring system (CEMS) and meteorological data. To both provide estimation of actual operating conditions, and to provide realistic inputs into a subsequent Health Risk Assessment, the verification utilises average emission rates from periodic stack monitoring data, in combination with CEMS data, where available.

Local residents were interviewed for an air quality assessment undertaken for Visy in late 2003. It was reported that neighbours had observed the plume from Visy's main stack to impact on areas to the north of the mill generally during times of poor dispersion, early in the morning and late at night. It was also reported by a resident of 'Pleasant View' that the plume was sometimes observed to travel up the valley to the north-west and affect residences further up the valley.

Residents to the south reported that odours from Visy were detectable under prevailing wind conditions. It was considered in the study that odour impacts on the high ground to the north of the plant were probably from the stack emissions, whilst odour impacts to the south were probably from multiple sources.

The nearest sensitive receptor to the Visy site is the residence 'Woomera', located approximately 1 km north-west of the Mill. The residence 'Pleasant View' is located 1.8 km north-west of the site and 'Gadara Park' (owned and occupied by Visy) is located approximately 1.4 km south-east of the site. There are 22 residences within a 5 km radius of the site. A list of these residences are presented in **Table 4-2**. The nearest populous centres are Tumut and Adelong, located approximately 7 km east and west of the site respectively.

Sensitive receptors listed in Tumut include housing, a hospital, eight schools/TAFEs (including one infant's school), two aged care facilities, a swimming pool, seven churches, 11 parks/recreational facilities and a library. In addition, a town common - wetlands and nature reserve are present on the northern and southern outskirts of Tumut respectively. A golf course is also located on the western outskirts of Tumut, approximately 6 km east of the site.

Sensitive receptors in Adelong included housing, a swimming pool, two parks/recreational facilities and two schools/TAFEs.

Receptor locations are shown on a codified map in **Figure 4-2**.

Table 4-2: Receptors for which air dispersion for prediction of ground level concentrations of emissions was conducted.

Receptor Number	Description
1	Havilah
2	Pleasant View
3	Minjary
4	Reka
5	Woomera
6	Whispering Pines
7	Deep Creek
8	Glengarry
9	Glenroy Park
9a	M Bradley
10	The Lagoon
11	B&K Gentle
12	Moonapinna
13	S Bevan
14	Willow Bend
19	R&C Beale
21	J Adams
22	Bradley & Whiting
26	Adelong Main Street
27	Tumut Main Street

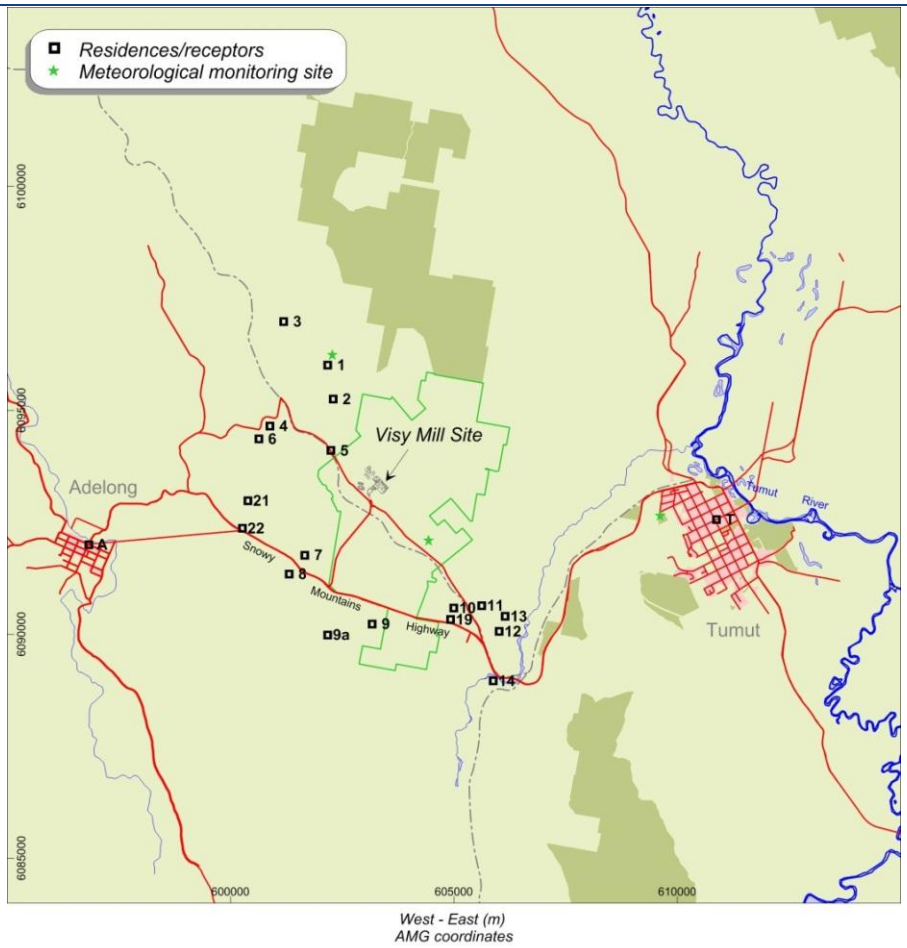

Figure 4-2: Location of the site with receptor locations and identification labels

4.2.2 How are potential exposures determined?

To main factors determine the extent to which people are exposed to substances in the mill emissions; how much is in the air and the behaviour of the person.

4.2.2.1 How much is in the air?

Concentrations of compounds in the air from point industrial sources are not constant; the concentration varies according to the direction and strength of the wind, time of day, how far away the location is from the emission source, activities being conducted at the source location etc.

The ground level concentrations were determined using the air dispersion model, CALPUFF. CALPUFF (Scire, et al., 2000) is a non-steady state puff dispersion model that can simulate the effects of time and space varying meteorological conditions on emissions transport, transformation and removal. The model contains algorithms for near-source effects such as building downwash, partial plume penetration, sub-grid scale interactions as well as longer-range effects such as substance removal, chemical transformation, vertical wind shear and coastal interaction effects. The model uses dispersion equations based on a Gaussian distribution of substances across the puff and takes into account the complex arrangement of emissions from a variety of source types (i.e. stack sources, fugitive emissions). A variable emissions file can be generated for input into

CALPUFF that adequately describes the emissions as they relate to the production rate specific to the expanded mill.

As with any air dispersion model, CALPUFF requires inputs in three major areas:

- Emission rates and source details;
- Local meteorology; and
- Terrain and surface details, as well as specification of specific receptor locations.

Predicted ground level concentrations for specific averaging periods can be output from the model for every grid point within the modelled domain and at any defined receptor location.

The dispersion modelling provides a statistical probability for the number of averaging periods in a year the concentration of a pollutant will be at a certain level. The concentrations of most interest are the high ones because these are the ones most likely to affect people. If the high concentrations are less than the exposures needed to cause a health effect then it logically follows the lower ones will also not be of health concern. The output of the dispersion modelling is a list of air concentrations and how often they occur during a year of typical meteorology. These are expressed as percentiles, and can be pragmatically regarded as code for the number of times a concentration will occur during the year, or the number of times during the year a person is likely to be exposed to a certain concentration if they are at the same spot at the same time the high concentration occurs. **Table 4-3** provides the key to the code. Only the high percentile concentrations are used in the risk assessment. These represent maximum or near maximum exposures. A summary of the modelling methodology used by PAEHolmes as reported (**2012**) is provided in the following sections.

Table 4-3: Frequency percentiles and number of times they occur for a particular averaging time.

	Frequency Percentile					
	95th	99th	99.5th	99.7th	99.9th	Max
Approx. number of times[a] per year a 1 hour average concentration might occur at a given percentile.	438	88	44	27	9	1
Approx. number of times[a] per year a 24 hour average concentration might occur at a given percentile.	19	4	2	1	1	1

[a] The predicted ground level concentrations of the various chemical of interest for the risk assessment are provided in Appendix C for each of the percentiles at each receptor location. This information shows how rapidly the concentration decreases from the maximum down to the 95th percentile. The number of times a percentile will occur has been rounded up to a whole number. For example the concentration at the 99.9th percentile for the 1 hour and 24 hour average occurs 8.76 and 0.36 times.

4.2.3 Modelling Methodology

This assessment was prepared in accordance with the NSW Approved Methods for the Modelling and Assessment of Air Pollutants in NSW (hereafter, "the Approved Methods", **EPA, 2005**). The approach used to verify the previous modelling is consistent with the HAS Air Report unless stated. The approach uses CALMET wind fields, TAPM upper air data and CALPUFF dispersion modelling. For consistency, an equivalent meteorological input file (calendar year 2005) was used in the current assessment as that described within the HAS Air Report. The modelling approach is outlined below and in greater detail in the previous assessments (**HAS, 2007a; 2007c**).

4.2.3.1 Meteorological Modelling

The CALPUFF atmospheric dispersion model makes use of wind fields generated by the CALMET meteorological model. CALMET generates a three-dimensional wind field on an hourly basis by taking observations of winds at selected location and interpolating these to produce information on wind speed and direction at a grid of regularly spaced points covering the area of interest. Modifications that are imposed on this interpolated wind field (by topography and differential heating and differential surface roughness) are then applied to the winds at each grid point to develop a final wind field.

Meteorological observations have been collected from three sites in the area, referred to as Point 5, Point 7 and Point 16. In addition, Visy commissioned Monash University to conduct boundary layer measurements by Monash University during 2005 to further characterise the local meteorology.

The 2005 meteorological dataset was used for this assessment to be consistent with the HAS Air and Odour Report and the amended modelling assessment (**HAS 2007a; 2007b; 2007c**). Further discussion of the CALMET model grid, meteorological station and terrain information can be found in Figure 5 of the HAS Air Report. For further details on the parameters used to create the meteorological field, refer to Table 3 of the previous assessment. Refer to the previous assessment for Annual and seasonal wind roses for Point 5 at the site (Figure 6 to Figure 9), ground-level wind patterns simulated by CALMET (Figure 10), meteorology simulated by CALMET at the surrounding site (Figure 11), wind speed and temperature profiles simulated by CALMET (Figure 12) and a histogram of surface temperature inversion depth as simulated by CALMET (Figure 13).

This CALMET dataset was further validated in a letter report where a comparison is made between 2002 and 2005 meteorological data (**HAS, 2007d**). The assessment concluded that while 2005 has a greater proportion of calms, these were unlikely to be a dominant factor in determining predicted maximum ground-level concentrations.

4.2.3.2 Dispersion Modelling

The CALPUFF atmospheric dispersion modelling system is the US EPA's preferred model for assessment of long range pollutant transport and for near field applications with complex meteorology. A domain of 20km by 20km around the facility was used for modelling impacts.

Information to characterise the influence of building downwash were included in the model in the form of building profile information, consistent with the HAS Air and Odour Reports. Only normal operations (normal design loads) have been evaluated in this assessment; start-up, shutdown and process upset conditions have not been quantitatively addressed.

4.2.3.3 Behaviour of the person

To factor a person's behaviour (i.e. average daily movements) into a risk assessment is quite challenging, and is rarely done. Instead, an assumption is made that throughout their entire life a person is in a situation where they could be exposed to the highest concentrations predicted to occur by the dispersion modelling. This assumption adds conservatism (i.e. safety) into the risk assessment. Based on this assumption, whether or not a person is affected by a compound in air from an industrial source requires them to be present at the location at the same time the high concentration occurs. However people do not spend all their time in one spot, for example an average adult only spends 1.5 hours outdoors per day (**US EPA 1997, EA 2003**). Given that people also move around during the time they spend outdoors, the chance of being present when a very high concentration of a compound from a point industrial source occurs only a few times per year is therefore quite low.

In contrast, the chance of being exposed is much more likely when the pollutant is from a number of sources in an area and the resultant pollution is spread over a wide area. This is what happens with the particulate matter from wood heaters in the cooler months of the year in Australian country towns, like Armidale and Launceston. The smoke from many wood heaters becomes trapped by an inversion layer in the air and cannot readily disperse. Instead it hangs in the air, and a relatively even concentration occurs over a wide area. In this situation, outdoor movement from one place to another does not decrease one's chance of being exposed.

5 HAZARD IDENTIFICATION/TOXICITY

11Appendix A contains very brief summaries, in tabular format, on the primary health hazard associated with emission components. Included is information associated with the sensitive endpoint upon which the air guideline value was set, and a sketch overview on how the air guideline value was established.

The information in **11Appendix A**, which summarises the potential health effects of each of the emission components, has been confined to identifying the broad toxicological endpoint categories for each chemical (e.g. carcinogenicity, genotoxicity, reproductive effects, central nervous system depression (e.g., narcosis), respiratory tract effects etc.). These may not necessarily be the health effects of concern for which the relevant health guideline has been established. Because this is a preliminary hazard assessment, review documents and electronic databases produced by competent agencies [α] have been used as information resources rather than conducting a thorough toxicological evaluation for each compound. The information in **11Appendix A** does not take into consideration the exposures necessary to cause the noted health effect that has led to the categorisation. Consequently, although a competent authority, or independent scientific review, may consider the substance of being capable of causing the effect at some level of exposure, in reality exposures may never be high enough for it to be realised.

Because overview documents or electronic databases have been used to determine the hazard category of emission components no assessment has been made regarding dose response aspects, or whether the toxicological effects used to categorise the potential hazard have a realistic probability of being realised at the exposure levels in question for the scenarios evaluated herein. That is, there has been no evaluation to determine the exposures required for different effects to be elicited for a given chemical. General toxicological knowledge shows that for many of the compounds the doses required causing, say, liver toxicity are much higher than the dose required to cause the most sensitive health effect (which forms the basis of the relevant guideline). For example, the acute or chronic health guideline for a particular chemical may be based on irritancy or perhaps central nervous system depression because this is the most sensitive health end point, but at higher concentrations some other effect may occur that another agency has used to classify the compound as possessing a particular hazard capability if the exposure is high enough. Thus because public health guidelines are based on the most sensitive effect, usually the effect that occurs at the lowest concentration, comparison of modelled ground level concentrations with the guideline will automatically take into account effects that may occur at higher exposures of the compound.

There are a number of substances provided in the modelled GLCs that are known human carcinogens, or are regarded by the International Agency for Research on Cancer (IARC) as being probable or possible human carcinogens(e.g., Arsenic, cadmium, chromium^{VI}, nickel and PAHs). For these substances it is commonly regarded for regulatory purposes that any exposure , no matter how small equates to some increased risk of cancer over an individual's lifetime. The level of this risk is calculated for each carcinogenic substance identified in the modelled GLCs and described in **Section 7.3**.

[α] National Environment Protection Council (NEPC), Australia; World Health Organization (WHO)- International Programme for Chemical Safety (IPCS) & International Agency for Research on Cancer (IARC); Agency for Toxic Substances and Disease Registry (ATSDR), US Dept Health & Human Services; Office of Environmental Health Hazard Assessment (OEHHA), California EPA; The Dutch National Institute of Public Health and the Environment (RIVM); and the Integrated Risk Information System (IRIS), US EPA. Wherever it has been practical to do so, the hierarchal preferred reference list of enHealth (2002) has been used to source guidelines.

6 METHOD FOR CHARACTERISING RISK

6.1 'Chemical classes' within the emissions:

For the purposes of the risk assessment, components of the emissions have been grouped into 'chemical classes'; these are not necessarily groups of compounds that have the same chemical properties but rather those that may broadly have the potential for interactive (additive) health effects. This approach has been taken as a conservative assessment of the potential additive risks from exposure to each of these groups of compounds. While not specifically supported from a toxicological perspective (e.g., all compounds within each group do not necessarily exhibit the same toxicological endpoint), this approach provides a conservative estimate of the potential risks to exposed individuals. As such, with this level of conservativism, there is a higher confidence in the conclusions of the risk assessment.

The chemical class groupings are:

- Criteria pollutants: SO_2, NO_2, CO, PM_{10} (taken to be TSP).
- Metals: Sb, As, Be, Cd, Cr, Co, Cu, Pb, Mn, Hg, Ni, Se, Sn, V.
- Acids: Cl_2, HCl, HF, H_2SO_4.
- TRS: Evaluated as H_2S.
- PAHs and dioxins.

6.2 Introduction to hazard quotients and the hazard index

Hazard quotient (HQ) = ground concentration ÷ air standard

Hazard index (HI) = sum of all hazard quotients

Conservatively assumes additive health effects

For assessing the potential non-cancer health impact of individual chemicals, predicted ground level concentrations are compared to individual health-based ambient air guidelines generated to protect public health. This comparison is performed by calculating a hazard quotient[b] (HQ) which is the ratio of GLC to the ambient AGV[c].

The hazard quotient is calculated for each contaminant using the simple equation below.

HQ = GLC/AGV .....**Equation 1**

For assessing the potential effects of the mixture of chemicals within each of the identified chemical groupings in the emissions, it has been assumed individual components may have additive effects and an overall hazard index (HI) has been calculated (**US EPA 2000a**). The HI is the sum of all the emission component hazard quotients determined from either the acute or chronic air guideline values, thus an acute and a chronic hazard index can be generated.

[b] Some investigators call the 'hazard quotient' the 'hazard ratio' (e.g. Fox et al. 2004, Tam et al. 2004).

[c] The hazard quotient is commonly reported to one significant figure (US EPA 1989). For example, a hazard quotient of 0.13 is rounded to 0.1, and a hazard quotient of 1.6 is rounded to 2. In this risk assessment HQs and HIs have been calculated to one decimal place. This is not to imply there is a level of precision in the assessment; far from it, it has been done merely to allow proper accounting of the summing of HQ's in this report.

$$HIj = \sum HQ\ i \ldots j \quad \text{.....Equation 2}$$

Where HIj is the sum of HQ's for all emitted compounds from i to j

This process assumes:

- There is a threshold level of exposure below which no adverse health effects will occur.
- Either the toxicological effect of chemicals and/or the dose is additive.
- Multiple sub threshold exposures may result in an adverse health effect.

As noted previously, in toxicological terms it is only valid to sum the effects and/or dose of chemicals if they have the same mode of toxicological action and affect the same target tissues . Similarly it would not be expected for substances in a mixture to have interactive health impacts if they were individually present at concentrations significantly below their biological threshold levels (i.e. below their true low observed effect level) . Some investigators therefore prefer only to sum hazard quotients for pollutants that effect common organs, thus yielding effect-specific cumulative HIs (**Fox et al. 2004, Morello-Frosch et al. 2000**). Others, while recognising that adding HQs with different health end points will not give an accurate idea of the non-cancer HI nonetheless add all HQs together (**Pratt et al. 2000**). Some investigators limit this latter practice to only those pollutants whose HQ is greater than unity (**Tam and Neumann 2004**) (i.e. for substances whose concentrations may be exceeding guidelines and perhaps nearing their biological thresholds).

Most health based guidelines inherently contain safety factors to assure protection against ill health being caused by exposure to the chemical. If the guideline has been established using animal toxicological data then there is usually a safety factor of at least 100, sometimes 1,000 or more, that has been applied to the exposure that does not cause effects in animals (i.e. to the No Observed Effect Level = NOEL), i.e. guideline usually = NOEL/100, if human data has been used the safety factor may be anywhere between 3 - 100. Thus the hazard index is not an evaluation predicting whether health effects will/will not occur, but rather whether the health guideline value will/will not be exceeded. If the health guideline is not exceeded then it follows that health effects are very unlikely to occur, if the health guideline is exceeded it does not naturally follow that health effects will occur. This is because of the conservatism embedded in the exposure estimate (i.e. the numerator of equation 1 which is the modelled GLC) and the uncertainty (safety factors) used to establish the health guideline value (i.e. the denominator of equation 1). The uncertainty factors used in the derivation of the health based air guideline value by competent agencies is included in Appendix 1 of this risk assessment, this information provides an appreciation of the margin between the AGV and the exposure that may actually be required to cause an effect.

Chemicals can have more than one toxicological effect but often require different levels of exposure for the different effects to become apparent. However it is impractical to determine the dose effect(s) relationships for all effects of all emission components. Hence it is difficult to identify with confidence all the substances that will have common sites of toxicological action. We have therefore adopted the pragmatic approach, regardless of the mode of toxicological action or site of adverse health effect, of generating overall acute and chronic non-cancer hazard indices for all chemicals of concern, as if they were acting in concert on the same tissues. If the resulting composite HI is greater than the 'target' hazard index (THI, see Section 6.2 and Attachment 3) then the pollutants significantly contributing to the HI are examined in more detail to determine whether or not there is biological plausibility for the additive effects assumed in the calculation of the HI. At this stage of assessment interrogation the dose effect(s) relationships may be also examined.

It should be noted however that many of the substances modelled to be emitted from the mill have the respiratory tract as the primary target organ. It is therefore appropriate that they be considered as potentially having interactive effects. Indeed the priority pollutants (SO_2, NO_2 and PM_{10}) are known to have interactive effects, with NO_2 making the bronchi of asthmatics more reactive to other bronchoconstrictors.

6.3 Interpretation of hazard quotients and indices

An 'unacceptable' risk, as defined by regulatory standards and requirements, is often determined as the exposure being greater than the AGV used to calculate the hazard quotient, i.e. the HQ>1. This definition of unacceptable risk does not equate with imminent adverse health effects or even high risk of adverse health effects. It simply means that the health guideline level has been exceeded.

The common practice of summing the HQs of all chemicals in screening (i.e. preliminary) risk assessments, regardless of biological mode of action or target tissue grossly overestimates the risk estimation for systemic health effects from exposure to the emission mixture of chemicals. It is not unreasonable to assume additive effects for pollutants that have direct effects on airways function.

Notwithstanding their use in this risk assessment, HQs and HIs are relatively blunt tools used to assist in characterising and prioritising risks. Care must be taken as to the level of importance that is placed on the numerical value of the HI. HIs should not be used in isolation of other pertinent data such as mechanistic information on the toxic mode of action and knowledge of the conservatism incorporated into the exposure assessment and the toxicity values.

The HI calculation focuses on components that are likely contributors to health risks either because their individual exposure levels exceed health guidelines, or because joint mechanisms of action with other components may pose a health hazard. Generally mixture components whose HQs are less than 0.1 (HQ<0.1) are considered unlikely to pose a health hazard due to interactions, and unless there are a relatively large number of components that act similarly, are not likely to pose an increased hazard due to additivity (**ATSDR 2001a**). The general rule of thumb for interpreting a HQ and HI is that values less than 1 present no cause for concern; values greater than 1 but less than 10 generally also do not represent cause for concern because of the inherent conservatism embedded in the exposure portions of a preliminary risk assessment. However, it is usual to examine, and perhaps refine, the level of conservatism that has been assumed in the exposure assumptions. HQs and HIs that are around 10 present some concern regarding possible health risks, and in these circumstances it is usual to evaluate the extent to which the "safety margins" in the health guideline value used to compare estimated exposures may have been eroded in order to gauge whether concern is warranted. It is common that the risk assessment needs to be refined using site specific exposure information or additional analytical data when HIs are greater than unity.

6.4 Calculating cancer risk

The lifetime risk of developing cancer for exposure to a carcinogen is calculated by multiplying the average lifetime chemical exposure by an estimate of the carcinogenic potency of the chemical. The latter is commonly called the unit risk factor, or slope factor. For air borne carcinogens, the "unit" is generally 1 µg/m^3 and depending on the nature of the data used to determine the carcinogenic potency, the numerical value refers to the probability of developing cancer. Thus a lifetime exposure to 1 µg/m^3 of a substance may carry a risk of 1 chance in 200 of developing cancer; this is often interpreted as meaning, if 200 people were exposed to 1 µg/m^3 for their lifetime then one individual may develop cancer. This probability is expressed as 0.5 in 100, or 0.5 x 10^{-2} per µg/m^3, written as 0.5 x 10^{-2} (µg/m^3)$^{-1}$. The target acceptable risk band adopted in many countries is 1 x 10^{-6} to 1 x 10^{-5}, i.e. with a lifetime exposure there is a chance developing a tumour between one in a million and one in one hundred thousand.

Lifetime cancer risk = lifetime average air concentration (µg/m^3) x unit risk factor (µg/m^3) $^{-1}$

= AC (µg/m^3) x UR (µg/m^3) $^{-1}$ .....**Equation 3**

In this risk assessment literature values of carcinogenic potency have been used without any attempt to evaluate the veracity of the potency value. Where several unit risk values are in the literature, the value indicative of the highest potency has been used except where there is appropriate precedence for either an Australian authority or the WHO using a different value for deriving a standard, in which case the latter has been used in the risk assessment.

It is common practice to assume cancer risks due to different genotoxic carcinogenic air pollutants is additive. Summing the individual cancer risks is used to estimate a total lifetime risk of developing cancer (**Morello-Frosch et al. 2000, Tam and Neumann 2004, Pratt et al. 2000**). However unit risk estimates are upper bound 95% confidence estimates and do not reflect the central tendency or average. When several upper bound estimates are added together, a question is raised as to whether the predicted cancer risk is plausible. The greater the number of carcinogens being considered the more unlikely, in theory, the true risk for each carcinogen will lie near the upper bound estimate. The process of adding upper bound cancer risk estimates together is inherently conservative. Cogliano (**1997**) has shown that the resulting risk estimate becomes increasingly improbable the greater the number of risk estimates, but is not necessarily misleading. However, to obtain a cancer risk estimate closer to the true risk Cogliano (**1997**) considers central estimates to be more plausible. Unfortunately, central estimate unit risk factors are not readily available. Additionally, based on the weight of evidence from the available literature it is likely that exposure to multiple carcinogenic compounds results in an additive (or synergetic) increase in the potential risk of developing cancer. However, this may not always hold true in cases where the mode of action of each of the carcinogens is the same, such as in the case of PAHs.

6.5 Consideration of background exposures

It is usual to include background exposures when assessing health risks to industrial emissions.

As has been mentioned previously there is not another significant point source of emissions in the immediate area that is creating a 'background' level. Consequently evaluation of 'cumulative' effects with background air concentrations of pollutants has not been undertaken,

Omission of background considerations in this risk assessment is likely to be an issue only if the calculated HI is approaching unity, this would signify there is not much room in the overall hazard index to accommodate background airborne substances. However, since the calculated hazard indices are below unity (i.e. HI<< 1) (**Section 7**) it is unlikely incorporation of background information into the risk assessment will materially change the conclusion of the assessment.

7 RISK CHARACTERISATION

7.1 Potential acute health effects

Appendix C contains a summary of all the acute HIs at each percentile ground level concentration for all receptors. The potential for acute health effects have been assessed for modelled emissions and for representative locations.

Acute hazard indices are all below unity, indicating that acute direct health effects are unlikely.

Table 7-1 and **Figure 7-1** show the acute HIs for all percentiles of predicted ground level concentrations of substances in the Visy emissions. All the HIs are less than unity, even for the 100th percentile which is only expected to occur once per year. This indicates acute direct health effects are unlikely.

Table 7-1: Acute HIs for all percentile ground level concentrations for representative receptors

Receptor	Percentile			
	100th	99.9th	99.5th	99th
1	0.46	0.19	0.08	0.04
2	0.51	0.19	0.08	0.05
3	0.30	0.11	0.05	0.03
4	0.45	0.17	0.08	0.05
5	0.67	0.31	0.13	0.08
6	0.43	0.18	0.09	0.06
7	0.52	0.15	0.06	0.04
8	0.64	0.14	0.06	0.04
9	0.17	0.06	0.03	0.02
9a	0.24	0.09	0.04	0.02
10	0.18	0.06	0.03	0.02
11	0.20	0.07	0.04	0.02
12	0.15	0.07	0.03	0.02
13	0.23	0.07	0.03	0.02
14	0.11	0.04	0.02	0.01
19	0.14	0.06	0.03	0.02
21	0.38	0.16	0.07	0.05
22	0.54	0.13	0.06	0.04
26	0.17	0.08	0.04	0.02
27	0.13	0.05	0.02	0.01

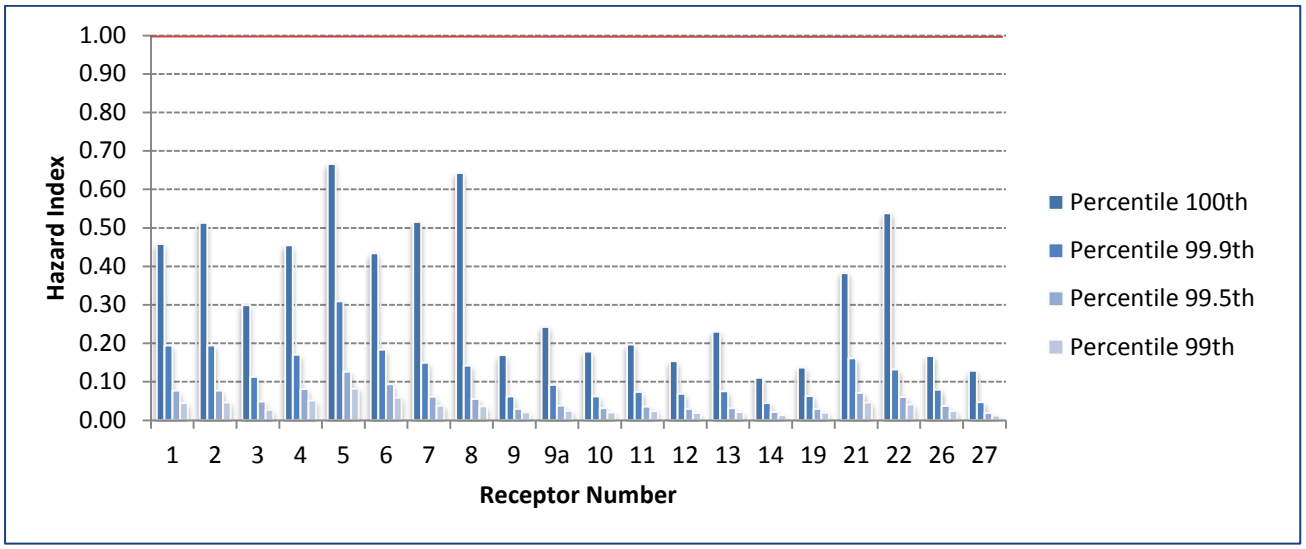

Figure 7-1: Acute Hazard Indices for each percentile

Table 7-2 shows the contribution, at the 99.5th percentile, of the major chemical classes present in the Visy emissions to the HIs shown in **Table 7-1**. The relative contribution of the chemical classes to the HI is the same across all percentiles.

Table 7-2: Acute 99.5th percentile hazard indices for classes of chemicals and total hazard index for each receptor

	Chemical class				
Receptor	Criteria pollutants	Metals	Acids	TRS	**Total hazard index**
1	0.04	0.001	0.03	0.007	0.08
2	0.03	0.002	0.04	0.005	0.08
3	0.02	0.001	0.03	0.003	0.05
4	0.03	0.002	0.04	0.008	0.08
5	0.03	0.003	0.07	0.017	0.13
6	0.04	0.002	0.04	0.007	0.09
7	0.02	0.002	0.03	0.004	0.06
8	0.02	0.001	0.03	0.003	0.06
9	0.01	0.001	0.02	0.002	0.03
9a	0.01	0.001	0.02	0.002	0.04
10	0.01	0.001	0.02	0.001	0.03
11	0.01	0.001	0.02	0.002	0.04
12	0.01	0.001	0.02	0.001	0.03
13	0.01	0.001	0.02	0.001	0.03
14	0.01	0.001	0.01	0.001	0.02
19	0.01	0.001	0.02	0.001	0.03
21	0.03	0.001	0.03	0.006	0.07
22	0.03	0.001	0.03	0.003	0.06
26	0.02	0.001	0.02	0.002	0.04
27	0.01	0.000	0.01	0.001	0.02

7.2 Potential chronic health effects

7.2.1 Non-cancer health effects

Table 7-3 contains the HIs for the selected representative receptors. **Figure 7-2** summarises the chronic hazard quotients and indices for all scenarios and locations assessed around the Visy mill.

Table 7-3: Hazard indices for chronic health effects

Receptor	Chemical class				Total hazard index
	Criteria pollutants	Metals	Acids	TRS	
1	0.009	0.004	0.13	0.006	0.15
2	0.015	0.006	0.17	0.004	0.20
3	0.005	0.003	0.10	0.003	0.11
4	0.011	0.006	0.18	0.010	0.20
5	0.022	0.011	0.34	0.018	0.39
6	0.012	0.006	0.20	0.007	0.22
7	0.009	0.005	0.15	0.003	0.17
8	0.008	0.005	0.14	0.003	0.16
9	0.004	0.003	0.08	0.002	0.09
9a	0.006	0.003	0.10	0.002	0.11
10	0.004	0.003	0.08	0.001	0.09
11	0.005	0.003	0.09	0.002	0.10
12	0.004	0.003	0.08	0.001	0.09
13	0.005	0.003	0.09	0.002	0.10
14	0.003	0.002	0.05	0.001	0.06
19	0.004	0.002	0.07	0.001	0.08
21	0.010	0.005	0.16	0.007	0.19
22	0.009	0.005	0.16	0.004	0.18
26	0.005	0.003	0.09	0.003	0.10
27	0.003	0.002	0.05	0.001	0.05

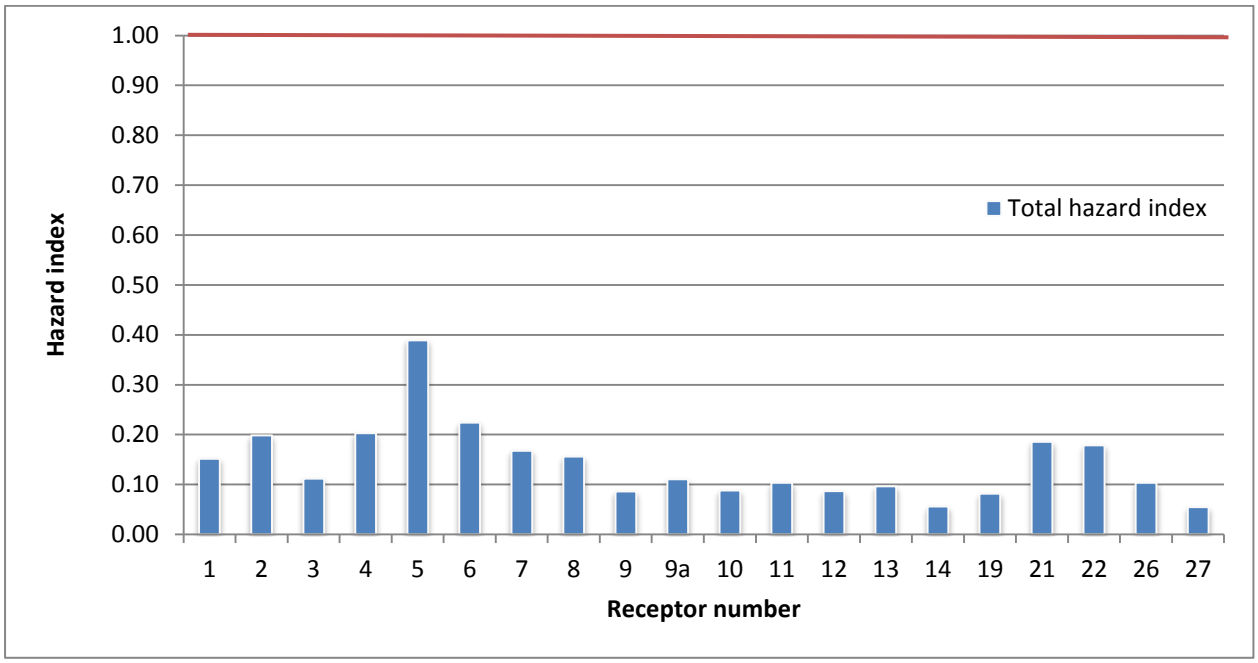

Figure 7-2: Chronic hazard indices for all receptors

7.3 Cancer risks

The methodology for calculating cancer risks is described in **Section 0**. **Table 7-4** contains the carcinogenic risks for the individual compounds and also the total cancer risk, assuming additivity between substances.

Overall the emissions from the mill expansion do not pose a significant carcinogenic risk to persons living around the Visy mill.

The highest calculated total cancer risk[d] is approximately 1.6 x 10^{-7} (**Table 7-4**). This is below the commonly accepted risk band of between one in one million and one in one hundred thousand[e].

[d] Although all of the substances may produce tumours of the lung they don't all necessarily act on the same cell type or by the same toxicological mechanism.

[e] To the best knowledge of Toxikos an official acceptable carcinogenic risk level for Australia has not been formally announced by any agency. In the US a risk of 1 in a million is regarded as being de minimus and is the risk level used by the Australian NHMRC for establishing drinking water guidelines for genotoxic carcinogens. However many of the risk assessment guideline documents for Australia recognise the level of carcinogenic risk deemed to be acceptable is a matter for the community as a whole or the community bearing the risk to decide. In New Zealand an incremental risk level of 1 in 100,000 per lifetime (1 x 10-5) is considered as being acceptable (NZ MfE 1997, 1999, NZ MoH 2000). This is a policy decision based on Ministry of Health deliberations for derivation of public health guidelines for New Zealand and the objective of protecting 'almost all' individuals. There are also examples in Australia where a lower risk level than 1 x10-6 has been used for evaluation of public health impacts or establishment of standards, for example the Air Toxics NEPM.

Table 7-4: Carcinogenic risk from genotoxic compounds for receptors

Receptor	As	Be	Cd	Cr (VI)	Ni	Total
1	3.0E-09	6.2E-09	2.2E-10	3.0E-08	6.9E-08	**1.1E-07**
2	4.1E-09	8.2E-09	3.0E-10	4.0E-08	9.5E-08	**1.5E-07**
3	2.1E-09	5.7E-09	1.7E-10	2.4E-08	5.4E-08	**8.6E-08**
4	4.1E-09	8.4E-09	3.0E-10	4.0E-08	9.5E-08	**1.5E-07**
5	8.0E-09	1.5E-08	5.7E-10	7.5E-08	1.8E-07	**2.8E-07**
6	4.6E-09	9.1E-09	3.3E-10	4.4E-08	1.1E-07	**1.6E-07**
7	3.1E-09	8.4E-09	2.5E-10	3.6E-08	8.0E-08	**1.3E-07**
8	2.9E-09	7.9E-09	2.3E-10	3.4E-08	7.5E-08	**1.2E-07**
9	1.6E-09	4.3E-09	1.3E-10	1.8E-08	4.1E-08	**6.5E-08**
9a	2.1E-09	5.5E-09	1.7E-10	2.4E-08	5.3E-08	**8.5E-08**
10	1.6E-09	4.6E-09	1.3E-10	1.9E-08	4.3E-08	**6.8E-08**
11	1.9E-09	5.3E-09	1.6E-10	2.2E-08	5.0E-08	**7.9E-08**
12	1.6E-09	4.5E-09	1.3E-10	1.9E-08	4.2E-08	**6.7E-08**
13	1.8E-09	4.9E-09	1.5E-10	2.1E-08	4.7E-08	**7.4E-08**
14	1.0E-09	2.9E-09	8.4E-11	1.2E-08	2.7E-08	**4.3E-08**
19	1.5E-09	4.2E-09	1.2E-10	1.8E-08	3.9E-08	**6.3E-08**
21	3.5E-09	8.1E-09	2.7E-10	3.7E-08	8.5E-08	**1.3E-07**
22	3.4E-09	8.6E-09	2.7E-10	3.8E-08	8.5E-08	**1.4E-07**
26	2.0E-09	4.9E-09	1.5E-10	2.2E-08	4.9E-08	**7.8E-08**
27	1.0E-09	2.8E-09	8.2E-11	1.2E-08	2.6E-08	**4.2E-08**

It is noted however that PAHs are not included in the cancer risk calculations as in-stack measurements were not undertaken and subsequently PAHs were not modelled by PAEHolmes, therefore no assessment could be undertaken. Not including PAHs consequently underestimates the calculated cancer risk.

In 2006, prior to expansion of the Visy mill, Toxikos undertook a HRA for the impact of emissions from the existing mill and the proposed expansions. At that time, Toxikos were provided in-stack concentrations for specific PAHs and a scaling factor from the then existing single stack, and data were supplied for the two stack situation for the expansion.

Nevertheless at the location with the highest predicted GLCs, the PAH cancer risk was 6.8×10^{-6} and within the acceptable cancer risk band. Given that the PAH cancer risk calculations are conservative and PAH concentrations will be lower at all other discrete receptor locations it is concluded the cancer risks at those locations are low.

The historic risk assessment for PAHs was conservative, as there are at least four factors that needed to be considered in interpreting the cancer calculations.

1 The fact that these are theoretical risks calculated using the upper 95th percentile estimation of the unit risk from linear multi-stage modelling of the dose response curves for benzo(a)pyrene. The actual cancer potency of benzo(a)pyrene is likely to be less than the value used in these calculations, and therefore the calculated risks probably overestimate the actual cancer risk.

2 It has been assumed all the PAHs released from the mill were as benzo(a)pyrene. This yields the most conservative calculations because of the different PAHs that will be released, benzo(a)pyrene is the most potent. In many instances where mixtures of PAHs have been experimentally assessed for carcinogenicity, there has been a decrease in carcinogenic response relative to the benzo(a)pyrene equivalents of the mixture because less potent PAHs prevent the metabolic activation of the more potent PAHs to their ultimate carcinogens and/or compete for binding with the aryl hydrocarbon (Ah) intracellular receptor (**ATSDR 1995, WHO 1998a**). The benzo(a)pyrene toxicity equivalence factors tend to overestimate the carcinogenic risks of PAH mixtures (**WHO 1998a**). However this is not always the case as there have also been instances where the benzo(a)pyrene equivalents approach has underestimated the experimental carcinogenicity of PAH mixtures because some PAHs in the mixture were either not identified or did not have a benzo(a)pyrene equivalence factor. In experiments where the PAH mixture has been defined and the all components have a benzo(a)pyrene equivalence factor, then the overall carcinogenicity potency of the mixture has generally shown additivity (**WHO 1998a**).

3 Most PAHs will be associated with particulates (**Bidleman 1988**). Modelling particulates as if they behaved like gases (i.e. assuming no gravitational loss during transport) will tend to overestimate ground level concentrations at receptors distant from the mill.

4 In-stack concentrations of PAHs after the proposed expansions are predicted to be below analytical detection limits for the individual PAHs. Toxikos has therefore assumed the concentrations may be at half the detection limit and calculated the cancer risks at the 'most affected anywhere' location accordingly. This assumption tends to overestimate the ground level concentrations and therefore the cancer risks.

Additionally, the highest predicted annual ground level concentrations of total PAH was approximately 5.0 x 10^{-4} µg/m^3, i.e. 0.4 ng/m^3. This concentration is within the range of rural background of 0.1 – 1 ng/m^3 reported by WHO (**1998a**). Since one does not equate rural background concentrations of PAH as presenting a significant carcinogenic risk to people this information provides a contextual benchmark for the cancer calculations

7.4 Conclusions for systemic health effects

7.4.1 Acute

Because the combined HIs within the identified chemical categories for all mill emission components for each scenarios, even at maximum ground level concentrations and for locations quite close to the Visy mill, are all less than unity *it is unlikely that the mill emissions will cause any adverse acute health effects.*

7.4.2 Chronic non-cancer

Similarly, the HIs for chronic health effects are less than one indicating that it is unlikely that adverse health effects will result from long term exposures to the Visy mill air emissions.

7.4.3 Cancer

Total cancer risks that might be associated with exposure to the low levels of genotoxic carcinogens in the Visy mill air emissions are calculated to be less than the acceptable cancer risk range commonly used by international agencies for protecting public health.

8 DIOXINS

8.1 What are dioxins?

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are collectively called 'dioxins'. Co-planar polychlorinated biphenyls (co-planar PCBs) possess toxicity similar to that of dioxins and are called 'dioxin-like' compounds. Dioxin and furan molecules consist of two benzene rings joined together by oxygen atom(s) with various amounts of chlorine or hydrogen atoms attached in the numbered positions of **Figure 8-1**. There are 75 kinds of PCDDs, 135 PCDFs and more than 10 co-planar PCBs. The different types of dioxins are called congeners.

Dioxins have no known industrial use but occur as unwanted by-products of some industrial and combustion processes such as metal smelting and burning wastes and fuel. In contrast, PCBs have been used for a variety of industrial purposes including heat transfer agents, dielectric fluids for capacitors and transformers, plasticisers and paint additives (**Safe 1990**). Dioxins are ubiquitous in the Australian environment (e.g. **Gaus et al. 2001**), and it has been estimated that bushfires contribute at least 20 – 30% of the total release of dioxin-like compounds to the Australian environment. Thus humans have been exposed to low levels of dioxins, and human metabolism has coped with dioxins, for thousands of years (**OCS, 2004**). More than 95% of dioxin intake by the general public is via the diet, with the majority of this due to fatty foods derived from animals and fish/shell fish as a result of the lipophilicity of dioxins and their ability to bioaccumulate (**Liem et al. 2000, Llobet et al. 2003, WHO 2000**).

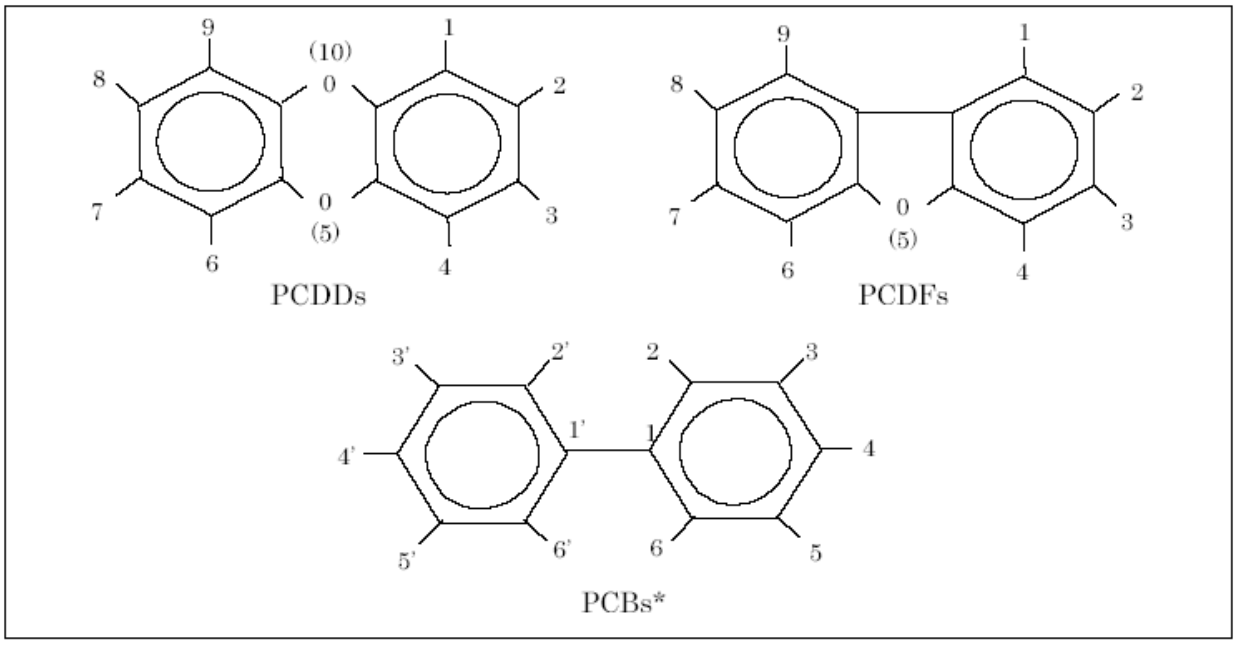

Figure 8-1: Structures of dioxins, dibenzo furans and PCBs.

Humans are invariably exposed to a complex mixture of many dioxins and furans, but the degree of toxicity of different dioxins varies from compound to compound. Not all congeners produce toxicity, only a subset. The tetrachlorinated dibenzo-p-dioxin with chlorine atoms attached in the 2, 3, 7 and 8 positions (2,3,7,8-TCDD usually simplified to TCDD) is known to possess the highest toxic potency and toxic effects of this congener have been the most studied. Because dioxin congeners that cause toxicity appear to do so via a common mode of biological action (binding to a specific receptor inside cells) it is possible to rank the toxicity of various dioxins, furans and co-planar PCBs relative to the toxicity of TCDD. Thus the 'toxic' dioxins are assigned a 'Toxicity Equivalency Factor' (TEF) relative to TCDD according to their ability to bind to the receptor and elicit the activated receptor mediated biochemical and toxic responses. The TEFs developed by the WHO Organisation (**van den Berg *et al.* 1998**) are widely accepted as being the most appropriate for human risk assessment and have been adopted by Australian authorities (**OCS 2004**). They are however heavily dependent upon the biochemical responses elicited in rat tissues, especially the relative ability of the congeners to induce cytochrome P450 mediated enzyme activities. Development of a recent refined database of potency estimates for dioxin-like compounds indicates that on balance the TEF value recommended by the WHO (**van den Berg *et al.* 1998**) are in the upper range of potencies based on different endpoints (**Haws et al. 2006**). However they do not necessarily represent the maximum value.

The toxic potency of a dioxin mixture is estimated by multiplying the mass concentration of each individual congener by its respective TEF. The sum of the products provides the TCDD toxic equivalence (TEQ) for the mixture. Thus TEF values for individual congeners in combination with their chemical concentration can be used to calculate the total TCDD toxic equivalent concentration (TEQs) contributed by all dioxin-like congeners using the following equation (assuming dose additivity).

$$TEQ = \sum (PCDDi \times TEFWHO\ i) + \sum (PCDFi \times TEFWHO\ i) + \sum (PCBi \times TEFWHO\ i) \quad \textbf{Equation 4}$$

The equation assumes there will be no competition between antagonists, weak agonists and full agonists for binding to and activation of the Ah receptor. However, the presence of weak agonists or antagonists in a dioxin/PCB mixture will interfere with the molecular action of the high potency (i.e. full agonists) components and the toxicity of the mixture will be less than that of the same mass exposure of the full agonists alone (**Schwarz and Appel 2005**). Equation 1 however assumes additivity and hence likely overestimates the toxicity of the mixture.

Exposure to dioxins is expressed as the amount (usually in picogram, pg) of dioxin TEQ in the exposure media, e.g. pg TEQ/m^3 if exposure is via air, or pg TEQ/kg if exposure is through food or soil.

8.2 Dioxin toxicity and health guideline

8.2.1 Dioxin Toxicity

> *"there is currently no clear indication of increased disease in the general population attributable to dioxin-like compounds"*
>
> US EPA (2000c)

Adverse effects reported in animals following administration of dioxins include immunotoxicity, endometriosis in Rhesus monkeys and developmental and behavioural effects in offspring of treated monkeys. Developmental effects have also been observed in treated rats. The most sensitive effect, i.e. the one occurring at the lowest dioxin exposure, was decreased sperm production and sexual feminisation in male off-spring of exposed rats. TCDD is carcinogenic in several species, but does not damage DNA (**NHMRC 2002, OCS 2004**).

In humans, the data mostly from relatively highly exposed populations, indicate a variety of subtle biochemical responses may occur. These include induction of hepatic enzymes, changes in hormonal levels

and reduced glucose tolerance. However, these effects are of unknown clinical significance, and may or may not indicate a toxic response or potential for toxic response. Of the many health effects evaluated in exposed adult populations, many were transient and not observed when exposure ceased. Human studies have failed to provide compelling evidence for endometriosis. The most consistently observed effect following high dose exposure is chloracne and other skin conditions. There is also some evidence that high paternal exposure to TCDD may be associated with the birth of more girls than boys. From animal cancer experiments with TCDD and *occupational* studies, plus an understanding of the plausibility of a common mechanism of action for animals and humans the International Agency for Research on Cancer (IARC) has concluded TCDD is carcinogenic to humans (**NHMRC 2002**, **OCS 2004**).

There is compelling data that in animals and humans there is common mechanism of action for the biochemical and toxicological effects, i.e. binding to and activation of the *Ah* receptor. Thus results of animal experiments are used to predict the possibility of health effects in humans that have not been observed in human studies. Effects in animals are therefore used to establish a health guideline for dioxin intake by humans that is regarded by authorities as being safe (see below).

8.2.2 Human sensitivity

The dioxin health guideline is established based on effects in rats and other animals with an uncertainty factor of 10.

According to WHO (**van Leeuwen et al. 2000**), data for Ah receptor binding affinity and responses directly dependent on Ah receptor activation suggest humans may be less susceptible to dioxin than the 'responsive' rodent strains often used in experimental studies. Conversely, other biochemical or cellular effects suggest comparable susceptibility, however these latter effects are not associated with adverse health and their clinical significance is largely unknown (**OCS 2004**).

Hays et al. (**1997**) evaluated the relative susceptibility of humans and rats for cancer using several dose metrics applied to the pivotal rat bioassay (**Kociba et al. 1978, Goodman and Sauer 1992**) and the US National Institute of Occupational Safety and Health (NIOSH) worker cohort (**Fingerhut et al. 1991**). Both these studies had data available on biological dose (blood lipid or adipose tissue TCDD levels) and cancer response. The authors concluded humans are much less sensitive than rats to the carcinogenic effects of TCDD. Others have also suggested that humans are less or no more susceptible to the toxic effects of TCDD and hence exposure of the general population to environmental levels of dioxins should not be of concern (**Kimbrough 1990, Leung et al. 1990**). More recent comparisons of cytochrome P450 (CYP1A1) induction by TCDD in fresh hepatocytes from human donors, rats and rhesus monkeys indicates that humans are about 10 – 100 times less sensitive than are rats (**Silkworth et al. 2005**). Since the TEFs for dioxin congeners are in large part based on the responsiveness of the rat tissues to Ah –receptor mediated biochemical responses it suggests the TEF allocation for congeners may be over estimating the risk to humans by at least an order of magnitude.

A recent review of the molecular structure, function and dose-response data for the human Ah-receptor indicates the human receptor shares key mutations with a mouse strain that compared to sensitive rat strains is relatively unresponsive to TCDD. Binding of TCDD to human Ah-receptor is approximately an order of magnitude lower than that observed with Ah-receptors of sensitive rodents. The TCDD binding data and molecular structure information support the hypothesis that the human Ah receptor is less functional than the Ah receptor of the more sensitive laboratory animals upon which the TEFs are based (**Connor and Aylward 2006).**

8.2.3 Health Guideline

> Dioxin intake health guideline = 70 pg TEQ/kg bw/month

To emphasise the relatively long time frames required for exposure to dioxin like substances before human health effects are likely to occur the Australian NHMRC/TGA recommend (**NHMRC 2002**) a 'Tolerable Monthly Intake' (TMI) of 70 pg TEQ/kg bw; this is instead of the more common 'Tolerable Daily Intake' recommended for most other substances. The TMI is a monthly intake of dioxins and dioxin like PCBs that can occur over 40 - 50 years, such that the body burden associated with adverse health effects is not achieved. The TMI is based on accumulated body burdens in experimental animals associated with subtle adverse effects and a safety factor of about 10 fold is incorporated for humans. That is the TMI is an intake that can pragmatically be considered safe.

In 1990 the WHO established a tolerable daily intake (TDI) for PCDD/PCDF of 10 TEQ/kg bw/d. Re-evaluation of the TDI in 1998 (**WHO 1998**) resulted in a lowering of the TDI to 1 - 4 pg TEQ/kg bw/d. The maximal tolerable intake is 4 pg TEQ/kg bw/d but the target is reduction of intake to below 1 pg TEQ/kg bw/d. More recently the National Health and Medical Research Council of Australia (**NHMRC 2002**) have endorsed the Australian Department of Health and Aged Care recommendation for a TDI of 70 pg TEQ/kg bw/month (this is equivalent to 2.3 pg TEQ/kg bw/d) for dioxin like substances, this in turn takes into consideration the revaluations and recommendations of the European Commission (**EC-SCF 2001**) and JECFA (**2001**).

Because of the wide variation in elimination of PCDD/PCDF and dioxin-like PCBs between species, the WHO (**1998**) TDI was established by using the body burden of TEQ in animals rather than the daily intake. In a number of animal studies the sensitive adverse endpoints (hormonal, reproductive and developmental) occurred within a narrow range of body burdens i.e. 10-50 ng TEQ/kg bw. The human daily intake that would result in an equivalent body burden was calculated to be 14-37 pg/kg/d (i.e. this represents a calculated human low observed adverse effect level [LOAEL]). WHO (**1998**) considered an uncertainty factor of 10 was sufficient to convert this human LOAEL to a TDI, i.e. to a level at which it is anticipated humans will not experience adverse health effects from having that quantity of dioxin like material in their bodies.

The uncertainty factor of 10 was based on the following rationale. Since differences in toxicokinetics (i.e. absorption, metabolism and elimination) are inherently accounted for by using body burden rather than dose it was considered that an uncertainty factor for differences in toxicokinetics between species was not required. It was noted by the WHO working group that the animal 'no-effect' body burdens were within a factor of 2-3 of the animal 'effect' body burdens, hence a lower uncertainty factor than the traditional factor of 10 for conversion of LOAEL to NOAEL was warranted. In addition, the working group noted that for many of the effects observed experimentally, humans are less sensitive than animals so the full uncertainty factor based on the traditional presumed assumption of higher sensitivity of humans to a chemical was not required. This, together with the fact that different components of a dioxin mixture have different half lives in the body, prompted the WHO to use an overall composite factor of 10 to account for the uncertainties.

Thus by applying an uncertainty factory of 10 to the range of animal LOAELs of 14-37 pg TCDD equivalents /kg bw/d, a TDI, expressed as a range, of 1-4 WHO-TEQ pg/kg bw, was established for dioxins and dioxin like compounds. The NHMRC (**2002**) acknowledge this range in their proposal for a TDI for PCDDs/PCDFs in Australia, and has embraced the WHO methodology for calculating toxicity equivalent factors (**Van den Berg et al. 1998, WHO 1998**).

There have been additional risk assessments of TCDD recently conducted by the European Commission (**EC-SCF 2001**) and JECFA (**2001**). These organisations have recommended the tolerable intake of dioxin like compounds be based on long term exposures and have suggested exposure standards that are close to the mid-range of the WHO (**1998**) 1-4 TEQ pg/kg bw/d. These recommendations are 14 TEQ pg/kg bw/week (**EC-SCF 2001**) and 70 TEQ pg/kg bw/month (**JECFA 2001**). These convert to 2 and 2.3 TEQ pg/kg bw/d respectively. All organisations have reviewed the same data but have used different processes to derive their recommended exposure standards. It is noteworthy that approximately the same recommendations have been made.

The NHMRC (**2002**) report a principal finding of the US EPA's evaluation of dioxins on human health (**US EPA 2000c**) that although dioxins can initiate biochemical and biological events resulting in the potential for a spectrum of cancer and non-cancer responses in animals, "*there is currently no clear indication of increased disease in the general population attributable to dioxin-like compounds*". This is important because dioxins are ubiquitous in the environment, they are formed during any combustion process (car engines, waste incineration, wood fires, bush fires etc.), and hence exposure and accumulation of dioxins in the body cannot be avoided.

8.3 Thresholds and dioxins

An important aspect of the risk assessment for dioxins is the implication that the toxic effects of dioxins have a threshold exposure (or dose) below which no adverse health effect will occur. This is the fundamental premise underpinning the establishment of the TMI health guideline.

Dioxins can cause both non-cancer and cancer effects. It is widely accepted that thresholds exist for the non-cancer effects (**ECSCF 2001, JECFA 2001, FSA 2001, NHMRC 2002, OCS 2004).** However the US EPA (**2003**), contrary to other regulatory agencies around the world, has adopted a policy of using a linearised low-dose mathematical model for estimating cancer risks from small dioxin exposures. Such a model assumes no threshold for the cancer effects and implies any dose carries with it a statistical likelihood of cancer for those exposed. This dose response model is usually reserved for risk assessment of substances that cause cancer by direct damage to DNA, i.e. genotoxic substances. Although dioxins are animal multisite carcinogens they are not genotoxic and hence are not initiators of cancer. They are however tumour promoters (**OCS 2004**). In addition to promoting cancer initiated by genotoxic agents, dioxins also appear to cause cancer in targeted tissues through Ah receptor activation and hormonal imbalances, and also perhaps by inducing the metabolism of procarcinogens (**Pohl et al. 2002**). These biological mechanisms indicate thresholds exist for dioxin induced cancer. The animal and human carcinogenicity data for TCDD has recently been reviewed by Popp et al. (**2006**), who concluded the level of certainty for a non-linear cancer dose response was substantial because there is concordance of many lines of evidence and consistency of repeated observations pointing to non-linearity.

Thus both mechanistically and experimentally, the weight of evidence robustly supports a non-linear dose response for the carcinogenic effects of dioxins (i.e. the data supports the existence of a threshold for the cancer effects). It is noted the US EPA has been criticised for their policy position for assuming linearity (**Kayajanian 2002, Phol et al. 2002, Popp et al. 2006**). The WHO, Australia, scientists advising the US EPA and others support the concept of a non-linear dose response for dioxins and cancer (**SAB 1995, van Leeuwen et al. 2000, ECSCF 2001, JECFA 2001, FSA 2001, NHMRC 2002, OCS 2004, Schwarz and Appel 2005**).

This risk assessment does not follow the US EPA approach of calculating cancer risks from dioxin exposure. Consistent with other epigenetic carcinogens and the deliberations of most international authorities, we consider a practical threshold exists for the cancer effects of dioxins and that the TMI established by the WHO and Australian authorities provides protection against cancer as well as non-cancer health effects. In fact the reproductive and hormonal effects in experimental animal studies seem to occur at lower body burdens than required for cancer (**Pohl et al. 2002, OCS 2004**).

8.4 Data used for the risk assessment

Annual average ground level concentrations were provided to Toxikos as µg TCDD/m^3, since TCDD has a TEF of 1.0 the data provided is equivalent to µg TEQ/m^3. The dioxin ground level concentrations used provided are reproduced in **Error! Reference source not found.**.

Table 8-1: Annual average ground level concentrations of dioxins[a]

Receptor	TCDD (µgTEQ/m3)
1	8.17E-12
2	1.11E-11
3	6.67E-12
4	1.11E-11
5	**2.08E-11**
6	1.23E-11
7	9.90E-12
8	9.28E-12
9	5.07E-12
10	6.54E-12
11	5.32E-12
12	6.18E-12
13	5.18E-12
14	5.76E-12
15	3.37E-12
16	4.90E-12
17	1.02E-11
18	1.04E-11
19	5.98E-12
20	3.25E-12

[a] Bolded number represents the highest annual ground level concentration at any receptor site modelled. This is used as the annual ground level concentration for the risk assessment. This is the same as 2.1 x 10^{-5} pg TEQ/m^3, i.e. 0.000021 pg TEQ/m^3.

8.5 Contextual information on air concentrations of dioxins

As far as Toxikos has been able to ascertain, an ambient air guideline level for dioxins/furans has not been declared in Australia, nor by the European Commission or its member states (**EC 1999**), the US EPA or Environment Canada. However an Ontario Ministry of the Environment air quality standard of 0.1 pg TEQ/m^3, , a Japanese air quality standard of 0.6 pg TEQ/m^3 has been established and several US States (**ATSDR 1998b**) also have set ambient air guideline values for dioxins. As an annual average, these are reported to range between 0.023 to 35 pg/m^3 (**ATSDR 1998b**). DPIWE (**2004**) have specified a design level criterion for dioxins of 3.7 pg/m^3 for TCDD, this is the same as 3.7 pg TEQ/m^3 because TCDD has a TEF equal to 1.

Information on relative concentrations of dioxin like substances in air is provided in **Table 8-2**. The highest annual average ground level concentration predicted at the nominated receptors is 0.000021 pg TEQ/m^3 (**Table 8-2**). In Europe, a background concentration of 0.1 pg TEQ/m^3 is assumed but certain industrial and urban areas, as well as areas close to major sources, may air concentrations that are up to 20 times higher (**WHO 2000**). In Japan, atmospheric concentrations of 0.55 pg TEQ/m^3 for dioxins/furans plus PCBs have been measured and used for assessing risk (**EA/MoHW 1999**). Concentrations at Griffith University, Queensland, are approximately 0.009 – 0.017 pg TEQ/m^3 (**Muller et al 1998**).

Table 8-2: Relative concentrations (pg TEQ/m^3) of dioxin like substances in air.

Location	Concentration	Reference
Woomera	0.000021 [a]	Table 8.1, this report.
Wattleup [c]	0.016	Gras et al. (2004).
Duncraig [d]	0.057	Gras et al. (2004).
Griffith University, Brisbane	0.009 – 0.017 [b]	Muller et al. (1998).
Urban Brisbane	0.0047 b	Muller et al. (1998).
Urban Sydney	0.0016 – 0.062 [b]	Cited in Muller et al. (1998)
Assumed for Europe	0.1	WHO (2000).
Japan	0.55	EA/MoHW (1999).

[a] Highest predicted incremental increase at any modelled receptor.
[b] Total TCCD equivalents calculated with NATO factors.
[c] Wattleup, in the Kwinana area, Perth, WA (industrial).
[d] Duncraig, Perth, WA (mid-sized urban).

8.6 Relative contribution to intake by inhalation

WHO (**2000**) has not established an air quality guideline for dioxins because they consider direct inhalation exposures to constitute only a small proportion of the total body burden, generally less than 5% of the daily intake being from air (more than 95% of human intake of dioxins comes from fatty foods). According to Edjuljee and Gair (**1996**), for exposure conditions typically encountered by the general population, inhalation contributes up to 2% of the total intake of dioxins. **Table 8-3** summarises some of the available information regarding the relative contribution of exposure via air to dioxin-like substances. From this information, a relative percentage contribution of 1% by inhalation to the total intake has been used in the screening risk assessment. This is lower than most estimates in **Table 8-3** but close to that for Australians. That is the ratio of intake from inhalation compared to other exposures is 1:99. With respect to the calculations for overall dioxin intake from all pathways in **Equation 6** a lower proportion assigned to inhalation is more conservative.

Table 8-3: Contribution of exposure via air to total intake of dioxin like substances.

Situation	Contribution by inhalation	Reference
Average daily intake by individuals living near waste incinerator	6.8%	Hattemer-Fry & Travis (1991)
A variety of receptor types living near waste incinerator	1 – 11%	UK (1996)
General UK population	<2%	Edjuljee & Gair (1996)
Background intakes in Japan	6.5%	EA/MoHW (1999). (2001) (2003)
Living near a municipal waste incinerator	2.1%	Ma (2002)
Australian adults	2.3%	OCS (2004, Table 3 -34)
American adults	9.3% at 50th percentile cumulative risk 6.6% at 75th percentile 3% at 95th percentile 0.8% of upper bound 95th percentile estimate.	As cited by OCS (2004)

8.7 Background intake of dioxin-like substances

A pivotal aspect of the screening risk assessment for exposure pathways for dioxin exposure is the estimation used for background intake of dioxin like substances. The Australian Government has recently published estimates for background intake of dioxin like substances for Australians (**OCS 2004**). The estimated total background intakes from all sources of exposure for dioxins and furans, polychlorinated biphenyls (PCBs) and total dioxin like substances for Australian adults are presented in **Table 8-4**. Intake from food accounts for between 95-99% of the total intakes and intakes from air are generally less than 1-5% (**OCS 2004**). For the purposes of this risk assessment, the upper bound total intake estimates are used.

Table 8-4: Estimated total intakes of Australian adults to dioxin like substances

Total Intake (pg WHO TEQ/kg bw/month)					
DIOXINS & FURANS		**PCBS**		**TOTAL DIOXIN TEQ**	
Lower bound	Upper bound	Lower bound	Upper bound	Lower bound	Upper bound
1.06	10.37	2.83	5.42	3.89	**15.79**

8.8 Methodology for assessing risk from dioxins emissions

The methodology and equations described below assess all the exposure pathways depicted in **Figure 4-1** (i.e. direct and indirect secondary pathways) but they do not provide a detailed examination of each of the secondary pathways.

The method of assessment inherently includes potential exposure from secondary exposure pathways for the dioxin emissions because it assumes:

- Steady state equilibrium is established between the presumed environmental incremental increase of dioxins resulting from facility emissions and human food sources.
- The same proportional relationship between inhalation exposure and total intake is maintained in the new steady state conditions surrounding the mill as exists for background intakes, and exposures to dioxins from incinerator emissions, for which there is data.
- Existing background intake of dioxins, from all sources, is conservatively incorporated into the risk assessment by assuming people in the Tumut area have current intakes of dioxins equivalent to the upper bound estimate of the general Australian population.

The general principle for assessing potential health impacts of dioxins emitted from the Visy mill is to determine an incremental monthly intake from all exposure routes that can be attributable to the dioxin content of emissions. Added to this is the upper bound estimate of background monthly intake, so that the sum is then compared to the monthly intake declared tolerable (TMI) by the Australian Government Department of Health and Ageing (**NHMRC 2002**). If the combined intake is markedly less than the TMI then the risk of health effects from dioxins in the mill emissions is very low.

The intake of dioxin like substances occurs from a range of sources (food, air, soil) whose contribution to the total intake is not equal; the majority of dioxin intake by humans comes from animal fat (**US EPA 2000b**).

The total monthly intake (MITOTAL) of dioxins is mathematically represented thus:

(MITOTAL) = MBI + MIINHAL + MIFOOD + MISOIL + MIWATER……..**Equation 5**

Where MITOTAL = Total Monthly Intake

MBI = Monthly Background Intake from all sources.

MIINHAL = Monthly Intake from direct inhalation

MIFOOD = Monthly Intake from food due to incremental increase in air concentration from emissions.

MISOIL = Monthly Intake from soil due to incremental increase in air concentration from emissions.

MIWATER = Monthly Intake from water due to incremental increase in air concentration from emissions.

MBI = 15.79 pg/kg bw/d (**Table 8-4**)

From Table 8.2, the appropriate maximum ground level concentration for dioxin like substances is 0.000021 pg TEQ/m^3. Hence the monthly average intake for an adult via inhalation (MIINHAL) is:

MIINHAL= [(0.000021 pg TEQ/m^3) x (22m^3/d[f] x 30d)] ÷ 70kg ……**Equation 6**

= 0.0002 pg TEQ/kg /m.

Since 1% of dioxin intake by humans is from inhalation and 99% from all other exposures, the incremental increase from pathways other than inhalation can be estimated as:

[MIFOOD + MISOIL + MIWATER] ~ 99 x MIINHAL …… **Equation 7**

(99 is the proportional ratio of intake from all other media compared to air, **Section 8.5**).

Substituting into Equation 9.2

MITOTAL = MBI + MIINHAL + [MIFOOD + MISOIL + MIWATER]……**Equation 8**

= MBI + MIINHAL + 99[MIINHAL]

= MBI + 100[MIINHAL] ……..**Equation 9**

= 15.79 pg TEQ/kg /m + 100 [0.0002 pg TEQ/kg /m]

= 15.79 pg TEQ/kg /m + 0.02 pg TEQ/kg /m

= 15.81 pg TEQ/kg /m

Therefore the total human intake of dioxins, including background, arising from a theoretical long term (30 – 40 years) increase in exposure due to air emissions is 22.6% of the Tolerable Monthly Intake. The conservatively estimated incremental increase in the total human intake of dioxins (including background) due to the mill emissions is 0.13%.

8.9 Conclusion

The increase in dioxin intake by people living around the mill due to air emissions from the mill is very small (0.13% of current maximum background intakes). The total intake including current maximum background intakes is 22.6% of the recommended tolerable intake considered by Australian health authorities to be without adverse health effects. The total intake includes current background intake, the incremental intake from air emissions (direct and indirect) plus from eating large amounts of fish from the ocean. More than 99% of the estimated total intake is due to current background exposures.

Based on the foregoing, it is concluded that dioxin released from the mill, is negligible compared to background exposures and is unlikely to cause health effects

[f] Average daily volume

9 SECONDARY EXPOSURE PATHWAYS

There are a large number of ways by which humans may be exposed to industrial emissions to air. For some substances with the appropriate physiochemical (e.g. solubility in fat), environmental (e.g. persistence) and biological (e.g. poor rates of metabolism and excretion) properties accumulation through the human food chain is a theoretical possibility. Increased exposure may also occur due to atmospheric deposition resulting in direct contact, inhalation of dust due to accumulated impacts of metals in soil. In this risk assessment, exposure via inhalation of emission components is referred to as the direct exposure pathway and has been examined in detail. All other exposure pathways are called secondary exposure pathways, which are summarised in **Figure 4-1.**

The process of evaluating health risks from exposure via secondary pathways is resource intensive, and requires many assumptions which inherently contain unquantifiable uncertainties. The assumptions are made because empirical relationships for the movement of most substances from air to other media to which humans may be exposed have not been established. Consequently, in most risk assessments the secondary exposure pathways are not considered, or unrealistic conjectures are made to establish gross worse case exposure scenarios. In lieu of these approaches, a pragmatic alternative is to perform screening assessments to evaluate the need for, and hence benefit from a detailed multi-pathway risk assessment. Usually the pre-evaluation is qualitative and based on the assessor's experience. The pre-screens in **Appendix B** for metals and PAHs have been structured to provide a more objective and transparent process.

Dioxins have the necessary properties to be bioaccumulated in the food chain and the relative proportion that each exposure pathway contributes to the overall intake has been well studied. This information has been applied in **Section 8** for assessing the risk that multiple pathway exposure (air, food, soil and water) to dioxins may pose. For emissions from the mill it was found that under steady state conditions (i.e. where long term concentrations of dioxins in air are assumed to be equilibrated with environmental compartments and human food) the likely increase in body burden by humans is extremely small. A conservative (i.e. overestimation) of exposure indicated only a 0.13% increase above current background intakes of dioxin was anticipated.

Appendix B describes the process used to determine whether other substances will need to be subject to a detailed multi-media evaluation. These processes draw upon the principles articulated in the dioxin assessment (**Section 8**), a comparison of predicted annual ground level concentrations with measured ambient air concentrations from around the world, and an assessment of the literature indicating whether bioaccumulation/biomagnification of a substance by terrestrial human food sources was likely.

PAHs are products of incomplete combustion of organic material. The predominant sources of PAH in the environment are motor vehicle traffic (both petrol- and diesel-fuelled), residential heating, especially with wood or coal, burning-off and bushfires. Reviews of the scientific literature indicate little uptake and translocation of PAHs by plants from soil. In addition, organisms that metabolise PAH, such as fish and higher invertebrates and mammals that are human food sources, accumulate little or no PAHs (**ATSDR 1995, WHO 1998a**). It follows therefore that if there is little or no bioaccumulation of PAHs by plants or animals likely to be consumed by humans, therefore evaluation of secondary exposure pathways for the PAHs is not warranted (see **Appendix B**).

For metals, the screening procedure is pragmatically grounded in an estimation of soil metal concentrations from deposition of mill emissions and comparison with health-based investigation guidelines. Additionally a comparison of predicted receptor ground level concentrations with rural background concentrations that are not associated with significant exposures via secondary pathways was also undertaken. This is augmented:

- By a requirement for a significant inhalation margin of exposure for individual metals such that if exposure was to occur via secondary pathways there is ample conservatism in the screening process to ensure the additional non-inhalation intakes will not result in adverse health effects in humans.
- With a brief review of the literature which indicated lack of potential for most metals to bio-magnify through the food chain.

It is concluded in **Appendix B** that since the screening criteria for metals were not satisfied, detailed examination of the secondary exposure pathways is not required.

10 UNCERTAINTY DISCUSSION

In interpreting the calculated risks associated with assumed exposure to emissions from the mill, uncertainties associated with the assessment need to be considered. The risk assessment process involves a number of steps (e.g. exposure assessment, toxicity assessment and risk characterisation), each of which incorporates the use of assumptions and simplifications to manage uncertainty or lack of knowledge about the correct value. Without such assumptions and simplifications it would not be possible to quantitatively evaluate the potential for health effects. Although uncertainties in the risk assessment may influence its accuracy, reliability and interpretation, the assumptions used to cope with the uncertainties err on the side of caution and therefore bias the evaluation to an over estimation of the potential health risk. This is appropriate for a prospective assessment for possible impacts on public health. It must be realised however the conservatism regarding one value is at least additive, most times multiplicative, with other conservatisms such that the cumulative or compound conservatism incorporated into the assessment can be very large. This is especially so when gross, unrealistic default parameters are used in lieu of site-specific data.

This section contains a general qualitative discussion of the major uncertainties and their potential influence on the health risk assessment. The 'big picture' uncertainties fall into the following major categories.

- Those associated with exposure estimation.
- Receptor specific uncertainties.
- Contaminant specific uncertainties.

The above are addressed in **Table 10-1**, which presents a listing of the major areas of uncertainty. Elsewhere in the report, when particular risks, chemical classes or health endpoints are discussed/assessed, additional specific information on the uncertainty is provided to enable the reader to integrate the uncertainties with the assessment that has been performed at that point in the report. The HRA is dependent upon the prediction of GLCs, as while there is conservatism in the HRA, it may not be sufficient if GLCs are markedly under-predicted.

Table 10-1: Uncertainties in the risk assessment for mill emissions and potential effect on HRA outcome

Uncertainty/Assumptions	Comment	Effect on Risk Assessment
Exposure Estimation		
Some emission components may not have been identified or appropriately quantitated.	Toxikos has no information regarding the veracity of either the emission inventory or the representativeness for each of the scenarios, or of the dispersion modelling.	Emissions, and therefore GLCs, may be under – or over-estimated.
Mill process variability and hence emissions variability is not known at this time.	Toxikos has no information on this aspect.	Emissions, and therefore GLCs, may be under – or over-estimated.
Emissions during start-up and shut-down conditions have not been characterised.	Start-up & shut down not modelled.	Engineering safety features are designed for significant up-set & start-up/shut-down. Emissions, and therefore GLCs, may be under – or over-estimated.
There is uncertainty in the air dispersion modelling in its predictions of ground level concentrations of emission components at the receptor locations of interest.	Modelling techniques contain inherent uncertainty.	There is uncertainty in the predicted GLCs, they may be under - or over-estimated.
Receptor Uncertainty		
There may be people within the emission dispersion zone that are more susceptible than most to developing health effects if they are exposed to refinery emissions.	Public health air guidelines are established to account for the variability in human response and therefore largely compensate for lack of receptor characterisation in most HRAs. It is not usual to characterise the exposed population with respect to susceptibility in a HRA of this nature. Nevertheless the demographics of the region are similar to NSW as a whole.	Impact on the conclusions of the HRA is minimal. However it is recognised there may be a very small, unlikely, possibility of an adverse health reaction if unusually sensitive individuals are exposed. This is no different than any other public health assessment using regulatory guideline values. As far as possible the possibility of highly sensitive responders has been catered for by inclusion of reasonable conservatism in the assessment.

Contaminant Uncertainty		
Defining toxicological potency of emission components. Dose response relationships are not fully determined for all emission components.	The HRA relies on established regulatory guidelines to protect public health. Dose response relationships of some individual emission components have been provided. While summary information regarding NOELs and safety factors is provided with a brief basis of how guidelines were determined. An assessment of whether the guideline is specifically established using the most relevant sensitive health endpoint and safety factors are appropriate has not been done. In this sense the HRA has been of a screening nature; if the predicted GLC of any given emission component approached the guideline value then it was intended to review the toxicological/health database for that component in more detail. Since this has not occurred detailed dose response assessments have not been undertaken. Upper 95th percentile estimates of cancer unit risks for carcinogenic mill emission components have been used in calculations.	It is possible that the health guideline values used to characterise risk may not be protective of sensitive sub-groups in the exposed population. However given the large margins of safety between the NOEL and guideline for the majority of emission components (i.e. the use of safety factors in establishing guidelines) it is unlikely the guidelines used will fail to be protective of all or nearly all individuals. This is the very essence of the philosophy for creating public health guidelines. This over estimates cancer risks. This over estimates cancer risks.
There may be interactive health effects between emission components.	Regardless of effect or mode of toxicological action, additivity of either dose or effect has been assumed to occur between mill emission components.	This practice causes the HRA to grossly overestimate the risks to combined exposure to emission components from the mill. The health impact of background emissions may be under-estimated.

Health guideline values are not available for all substances of concern.	Calculation of risk is sensitive to the numerical value of the health guideline reference values. These are subject to science policy judgments of various regulatory regimes and are influenced by the legislative arenas. Wherever possible Australian or WHO guideline values have been adopted. However values from other regulatory regimes have been used, but only if they are regarded as being competent (e.g. Cal EPA, US EPA, RIVM). The aim of the guideline is the protection of public health. There is documentation supporting the value which demonstrates careful consideration of the available data, and/or the information used is more current than that of the Australian authority.	The HRA does not underestimate risk due to the use of an inappropriate guideline.

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APPENDIX A SUPPORTING TOXICOLOGICAL INFORMATION

A.1.1 TOXICITY CATEGORY SUMMARY

The toxicity category table identifies whether an emission component has been evaluated by a relevant competent authority for carcinogenic and/or reproductive toxicity potential. It also details the major target organ for which critical effects have been observed. This information can help in deciding whether there may be biological legitimacy for assuming additivity of effects between components. In strict biological terms additivity would only be expected if emission components were affecting the same tissue types in an equivalent manner.

Additional toxicity information is provided for the key contaminants of potential concern in **Section A1.2.**

Section A1.2 provides a brief summary of the human health-based guideline values for each contaminants of potential concern with a guideline value detailing the critical effect, the no observed effect concentration or lowest observed effect concentration and the uncertainty factors applied by the authority to create the toxicity guideline value.

Toxicity category summaries are presented for all contaminants of potential concern. There are nine substances that are considered carcinogens and eleven reproductive toxicants. The most common target organ is the respiratory system.

Chemicals were placed into 'toxicity categories' by consulting the following sources:

1 IARC Monographs and Supplements on the Evaluation of Carcinogenic Risks to Humans.

2 Environmental Health Criteria Monograph Series from the International Programme on Chemical Safety, World Health Organization.

3 Toxicological Profiles for Chemical Substances, Agency for Toxic substances and Disease Registry (ATSDR), US Department of Health and Human Services.

4 Office of Environmental Human Hazard Assessment (OEHHA), Californian EPA.

5 Re-evaluation of Human Toxicological Maximum Permissible Risk Levels, Dutch National Institute of Public Health and the Environment (RIVM 2001).

6 WHO Guidelines for Air Quality (1999); Air Quality Guidelines for Europe, 2nd Edition (2000), World Health Organization.

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9 Patty's Industrial hygiene and toxicology / George D. Clayton and Florence E. Clayton, editors. Electronic edition.

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Table 11-1: Toxicity category summary table

	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Criteria Pollutants				
Carbon monoxide	Blood & cardiovascular system.	N	N	Y
Lead (Pb)	Associated with impaired neurobehavioural functioning and IQ in children.	N	Y(IARC 2B, USEPA B2)	Y
Nitrogen dioxide	Respiratory system, decreased pulmonary function in asthmatics.	N	N	N
PM_{10} (Data presented as total suspended particles TSP)	Respiratory system effects, decreased pulmonary function in responding subpopulations. Cardiovascular system.	N	N	N
Sulphur dioxide	Respiratory system irritation and decreased pulmonary function in responding subpopulations of exercising asthmatics.	N	N	N
Metals				
Antimony	Pneumoconiosis in humans	N	Y (IARC 2B)	N
Arsenic (As)	Respiratory, skin, reproductive/developmental, cardiovascular, nervous system. Lung cancer.	Y	Y (IARC 1, USEPA A)	N
Beryllium (Be)	Respiratory inflammation (Pneumoconiosis) and Lung cancer.	Y	Y (IARC 1, USEPA B1)	N
Cadmium (Cd)	Respiratory system and kidney. Lung cancer.	Y	Y (IARC 1, USEPA B1)	Y
Chromium (Cr^{III})	Essential element.	N	N	N
Chromium (Cr^{VI})	Respiratory system. Lung cancer.	Y	Y (IARC 1, USEPA A Cr^{VI} only)	N
Cobalt (Co)	Respiratory, cardiotoxic effects	N	Y (IARC 2B, USEPA D)	N
Copper (Cu)	Respiratory system irritation.	N	N (USEPA D)	N

	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Manganese (Mn)	Nervous system, lungs, and reproductive system	N	N (USEPA D)	Y
Mercury (Hg)	Central nervous system and kidneys.	N	N	Y
Nickel (Ni)	Lung and nasal tumours.	Y	Y (IARC 1, USEPA A)	N
Selenium (Se)	Clinical selenosis. Characteristic "garlic odour" thickened brittle nails, hair and nail loss, mottled teeth, lowered haemoglobin levels, skin lesions and possible CNS abnormalities with severe intoxication.	N	N	N
Tin (Sn)	Respiratory tract.	N	N	N
Vanadium (V)	Respiratory tract.	N	N	N
Dioxins and Furans	Liver, reproductive, developmental, endocrine, respiratory, hematopoietic effects.	N	Y (IARC 1)	Y
Polycyclic aromatic hydrocarbons	Respiratory system cancer.	Y	Y (IARC 2B, US EPA B2)	N
Acenaphthene	Respiratory system	Y	Y (IARC 2A)	N
Acenaphthylene	Respiratory system	Y	Y (IARC 2A USEPA D)	N
Anthracene	Respiratory system	Y	Y (IARC 2A USEPA D)	N
Benz(a)anthracene		Y	Not evaluated by IARC or USEPA	N
Benzo[b]fluoranthene	Respiratory system	Y	Y (IARC 2A USEPA B2)	N
Benzo[k]fluoranthene	Respiratory system	Y	Y (IARC 2A)	N
Benzo[ghi]perylene	Respiratory system	Y	Y (IARC 2A USEPA D)	N
Benzo[a]pyrene	Respiratory system	Y	Y (IARC 2A, USEPA B2)	N
Chrysene	Respiratory system	Y	Y (IARC 2A)	N
Dibenz[a,h]anthracene	Respiratory system	Y	Y (IARC 2A)	N

	Target Organ of Concern	Toxicity Category		
		Genotoxic	Carcinogenic[a]	Reproductive Toxicant
Fluoranthene	Respiratory system	Y	Y (IARC 2A, USEPA D)	N
Fluorene	Respiratory system	Y	Y (IARC 2A, USEPA D)	N
Indeno[1,2,3-cd]pyrene	Respiratory system	Y	Y (IARC 2A, USEPA D)	N
Phenanthrene	Respiratory system	Y	Y (IARC 2A, USEPA D)	N
Pyrene	Respiratory system	Y	Y (IARC 2A, USEPA D)	N
Sulphur-Containing Compounds				
Hydrogen sulphide	Olfactory mucosa lesions.	N [b]	Not evaluated	Y
Total reduced sulphur	As for hydrogen sulphide.	N [b]	Not evaluated	Y
Inorganic & Miscellaneous Compounds				
Chlorine	Respiratory tract irritation and damage.	N	N IARC 3 [c]	N
Chlorine dioxide	Respiratory tract irritation and damage.	N [d]	N (IARC 3) [c] US EPA D)	N
Hydrogen chloride	Respiratory tract irritation and damage.	N	N (IARC 3)	N

For the purposes of the report category 3 carcinogens have been designated with "N" for carcinogenity. A Category 3 classification does not mean that a substance is not carcinogenic, only that the information available is insufficient for classification (**WHO 2003**).

There is insufficient data with which to evaluate the genotoxicity of hydrogen sulphide (WHO 2033).

Evaluated as chlorinated drinking water.

A weight of evidence assessment indicates it may be weakly genotoxic at the site of contact (i.e. nasal mucosa) (**NICNAS 2006**).

Peer reviewed regulatory toxicology reviews of trichloroethylene acknowledge the substance, at best only has weak genotoxic activity.

Table 11-2: Carcinogen classifications of IARC and US EPA

IARC Carcinogen Classifications	
Group	**Category**
1	Is a human carcinogen.
2A	Is probably carcinogenic to humans.
2B	Is possibly carcinogenic to humans.
3	Is not classifiable as to its carcinogenicity.
4	Is probably not carcinogenic to humans.

US EPA Carcinogen classifications.	
Group	**Category**
A	Human carcinogen.
B	Probable human carcinogen.
B1	Indicates limited human evidence.
B2	Indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human.
D	Not classifiable as to human carcinogenicity.
E	Evidence of non-carcinogenicity for humans.

This table represents compounds for which readily identifiable guidelines from an authoritative source *were not available*. No attempt has been made to locate useable data from the open scientific literature from which health reference values could be derived because GLC for most of these compounds are very low.

Table 11-3: Health based air guideline values

Type of Guideline	Compound
Acute	Antimony, Beryllium, Cobalt, Manganese, Selenium, Tin, 1,2-Dichlorobenzene, 1,3-Dichlorobenzene, Ethylbenzene, 1,1,1,2-Tetrachloroethane, Trichloroethylene.
Chronic	Acenaphthene[a], Acenaphthylene[a], Anthracene[a], Benzo(a)anthracene[a], Benzo(b)fluoranthene[a], Benzo(k)fluoranthene[a], Benzo(a)pyrene[a], Benzo(ghi)perylene[a], Chrysene[a], 1,3- Dichlorobenzene, Dibenzo(a,h)anthracene[a], Fluoranthene1, Fluorene1, Indeno(1,2, 3-cd)pyrene1, Phenanthrene1, Pyrene1, 1,1,1,2-Tetrachloroethane.

1 These substances have been evaluated as genotoxic carcinogens. An evaluation of the non-carcinogenic effects of these substances was not carried out. Dioxins are evaluated separately in Section 8.

Table 11-4: Summary acute health effects guidelines

Compound	Health endpoint for guideline value	Critical effect level [mg/m^3] [b]	UF [c]	Air Guideline Value [µg/m^3] [a]	Averaging time for guideline value	Source
Arsenic (As)	Developmental toxicity	0.19 (LOAEL)	1,000	0.19	4 hours	OEHHA (1999a)
Cadmium (Cd)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Carbon monoxide	COHb formation & cardiovascular function	Critical level of COHb < 2.5%	Not applicable	10,000	8 hours	NEPC (1998)
Chlorine	Throat irritation human (respiratory & eye)	2.9 (NOAEL for 30 minutes)	10	210	1 hour	OEHHA (1999b)
Chlorine dioxide	No acute guidelines located. Acute health effects are similar to chlorine but based on occupational exposure standards in US and Germany being 5 times less, the chlorine guideline divided by 5 is adopted [d].			40	1 hour	
Chromium (Cr)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Copper (Cu)	Based on prevention of metal fume fever	1 (NOAEL)	10	100	1 hour	OEHHA (1999c)
Dioxins	Effects of concern are associated with long term exposure. No acute health guideline located.					
Hydrogen chloride	Eye and throat irritation (human)	2.7 (NOAEL for 45 mins)	1	2,100	1 hour	OEHHA (1999d)
Hydrogen sulphide	Bronchial obstruction in asthmatics	2.8 (LOAEL)	30	100	30min	WHO (2003)
	Lesions in rat olfactory mucosa	NOAEL(14) adjusted to 24 hr (3.5) and HEC (0.63)	10	20	24 hour	WHO (2003)
Lead (Pb)	Effects of concern are associated with long term exposure. No acute health guideline located.					
Mercury (Hg)	Developmental toxicity (behavioural deficits in foetus)	1.8	1,000	1.8	1 hour	OEHHA (1999e)
Nickel (Ni)	Slight changes in lung function	33 (LOAEL)	6	6	1 hour	OEHHA (1999f)

Compound	Health endpoint for guideline value	Critical effect level [mg/m³] [b]	UF [c]	Air Guideline Value [µg/m³] [a]	Averaging time for guideline value	Source
NO_2	Exacerbation of asthma & increased responsiveness to environmental bronchoconstrictors in asthmatics	LOEL ~376 – 565 (~200 – 300 ppb)[e] For 20' -4h exposures	2	226 (120 ppb)[e]	1 hour	NEPC (1998)
PM_{10}	Aggravation of existing respiratory & cardiovascular diseases			50	24 hour	NEPC (1998)
$PM_{2.5}$	Aggravation of existing respiratory & cardiovascular diseases			25	24 hour	NEPC (2003)
Polycyclic aromatic hydrocarbons (PAH)	Effects of concern are associated with long term exposure. No acute health guideline located.					
SO_2	Changes in lung function &/or exacerbation of respiratory symptoms in responding subpopulations of exercising asthmatics.	0.524 (NOAEL) (200 ppb)[e]	2	262 (100 ppb)[e] 456 (175 ppb)[e] 524 (200 ppb)[e] 210 (80 ppb)[e]	15 min 10 min 1 hour 24 hour	UK DoE (2000). WHO (2000) NEPC (1998)
TRS (Total Reduced Sulphur)	Taken the same as for hydrogen sulphide (see Section 12.4).					
Vanadium (V)	Respiratory effects in workers.	0.02 (LOAEL)	20	1	24 hour	WHO 1998c

[a] Because Australia has very few guidelines for acute exposures, guidelines have been sourced from a variety of competent authorities where documentation is available for subsequent verification of the guideline if required.
[b] NOAEL = No Observed Effect Adverse Level, LOAEL = Low Observed Effect Adverse Level
[c] UF = Uncertainty factor applied to the critical effect level (i.e. NOAEL or LOAEL).
[d] The Australian occupational standard for Cl_2 and ClO_2 are respectively 1 and 0.1ppm, however the Cl_2 standard was adopted in 1990 and is now out of date. Current OELs for Cl_2 in US (ACGIH) and Germany are 0.5ppm (8 hr TWA).
[e] Conversions from ppb into µg/m³ performed at 25°C and 101.7pa.

Table 11-5: Summary chronic health effect guidelines

Compound	Annual bkgd ambient air level [µg/m³] [a]	Health endpoint	Critical effect level [µg/m³]	UF	AGV[b] [µg/m³]	Av. Time	Source
Arsenic	0.001- 0.028	Developmental, cardiovascular, nervous system.	200 (LOAEL converted to a HEC [c] of 33)	1,000	0.03	1 yr	OEHHA (1997a)
Cadmium (Cd)	0.001 – 0.1	Decreased renal function in workers.	100 – 400 µg/m³-yrs (LOAEL)	N/A	0.005	1 yr	WHO (2000a)
Carbon monoxide	Effects are related to COHb formation from short term exposure. guideline not located.						Chronic
Chromium (Cr^{III})	Total Cr 5-200 $x10^{-3}$	Respiratory system	600 (NOAEL)	10	60	NS[d]	RIVM (2001)
Chromium (Cr^{VI})		Respiratory and nasal irritation.	1 (NOAEL)	10	0.1	NS[d]	USEPA (2004d)
Copper (Cu)	0.005-0.05	Respiratory and immunological effects.	100	100	1	NS[d]	RIVM (2001)
Hydrogen sulphide		Olfactory neuron loss in rats.	NOAEL(13900) adjusted for discontinuous exposure (3,480) and converted to a HEC (640)[c]	300	2	1 yr	US EPA IRIS (2003)
Lead (Pb)	0.01-2	Critical level of Pb in the blood (<100-150 µg Pb/L)	N/A	N/A	0.5	1 yr	NEPM (1998) WHO (2000b)
Mercury - inorganic (Hg)	0.002 – 0.01	Renal tubular effects in workers.	15-30 (LOAEL)	20	1	NS[d]	WHO (2000)
Nickel (Ni)	1-180	Respiratory system haematopoetic.	1.6 (HEC NOAEL) [c]	30	0.05	NS[d]	OEHHA (2001b)
NO_2	10-150	Development of recurrent upper and lower respiratory symptoms in children.	LOAEL 75-150 (40 – 80 ppb)[g]	2 on mid LOAEL	56 (30 ppb)[g]	1 yr	NEPC (1998)
Polyaromatic Hydrocarbons	Refer to text (Section A1.6)						

Compound	Annual bkgd ambient air level [µg/m³] [a]	Health endpoint	Critical effect level [µg/m³]	UF	AGV[b] [µg/m³]	Av. Time	Source
PM_{10}	15 [e]	Aggravation of existing respiratory diseases (increased use of bronchodilators among asthmatics)	Estimated (based on linear extrapolation of exposure response curves from epidemiological findings).	-	20	1 yr	EC (2004)
$PM_{2.5}$		As for PM_{10}			8	1 yr	NEPC (2003)
SO_2	0 - 130	Community patterns of respiratory illness measured by prevalence of respiratory symptoms.	100 (LOAEL in combination with particulates).	2	52 (20 ppb)[g]	1 yr	NEPC (1998)
TRS (Total Reduced Sulphur)	Taken as being the same as for hydrogen sulphide (see Section 12.4)						
Vanadium	50-200	Respiratory effects in workers	20 (LOAEL)	20[f]	1	NS[d]	WHO (2000)
Dioxins/ Furans	Refer to text (Section 10)						

[a] The source for all background levels is WHO (2000b) except for copper (WHO 1998d) and vanadium (WHO 2000).
[b] AGV is the air guideline value in units of µg/m³.
[c] HEC = Human Effect Concentration derived by converting the animal effect concentration with physiological scaling data.
[d] The averaging period is not specified (NS) however it is a chronic duration guideline value, hence the averaging time of 1 yr is used.
[e] The Department of Environment and Heritage State of the Environment 2001 Report states that the characteristic concentration which typifies the exposure for most of the population is only 15 µg/m³ PM_{10}. Characteristic was defined as 37% of the population exposed at least once per year.
[f] Since the adverse nature of the observed effects on the upper respiratory tract were minimal at this concentration, and a susceptible subpopulation has not been identified, a protection factor of 20 was selected by WHO (2000).
[g] Conversions from ppb into µg/m³ performed at 25°C and 101.7pa.

N/A = Not Available.

Table 11-6: Carcinogen potency factors

Compound	Bkgd annual ambient air level [a] [µg/m³]	Target Organ	Unit Risk [µg/m³]$^{-1}$	IARC group	Source
Arsenic (As)	0.001 – 1	Lung cancer in humans	1.5×10^{-3}	1	WHO 2000a
Cadmium (Cd)	0.001 – 0.1	Lung cancer in workers	1.8×10^{-3}	1	WHO 2000a
Chromium (Cr) Cr VI only	$5\text{-}200 \times 10^{-3}$ (total Cr)	Lung cancer in workers	4×10^{-2}	1	WHO 2000a
Nickel (Ni)	1-180	Lung cancer in humans	3.8×10^{-4}	1	WHO 2000a
Polycyclic aromatic hydrocarbons	<0.1 – 100 ng/m³	Lung cancers	8.7×10^{-2}	2A	WHO (2000)

[a] The source for all background levels is WHO (2000b).

A.1.2 Polyaromatic hydrocarbons (PAHs)

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and meat. Active or passive inhalation of tobacco smoke is a major source of exposure for many people. Cooking meat or other food at high temperatures increases the amount of PAHs in the food. There are more than 100 different PAHs which generally occur as part of complex mixtures, thus environmental exposures are not to single PAH compounds. PAHs occur naturally and enter the environment mostly as releases to air from volcanoes, burning off and bush fires, residential wood burning, and exhaust from automobiles and trucks. PAHs breakdown in air by reacting with sunlight and other substances over a period of days to weeks. In soil, water and sediment breakdown generally takes weeks to months and is primarily through the actions of microorganisms (**ATSDR 1995**).

In a total of 65 studies the most notable health effect in chronic studies with animals is cancer. This has occurred via the 5 exposure routes tested and in all 7 species tested (WHO 1998a). It is therefore a reasonable assumption that sufficient exposure of humans to PAHs by any exposure route is associated with risk of cancer induction.

Benzo(*a*)pyrene is genotoxic in a large number and wide variety of *in vitro* and *in vivo* tests that have the capacity for metabolic activation of the substance (**ATSDR 1995, WHO 1998a**).

In occupational epidemiological studies there is a clear dose related association between inhalation exposures to PAH mixtures which include benzo(*a*)pyrene, and increased risk of lung cancer. Benzo(*a*)pyrene is frequently used as a marker for PAH mixtures. It is a genotoxic carcinogen in all species tested. Generally the site of tumour formation depends on the route of exposure but is certainly not restricted to the site of application. It appears that the extent of absorption from different exposure modes plays an important role in initiation and tissue distribution of PAH – induced tumours. Obviously 'site of contact' tissues (stomach, lung and skin) experience the highest concentrations after ingestion/gavage, inhalation or dermal exposure respectively and logically would be expected to be amongst the most sensitive organs to the tumorigenicity of benzo(*a*)pyrene. This is in fact what is observed. Thus the health effect of concern for air borne PAH pollutants is lung cancer.

The concentrations of individual PAHs in ambient air around the world varies over several orders of magnitude but are generally in the range <0.1 – 100 ng/m^3. The average levels of individual PAHs in ambient air of rural areas are generally 0.1 – 1 ng/m^3, and in urban areas 1 – 30 ng/m^3 with some locations being greater than 200 ng/m^3 for specific PAHs (**WHO 1998a**). The highest predicted annual ground level concentrations of PAH from the mill in the historic Toxikos HRA was 0.08 ng/m^3. This concentration is below the rural background range of 0.1 – 1 ng/m^3 reported by WHO (**1998a**).

Each PAH varies in its potency to cause cancer. Some are not or are only very weak carcinogens, but others such as benzo(*a*)pyrene and dibenzoanthracene are quite potent. However it is evident from animal carcinogenicity feed studies that PAH mixtures, such as in coal tar, induce dose related increases in a wide variety of tumours including liver, lung, forestomach and small intestine whereas oral benzo(*a*)pyrene resulted only in forestomach tumours (**Culp et al. 1998**). It appears that the lung and liver tumours may be due to components contained in the coal tar mixture other than benzo(*a*)pyrene. While it is not known with certainty that inhaled PAH mixtures will exhibit similar differences in tumour profile and potency, the Culp et al. (**1998**) study questions the validity of either using benzo(*a*)pyrene as a surrogate for PAH mixtures or assuming simple additivity based on benzo(*a*)pyrene equivalents. Both methods of assessment could underestimate the true risk, nevertheless the assessment of public health impacts is better served by conducting risk assessments in which at least the possibility of interactive additive effects are considered rather than just doing the assessment with the benzo(*a*)pyrene content of the mixture, or assuming the entire mixture is benzo(*a*)pyrene.

It should also be noted however there is information to indicate that co-exposure to mixtures of PAHs often decreases the carcinogenic potency of the most potent PAH (usually benzo(*a*)pyrene), in the mixture, by competitive inhibition of metabolism to the pro-carcinogen or by competitive binding at the nuclear aryl hydrocarbon receptor (**WHO 1998a, ATSDR 1995**). We consider therefore that the method is more likely to overestimate carcinogenic risk than under estimate it.

In this risk assessment, the overall carcinogenic risk associated with the mixture of PAHs has been calculated as if the entire PAH mixture was just benzo(*a*)pyrene. This was done because a congener breakdown of the anticipated PAH emissions is not available at this time. This will overestimate the carcinogenic potency of the mixture because many of the congeners are not as potent in causing cancer as benzo(*a*)pyrene. The differences in potency between the various PAH congeners are shown in Table 1.1. Here the highest consensus benzo(*a)*pyrene potency equivalency factor for specific PAHs listed by the World Health Organisation (**WHO 1998a**) are listed.

Table 11-7: Potency equivalence factors for PAH congeners

PAH Congener	Potency Equivalence Factor
acenaphthene	0.001
acenaphthylene	0.01
anthracene	0.01
benzo(a)anthracene	0.1
benzo(a)pyrene	1
benzo(b)fluoranthene	0.1
benzo(ghi)perylene	0.01
benzo(k)fluoranthene	0.1
chrysene	0.01
dibenzo(ah)anthracene	1
fluoranthene	0.01
fluorene 9H-	0.001
indeno(1,2,3-cd)pyrene	0.1
naphthalene	0.001
naphthalene analogues[a]	0.001
phenanthrene	0.001
pyrene	0.001

[a] These include 2-methylnapthalene, 1,6- dimethylnaphthalene, alkylnaphthalene and trimethylnaphthalene.

Due to different assumptions and experimental approaches, estimation of the carcinogenic potency of benzo(*a*)pyrene using either epidemiological or animal data is complex. There are a range of cancer potency values (i.e. unit risk values) for benzo(*a*)pyrene that could be used to calculate cancer risk (Table A1.2). For this risk assessment we have deferred to the latest value of 8.7 x 10^{-2} (µg B(*a*)P /m^3)$^{-1}$ quoted by WHO (**2000**), this is an inhalation cancer potency value for benzo(*a*)pyrene derived from studies of coke-oven workers.

Table 11-8: Range of Unit Risk factors for inhalation exposure benzo(a)pyrene a

WHO 1987, WHO 2000[b]	8.7 x 10^{-2} (µg B(a)P /m^3)$^{-1}$
WHO 2000[c]	2 x 10^{-2} (µg B(a)P /m^3)$^{-1}$
Sloof 1989[a]	0.1 (µg B(a)P /m^3)$^{-1}$
Muller 1995a,b, 1996[a]	2.3 x 10^{-2} (µg B(a)P /m^3)$^{-1}$
OEHHA 2002	1.1 x 10^{-3} (µg B(a)P /m^3)$^{-1}$

[a] Except for WHO (2000) and OEHHA (2002) the values and references in this table are cited in WHO (1998, pp 671-672).
[b] Based on epidemiological evidence for lung tumours where B(a)P was an indicator for air borne PAHs.
[c] Based on animal data where B(a)P was present as part of a complex mixture.

A.1.3 Toxicity profiles for reduced sulphur compounds

Hydrogen Sulphide

A large proportion of pulp mill total reduced sulphur (TRS) emissions are reported to be present as hydrogen sulphide (H_2S) (**Haahtela et al. 1991, Marttila 1995**) and if measures were not taken to remove this substance from the process it would likely be the single largest TRS compound to be found in the environment (Nebraska DEQ 1997). Hydrogen sulphide can quickly become a nuisance, able to be detected in air by some people as low as 8 µg/m^3 (0.005 ppm) (**Amoore 1985**) with there being a good correlation between air levels and odour intensity (Suarez et al. 1998). Levels between 1-5 ppm (1,500-7,600 µg/m^3) are reported to be moderately offensive and may cause nausea and headaches upon prolonged exposure (**IPCS 2005**).

An assessment of studies of healthy individuals exposed to levels of H_2S between 0-100 ppm found that young healthy adults can tolerate up to 10 ppm (15,200 µg/m^3) without significant effects (**Davies and Haggerty 2002**). Animals were also found to tolerate short-term H_2S exposures up to 35 ppm (53,200 µg/m^3). The effects observed, in the majority of cases, were of a minor nature and only displayed at the local cellular level (**Davies and Haggerty 2002**). Eye irritation has been reported to generally occur at a concentration of 76,000 µg/m^3 (50 ppm) (**Sullivan & Krieger 1992**) with the irritation threshold between 15,000-30,000 µg/m^3 (10-20 ppm) (Savolainen, 1982), although conjunctivitis "sore eyes" has been observed with exposures in occupational environments from 5,000-7,600 µg/m^3 (3.3-5 ppm) (**Sullivan & Krieger 1992; Vanhoorne et al. 1995**). At higher concentrations of between 30,000-76,000 mg/m^3 (20-50 ppm) irritation to the nose, throat and lung increases and there may be digestive upset and loss of appetite. At these levels the sense of smell can become "fatigued" and odour cannot be relied upon as a warning of exposure (**IPCS 2005**). At an H_2S air level of about 225,000 µg/m^3 (150 ppm) the olfactory system can be paralysed (**IPCS 1981**).

Serious conjunctival irritation is caused at concentrations between 70,000-140,000 µg/m^3 (50-100 ppm) (**Savolainen 1982**) while at higher concentrations respiratory symptoms are the predominant symptom, with a risk of pulmonary oedema at concentrations of about 400,000 µg/m^3 (263 ppm) (**WHO 2000**). At 700,000 µg/m^3 (460 ppm) exposure to H_2S can be fatal (**Beauchamp et al. 1984, Hirsch and Zavala 1999**) with immediate collapse occurring at extremely high concentrations of around 1,400,000 µg/m^3 (920 ppm) (**WHO 2000**). At concentrations of 1,500-3,000 mg/m^3 (1000-2000 ppm) strong stimulation of the central nervous system occurs resulting in hyperpnoea (rapid breathing) leading to apnoea (inactivity), convulsions, unconsciousness, and death (**IPCS, 1981**). A summary of the dose-response relationship for H_2S is depicted in Table 11-9.

A comparative study of the effects of H_2S exposure between pulp mill workers and asthmatics was investigated by monitoring workers exposed to H_2S in the workplace at levels between 2-7 ppm (3,000-10,600 µg/m^3) and by exposing asthmatic individuals in chambers to 2 ppm H_2S (3,000 µg/m^3) for 30 minutes (**Jäppinen et al. 1990**). No significant changes in respiratory function were observed in either group, but in asthmatic subjects airways resistance was increased by 26.3% and specific airways conductance decreased by 8.4% , however these changes were not statistically significant and did not result in clinical symptoms (two subjects displayed slight bronchial obstruction with changes >30% in each metric). Asthmatic subjects initially found H_2S odours unpleasant, but rapidly became accustomed to it, sensed nasal and pharyngeal dryness and three of ten exposed individuals complained of headaches (**Jäppinen et al. 1990**).

In terms of low dose chronic exposures Bates and co-workers studied residents of Rotorua, New Zealand, a geothermal area with elevated H_2S levels, and found from hospital discharge data significant increases in the incidence of diseases of the nervous system and the eye. The effects were considered somewhat characteristic of H_2S exposures, but a note was made that there may have been confounders not accounted for in the study (**Bates et al. 1998**). Another study by Bates and co-workers reports the median H_2S concentration in Rotorua as 30 µg/m^3 (0.02 ppm), with 35% of the measurements >70 µg/m^3 (0.046 ppm) and 10% >400 µg/m^3 (0.26 ppm) (**Bates et al. 1997**). These levels are comparable to the levels measured in the vicinity of the pulp mills of South Karelia that were found to be associated with eye irritation, nasal symptoms and cough (**Jaakkola et al. 1990b; Haahtela et al. 1992**) but are much higher than the levels predicted in the areas surrounding the mill.

There is some limited epidemiological evidence that H_2S exposures might be associated with a slight increase in cardiovascular disease (**Jappinen and Tola 1990; Bates et al. 2002**) although studies of healthy individuals exposed at H_2S levels up to 10 ppm for 30 minutes found no cardiovascular effects (**Bhambhani and Singh 1991; Bhambhani et al. 1994 & 1997**) and animal studies indicate that cardiovascular effects occur at higher concentrations (**Kohno et al. 1991; Kosmida et al. 1967**).

Table 11-9: Dose-response relationships for hydrogen sulphide.

Effect	(mg/m^3)	(ppm)	Reference
Respiratory paralysis, collapse & death	1500-3000	1000-2000	IPCS (1981)
Strong CNS stimulation, hyperpnoea followed by respiratory arrest	750-1400	500-920	IPCS (1981) Beauchamp et al. (1984) Hirsch & Zavala (1999)
Pulmonary oedema with risk of death	450-750	300-500	IPCS (1981) WHO (2000)
Loss of olfactory sense	210-350	150-230	IPCS (1981) Savolainen (1982)
Serious eye damage	70-140	50-100	Savolainen (1982)
Increasing irritation to eyes, nose & respiratory tract. LOAEL for body weight loss (rats)	30-76	20-50	IPCS (2005)
Threshold for eye irritation	15-30	10-20	Savolainen (1982)
LOAEL increase in blood lactate level	7.6	5	Bhambhani & Singh (1991)
Increase in eye complaints	5	3.3	Sullivan & Krieger (1992) Vanhoorne et al. (1995) IPCS (2005)
LOAEL for bronchial constriction in asthmatics	3	2	Jäppinen et al. (1990)
Threshold for odour detection	0.008 0.0016	0.005	Amoore (1985) AIHA (1989)

The International Agency for Research on Cancer has not reviewed hydrogen sulphide, and recent consideration by the US EPA determined that the data was inadequate for an assessment of the carcinogenic potential of H_2S (**US EPA 2003a**). Roth and Goodwin (2003) report several papers that have investigated the relationship between exposure to environments that contain H_2S and cancer incidence with several of these studies reporting an increased incidence in a variety of cancers in pulp mill workers including lung, kidney, liver and brain cancers (**Schwartz 1988; Solet et al. 1989; Band et al. 1997; Langseth & Anderson 2000, Band et al. 2001, Lee et al. 2002**); as found for various other non-pulp mill emission sources that include H_2S (**Bates et al. 1998, Andersson et al. 2001**). However, these studies each involve exposures to H_2S in a mixture of other compounds with ATSDR concluding that there is limited data on the potential of H_2S to induce cancer in humans ( ).

A.1.4 Derivation of guideline values for criteria pollutants

Carbon Monoxide

CO binds with haemoglobin to form carboxyhaemoglobin (COHb), and when formation of this compound is high enough the oxygen carrying capacity of the blood decreases to such an extent that tissues highly dependent on oxygen can't get enough to function properly. Thus the toxic effects of CO become evident in organs and tissues with high oxygen consumption such as the brain, the heart, exercising skeletal muscle and the developing foetus. This is especially so when tissue oxygen utilisation is already compromised such as in people with ischemic heart disease. Because COHb stays longer in the foetus than in the pregnant mother the foetus is more vulnerable to the effects of CO than is the mother. We note asthmatics are not more sensitive to the effects of CO than are healthy people. In healthy subjects COHb levels are normally about 0.4–0.7%. The World Health Organisation recommends a COHb level of 2.5% should not be exceeded and have accordingly set an ambient air quality guideline. The Australian NEPC adopted the WHO recommendation (Streeton 1997). On the other hand California EPA estimate the no observed effect level to be 1.1-1.3% COHb and have set their guideline on the amount of CO that does not lead to COHb blood levels greater than those associated with this no effect level.

Nitrogen Dioxide

Only very high concentrations of NO_2 (approximately 2,000 µg/m^3 (~1,050 ppb)) affect breathing in healthy people[g]. However small changes in lung function (< 5%) and changes in airway responsiveness have been reported in several studies of sensitive asthmatics or the elderly exposed to concentrations as low as 375-575 µg/m^3 (~200-300 ppb) over 20 minutes to 4h (Bauer et al., 1986; Bylin et al., 1988; Roger et al., 1990; Morrow et al., 1992; Strand et al., 1996, 1997, Streeton 1997). These levels represent a clear low-observed-effect level (LOEL) for NO_2 based on increased responsiveness in mild asthmatics to bronchoconstrictors or in subjects with chronic obstructive pulmonary disease (COPD). The study by Bauer et al (1986) did not find a significant change in pulmonary function when asthmatics were exposed to 560 µg/m^3 NO_2 when resting, with decreases recorded only after the subjects exercised. Similarly, testing asthmatics the day after exposure to 490 µg/m^3 NO_2 did not decrease lung function before allergen challenge (Strand et al., 1997).

The identification of an obvious no effect level is less clear but it seems to be around 200 µg/m^3 (approx 100 ppb). Studies have shown that effects can be detected in mild asthmatics after short-term exposure to 488-500 µg/m^3 (260-240 ppb) NO_2 who are subsequently exposed to an inhalation challenge (Strand et al., 1996, 1997; Kraft et al., 2005). However, in a study where mild asthmatic subjects were exposed for 1h to 200 µg/m^3 (~100 ppb) NO_2 and then immediately exposed to a house dust mite challenge, the late asthmatic response (as tested using forced expiratory volume in one second; FEV_1) was found to be greater than when compared to air (NO_2 -7.76% vs. Air -2.85%), but the results were not found to be significant (Tunnicliffe et al., 1994). The current air guideline for acute exposure to NO_2 in the NEPM is 0.12 ppm (226 µg/m^3) measured as a 1h average.

According to Streeton (1997) there is an increasing body of evidence to suggest that longer term (years) ambient exposure to significantly lower concentrations of NO_2, of the order of 40 - 80 ppb (approx 75-150 µg/m^3) during early and middle childhood years can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school.

[g] Conversions at 25^{0}C and 101.7pa are: ppb = µg/m^3 x 0.53; µg/m^3 = ppb x 1.88. Many unit conversions in this section have been rounded.

Similarly, more recent studies of self-reported asthmatic individuals living in homes with flue-less gas heaters have shown significant effects of NO_2 exposures to those aged ≤14 years with chest tightness, breathlessness on exertion and asthma attacks experienced either the same day or with one day lag (Smith et al., 2000). The range of median indoor levels of NO_2 measured by positional passive sampler in homes during this study were indicated to be between 0-147 ppb (0-277 µg/m^3) with time weighted average levels measured by personal passive sampler of 0-1,760 ppb (0-3,300 µg/m^3)[h]. Subsequent investigations with flue-less space heaters in primary schools indicated that over the 12 week winter heating period asthma symptoms were significantly higher[i] in children exposed to gas combustion products with mean NO_2 levels of 47.0 ppb (88 µg/m^3) versus children in schools where a replacement intervention programme had removed or replaced the flue-less gas heaters, leading to a mean NO_2 level of 15.5 ppb (29.3 µg/m^3) (Pilotto et al., 2004).

Based upon a review of the literature, Streeton (1997) considered short-term ambient exposures to 200-300 ppb (375-565 µg/m^3) NO_2 and chronic exposures between 40-80 ppb (75-150 µg/m^3) capable of causing recurrent upper and lower respiratory tract symptoms, an increased incidence of respiratory infection and onset of symptoms in mild asthmatics. Streeton considered these effects as a low observed adverse effect levels (LOAEL) and has suggested that an uncertainty factor of 2 need apply to account for susceptible people within the population therefore establishing a short-term guideline in the range 100-150 ppb as a 1h average and a chronic guideline between 20-40 ppb for longer term exposures as an annual average (Streeton, 1997).

The WHO (1997, 2000) took a different approach to reach a similar conclusion. Similar to Streeton, the WHO noted the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures however the WHO (1997) state this is supported by animal toxicological findings showing increased susceptibility to respiratory infections and impairment of host defences as a result of subchronic or chronic exposures to NO_2 concentrations near ambient concentrations (i.e. 20-60 µg/m^3; 11-32 ppb). On the basis of a background level of 15 µg/m^3 (8 ppb) as determined in Finland during the 1980s (Jaakkola et al., 1991) and the fact that significant adverse health effects occur with an additional concentration of 28.2 µg/m^3 (15 ppb) or more, which is an estimate of an increased risk of about 20% for respiratory symptoms and disease (Hasselblad et al., 1992; WHO, 1997), an annual guideline value of 40 µg/m^3 (22 ppb) was derived by the WHO (1997). The WHO considers the guideline value will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined was emphasised.

Interactive effects with allergens in Humans

There is some evidence to suggest that NO_2 exposure can enhance the response of an asthmatic to allergens. Volunteers with mild asthma exposed to 400 ppb (820 µg/m^3) NO_2 for 1h and who then immediately underwent a fixed-dose house dust mite challenge displayed significant decreases in FEV_1 results for early (2h after allergen; -18.64%, $p<0.009$) and late (-8.13%, $p<0.02$) phase asthmatic responses compared to air (-14.92% & -2.85 respectively) (Tunnicliffe et al., 1994).

[h] The mean daily maximal 10 minute levels of pollutants measured outdoors were 9.8 ppb (19 µg/m^3) NO_2; 2.9 ppb (8.3 µg/m^3) SO_2; 17.5 ppb (37.5 µg/m^3) O_3.

[i] Unadjusted relative risks (RR) were 0.32 for difficulty breathing at night p=0.004; RR 0.41 for difficulty breathing during the day p=0.045; RR 0.45 chest tightness during the day p=0.008; RR 0.39 asthma attacks during the day p=0.034.

Similarly, subjects with mild asthma and allergies to birch or grass pollens, who were exposed on four consecutive days to 500 µg/m^3 (265 ppb) NO_2 for 30 minutes, had a significantly increased asthmatic response after exposure to NO_2 and allergen (non-symptomatic dose 4h after NO_2) with a fall in early phase (15 minutes following allergen exposure) forced expiratory volume in one second of -25% for NO_2 compared to -0.4% for air, which was still significant (p=0.01) 3-10h after allergen exposure (Strand et al., 1997; Strand et al., 1998). Similar findings have been made with ozone exposures followed by inhalation of grass pollens (UK DoE, 1996). The delayed effect of bronchial responsiveness has been investigated and it was found that 110 µg (median) of histamine diphosphate (vs. 203 µg on air) was required as the provocative dose 5h after 30 minutes exposure to 488 µg/m^3 (260 ppb)[j] NO_2 to cause 100% increase in specific airways resistance (Strand et al., 1996).

Conclusions:

- Concentrations of around 2,000 µg/m^3 (~1,000ppb) are needed to affect respiration of healthy people.
- The low effect level for increased bronchial reactivity in sensitive asthmatics is
- 375-575 µg/m^3 (~200-300 ppb) for exposures from 20 minutes up to 4hours.
- The no effect level for increased bronchial reactivity is ~200 ppb.
- The increased bronchial reactivity may remain for up to 10 hours after cessation of NO_2 exposure.

Particulate matter (PM_{10})

The detailed toxicological mechanism(s) by which particulate matter causes adverse health effects is not known. Most of the data indicates a role for oxidative stress causing inflammation and immunotoxicity in airways and lungs, or a mechanism involving impairment of respiratory and cardiac neurological functions. According to a publication from the Dutch Institute of Public Health and Environment [k] (RIVM 2002), rather than assigning the observed association between particulate matter and adverse health effects to the toxicity of particulate matter itself, a more plausible explanation is that the association is the result of reduced capacity of an individual to withstand stress and maintain a stable, relatively constant internal environment. It is therefore plausible, and indeed consistent with observations, that the population at risk is largely defined in particular by individuals with failing health, attributable to ageing or illness (RIVM 2002).

Populations shown to be susceptible to the effects of airborne particles are primarily those with compromised health, especially respiratory and/or cardiopulmonary function. At risk groups include the elderly, people with existing respiratory disease such as asthma, chronic obstructive pulmonary disease (COPD) and bronchitis; people with cardiovascular disease; people with pulmonary infections such as pneumonia; and children (Streeton 1997). In relation to the data underpinning establishment of the national ambient air standard (NEPC 1998) most of the 'effects' due to particles are associated with exacerbation of existing disease states. The 'effects' observed with elevated PM_{10} concentrations are increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increases in prevalence of pulmonary symptoms and increased mortality (Streeton 1997, RIVM 2002).

[j] Mean Temperature 25.5±0.6°C

[k] Although published by the Dutch Institute of Public Health and Environment (RIVM) the report was a collaborative effort of several Dutch institutes.

Ambient air quality standards around the world are based primarily on data obtained from large urban populations where background incidences of the 'effects' are measurably increased during episodes of high ambient PM_{10}. Dose response relationships are commonly articulated in terms of percentage increase of effect per 10 µg/m^3 increase in PM_{10}; for example changes in daily mortality are typically estimated at approximately 0.5 – 1.5% per 10 µg/m^3 increase in PM_{10} (Pope 2000).

From daily time series studies (these look at associations between short term, acute exposures to elevated ambient PM_{10} and various population health measures) there is increasing evidence the health effects associated with particulate matter may occur at quite low particulate levels, and also primarily with the $PM_{2.5}$ respirable fraction, furthermore studies have not identified a clear no effect(s) threshold (EU 2004, RIVM 2002, US EPA 2003a, WHO 2000). It should not be assumed that only the most old and most frail who may be very near death, are at risk of dying from PM_{10} exposure. There is evidence that mortality may also be advanced in other 'at risk' groups (Zeger et al. 1999). In addition the number of those susceptible to less serious health effects such as increased respiratory symptoms, decreased lung function, or other physiologic changes may be broader than those at risk of dying. For most people, these effects are likely to be small, transient, and maybe even unnoticed. For a few, the decline in lung function may be clinically relevant resulting in increased bronchodilator use and/or emergency hospital visits, or the effects may result in short-term absence from work or school (Pope 2000).

In producing the health effects document for the Australian ambient air quality NEPM, Streeton (1997) considered there was satisfactory evidence that PM_{10} pollution from crustal sources was significantly less harmful than that generated from combustion processes. Similarly more recent studies, summarised in RIVM (2002) and US EPA (2003), implicate ambient PM_{10} derived from fossil fuel combustion and vegetative burning, but not crustal particles, as important contributors to observed mortality effects. Laden et al. (2000) also concluded that fine combustion particles from mobile and coal combustion sources, but not fine crustal particles are associated with increased mortality.

The US EPA (2005) are currently considering a more narrowly defined indicator for thoracic coarse particles that would protect public health against effects linked with thoracic coarse particles present in urban areas. The US EPA have considered epidemiologic studies that examined exposures to particles generally found in urban environments and exposures to natural crustal materials and found that urban thoracic coarse particles are of concern to public health, in contrast to uncontaminated natural crustal dusts.

In summary, there is general agreement in the scientific literature that there is a concentration-response relationship (with no indication of a threshold) between PM_{10}/$PM_{2.5}$ and various measures of population based health effects. The exact form of the relationship is unclear, depending upon the health measure some studies indicate the relationship to be linear while others suggest non-linearity (RIVM 2002). It is noted that people with compromised respiratory or cardiopulmonary function (either through disease or old age) are more susceptible to the effects of particulates.

Sulphur dioxide

It is clear from a variety of reviews on SO_2 (US EPA 1994, 1996b, Streeton 1997, CalEPA 1999c, EC 2005) that healthy, non asthmatic individuals are essentially unaffected by acute exposures to SO_2 when concentrations are about 1-2 ppm (approximately 2,800 – 5,700 µg/m^3), and that the population of concern for the effects of short-term (approximately 5 – 15 minutes) SO_2 exposure consists of individuals who are mild to moderate asthmatics, are SO_2 responders and are undertaking exercise/activity that raises ventilation rate[l]. However not all asthmatics in these circumstances will experience adverse effects on exposure to SO_2 even at concentrations > 0.6 ppm. The proportion of asthmatic individuals who respond, the magnitude of the response and the occurrence of symptoms increase as SO_2 concentration and ventilation rates increase.

There have been two general approaches to standard setting in light of the above concentration response information for SO_2 responsive exercising asthmatics:

a ***Establish a 1 hour standard and guidance for local jurisdictions for interpreting short term exceedances.*** A discussion of whether to adopt a 10 minute sulphur dioxide standard is ongoing in Australia, however it is likely that a national 10 minute advisory standard for SO_2 will not be adopted, but instead a non-statutory guideline value will be developed to assist in health impact assessments of SO_2 emissions from individual sources [m]. This approach is similar to the approach taken in the United States. In 1994, 1996 and finally in 1998, the US EPA declined to establish a short term (5 or 10 minute) National Ambient Air Quality Standard (NAAQS) for SO_2. They argued that short term exposures to concentrations of SO_2 sufficient to produce adverse effects over 5 minutes were associated with point source emissions, were infrequent and affected only a small portion of the susceptible population (i.e. exercising asthmatics) hence a NAAQS for the whole population was not warranted. However the US EPA have proposed an 'Interim Protection Level Program' to assist states in managing risks associated with short term (5 minute) exposures but allowing due cognisance to be given to specific local factors in deciding whether adverse health risks were likely.

b **Establish a short term guideline value.** The WHO (2000) has established a 10 minute guideline value of 0.175 ppm however this is currently under review; the next edition of the WHO guidelines is expected to be published in 2006.

The UK (EPAQS 1995) considered that most of the acute clinical studies did not show an effect below 250 ppb and although 'occasional subjects' had responded to lower concentrations with transient changes in measurements of lung function, these transient changes were "*insufficient to be associated with symptoms*". Furthermore it was considered that the transient changes occurred in

[l] SO_2 is water soluble and much is scrubbed out of the inspired air by mucous in the upper respiratory tract, mouth breathing tends to bypass this protective mechanism. In addition increased breathing rate with exercise is accompanied by increased depth of breathing; this delivers more SO_2 to the bronchioles and lung parenchyma.

[m] Based on personal communication with the NEPC Secretariat, February 2005.

asthmatic subjects breathing through a mouthpiece while exercising on a bicycle thus the normal protective mechanism of nasal mucosal scrubbing of SO_2 was bypassed. The Panel recommended an Air Quality Standard for SO_2 in the United Kingdom of 100 ppb, measured over a 15 minute averaging period. Dose response analysis suggests an effect would not be anticipated in sensitive individuals until the UK guideline had been exceeded 2 -3 fold. The rationale used by the UK panel for setting such a low standard was that exacerbation of asthmatic symptoms could occur with exposures as short as 1 minute however it is impractical to measure SO_2 over this very short time frame. Fifteen minutes was considered the shortest practical period for measurement. Nevertheless this period of measurement could include very brief times of higher concentrations, which could be as much as double the average, and therefore have an effect on susceptible individuals when the average appears safe. The UK panel took this into consideration, as well as the need for an adequate margin of safety for those individuals more severely affected by asthma when they set a level of 100 ppb measured over a 15 minute averaging period. In essence the UK DoE took a NOAEL of 200 ppb and applied a safety factor of 2 to account for SO_2 spikes within the 15 min averaging measurement.

California EPA (Cal EPA 1999) based on a thorough review of the literature consider the LOAEL for 5-75 minute SO_2 exposure of moderately exercising asthmatics to be 0.4 – 0.5 ppm, with a NOAEL of 0.2 - 0.25 ppm which they believe would not result in discomforting respiratory effects in sensitive individuals, i.e. exercising asthmatics, for a period of 1 hour. The California EPA did not apply their traditional uncertainty factors for intraspecies variability in response and set an acute reference exposure level (REL) of 0.25 ppm. They state "*This level is felt to protect asthmatic individuals because adverse effects are consistently observed only at higher concentrations under conditions of moderate exercise (ventilation rates of >40 L/minute) and there is inconsistency in response to SO_2 exposure at lower concentrations*".

As outlined in Table A1.7, the Australian NEPC as well as WHO and UK have established a chronic standard for sulphur dioxide of 0.02 ppm as an annual average. The justifications for this standard provided by the WHO, NEPC (Streeton 1997) and UK (UK DOE 2000) include:

- According to Ayres (1998) the WHO assessed the available literature on long term exposure to SO_2 in 1990 (WHO, 1992) and concluded that:
  - With chronic exposure to SO_2 concentrations of 250 µg/m^3 (87 ppb) there was a measurable increase in respiratory morbidity amongst susceptible adults suffering COPD, and perhaps also in children, with the effect being more marked at concentrations of around 400 µg/m^3 (140 ppb).
  - At chronic SO_2 levels of approximately 200 µg/m^3 (70 ppb), quite small transient reductions in lung function could be seen in children and adults that could last for as much as two to four weeks.
  - Based on prevalence of respiratory symptoms the lowest observed adverse effect level of SO_2 in epidemiological investigations was judged to be 100 µg/m^3 (35 ppb) as an annual average. From this an air quality guideline of 20 ppb was set.
  - At that time i t was not possible for the epidemiological studies to reliably distinguish between the effects of particulates or SO_2. There was uncertainty as to whether or not SO_2 was in fact

responsible for the observed adverse health effects, or whether it might be acting as a surrogate for another pollutant, especially particulates.

- Since then further review by the WHO (2000b) concluded:
  - *"more recent studies related to industrial sources, or to the changed urban mixture, have shown adverse effects below the LOAEL identified in 1990, but a major difficulty in interpretation is that long-term effects are liable to be affected not only by current conditions but also by the qualitatively and quantitatively different pollution of earlier years"*. The effects noted were increased prevalence of respiratory symptoms and small reductions in lung function in children.
  - There still remains considerable uncertainty as to whether or not SO_2 is in fact responsible for the observed adverse health effects associated with air pollution.
  - It is also noted that WHO (2000b) considered the epidemiology data to consistently demonstrate effects on mortality and hospital emergency admissions for total respiratory causes and chronic obstructive pulmonary disease at levels of exposure lower than mean annual levels of 50µg/m^3 with daily levels usually not exceeding 125 µg/m^3.

Notwithstanding their assessment that population effects were occurring at exposure concentrations below the recommended air quality guidelines, the World Health Organisation did not amend the chronic air quality guideline from the 20 ppb value set in 1987 (WHO 2000b, WHO 1987). Reasons for this decision were not provided. They did however point out that unlike the 1987 guidelines the recommended guidelines in 2000 were not linked with particulate matter.

Conclusions

- The no observed effect level for acute exacerbation of asthma symptoms in SO_2 responding exercising asthmatics is about 200 ppb.
- The low effect level for acute effects is approximately 600 ppb.

Table 11-10: Summary of derivation of guideline values for criteria pollutants.

Guideline µg/m³	/ ppb		Derivation	Reference
Carbon monoxide (CO)				
29,000	33,222	Design criteria 1 hr average	Unknown	**DPIWE (2004)**
23,000	26,349	Inhalation reference exposure level (REL) 1 hr average	Prevention of angina in persons with known angina and cardiovascular diseases who are exercising heavily. No observed effect level 1.1 – 1.3% COHb which corresponds to 23,000 µg/m³ calculated toxicokinetically.	**Cal EPA (1999a)**
30,000	34,368	Ambient air quality guideline 1 hr average	To protect non-smoking, middle-aged and elderly population groups with documented or latent coronary artery disease from acute ischaemic heart attacks, and to protect the foetuses of non-smoking pregnant women from untoward hypoxic effects, a COHb level of 2.5% should not be exceeded. This is achieved at the guideline level. The guideline takes into account all the known physiological variables affecting carbon monoxide uptake.	**WHO (2000a&b)**
10,310	9,000	Ambient air quality guideline 8 hr avg	Consistent with the recommendations of the WHO see description of the WHO guideline above.	**NEPC (1998), Streeton (1997)**
Nitrogen dioxide (NO_2)				
300[b]	160	Design criteria 1 hr average	Unknown	**DPIWE (2004)**
226[b]	120	Ambient air quality guideline 1 hr average	The Australian National Environmental Protection Council ambient air quality standard. It is based on a low observed adverse effect level (LOAEL) of 0.2 to 0.3 ppm derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of 2 to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL.	**NEPC (1998), Streeton (1997)**
470	250	Inhalation reference exposure level (REL) 1 hr average	The REL is also the ambient air quality standard of California. It is the no observed adverse effect level in sensitive asthmatics for NO_2 mediated increased responsiveness to other bronchoconstrictors (e.g. exercising in cold air).	**Cal EPA (1999a)**
200	106	Ambient air quality guideline 1 hr average	Lowest concentration causing small (~5%) changes in lung function in mild asthmatics is 560 µg/m³. Some but not all studies show increased responsiveness to bronchoconstrictors at NO_2 levels as low as 376–560 µg/m³. In other studies, higher levels had no such effect. Allergen challenges showed no effects at 190 µg/m³. According to WHO there have been no studies of 1 hour exposures to NO_2 at 100µg/m³.	**WHO (2000b)**
40	21	Ambient air guideline Annual avg	WHO (1997) reviewed the epidemiological studies suggesting human health effects associated with long-term NO_2 exposures. On the basis of a background level of 15 µg/m³ (8 ppb) and the fact that significant adverse health effects could be expected occur with an additional level of 28.2 µg/m³ (15 ppb) or more, an annual guideline value of 40 µg/m³ (0.023 ppm) was derived by the WHO (1997).It is considered guideline will be protective of most serious effects. The fact that a no-effect level for subchronic or chronic NO_2 exposure concentrations has not yet been determined should be emphasized.	**WHO (2000b), WHO (1997)**

56	30	Ambient air guideline Annual avg	A low observed adverse effect level (LOAEL) of the order of 40 - 80 ppb (approx 75-150 µg/m^3) during early and middle childhood years can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent 'colds', a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school. An uncertainty factor of 2 was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 ppb (38-75 µg/m^3).	**NEPC (1998), Streeton (1997)**
Particulates (PM_{10})				
150 µg/m^3	N/A	Design criteria 24 hour	Unknown	**DPIWE (2004)**
50	N/A	Ambient air quality guideline 24 hour avg	The Australian National Environmental Protection Council ambient air quality standard was based on increased hospital visits and/or admissions for respiratory conditions, decrements in pulmonary function (especially in adults with obstructive airways disease but also in young children), increased prevalence of pulmonary symptoms and increased mortality.	**NEPC (1998), Streeton (1997).**
50[d]	N/A	European Union Limit value 24 hour avg	The European limit value is based on the lowest reasonably practical value. The European review was unable to identify a threshold concentration below which ambient PM has no effect (see WHO description below) therefore the limit value was based on the lowest reasonably practical value.	**EU (2004)**
150	N/A	National air quality standard Annual average[c]	The national air quality standard of 150 µg/m^3 with no more than one expected exceedance per year was first promulgated in 1979. The basis for the standard is not described in recent USEPA reviews of PM standards (USEPA 1996, USEPA 2005) It is important to note the standard is under review. The USEPA is currently considering whether to revise the primary standard for coarse particulate matter to be specific for urban particulate matter. Non urban sources are not consistently associated with health effects (USEPA 2005).	**US EPA (2004)**
50	N/A	National air quality standard Annual average[c]	The national air quality standard of 50 µg/m^3 was first promulgated in 1979. The basis for the standard is not described in recent USEPA reviews of PM standards (USEPA 1996a, USEPA 2005)	**US EPA (2004)**
40	N/A	European Union Limit value Annual avg	The European limit value is based on the lowest reasonably practical value. The Europeans reviewed the findings of the WHO and studies published since the WHO review and concluded that some studies suggest that long-term exposure to particulate matter is associated with possible effects below 20 µg/m^3 (as $PM_{2.5}$) or 30 µg/m^3 (as PM_{10}).	**EU (2004)**
No recommendation for acute or chronic duration.			WHO concluded that the existing database of studies did not enable the derivation of specific guideline values for either acute or chronic duration. The database of studies did show clear and consistent associations between concentrations of particulate matter and adverse effects on human health at low levels of exposure commonly encountered in developed countries. Associations were found for increased lower respiratory symptoms and reduced lung function in children, chronic obstructive pulmonary disease and reduced lung function in adults as well as mortality. Fewer studies were available investigating associations between PM and chronic health effects however some studies identified a reduction of life expectancy (in the order of 1–2 years), increased prevalence of bronchitis symptoms in children, and reduced lung function in children and adults. These effects were	**WHO (2000a & b)**

			considered to be observed at annual average concentration levels at or below current background levels (i.e. below 20 µg/m³ (as $PM_{2.5}$) or 30 µg/m³ (as PM_{10})). For this reason, no guideline value for long-term average concentrations is recommended.	
Sulphur dioxide (SO_2)				
459	175	Ambient air guideline 10 min avg	The WHO considered that only small changes of non clinical significance were seen at 524 µg/m³ (0.2 ppm).	**WHO (2000b)**
262	100[e]	Ambient air guideline 15 min avg	The UK (EPAQS 1995) considered that most of the acute clinical studies did not show an effect below 250 ppb. For the NOAEL of 200 ppb a guideline was derived by applying a safety factor of 2 to account for SO_2 spikes within the 15 min averaging measurement resulting in an ambient air guideline of 100ppb.	**UK DOE (2000)**
524	200	Design criteria 1 hr average	Unknown	**DPIWE (2004)**
524	200	Ambient air quality guideline 1 hr average	The derivation of this value could not be found from the documentation of the NEPC ambient air guidelines. Streeton (1997) does not discuss a 1 hour guideline value as the short term effects of SO_2 occur over a time frame of between 5 and 15 minutes.	**NEPC (1998), Streeton (1997).**
655	250	Acute Reference Exposure Level 1 hour	Considered that most of the acute clinical studies did not show an effect below 250 ppb (i.e. NOAEL in exercising asthmatics).	**CalEPA (1999c)**
210	80	Ambient air quality guideline 24 hr avg	Based on epidemiological studies in which the effects of SO_2, particulates, and other associated pollutants are considered. These studies mainly focus on the exacerbation of symptoms in groups of selected sensitive subjects, with symptoms generally developing when SO_2 levels exceeded 0.087 ppm (250 µg/m³), usually in the presence of particulates (PM_{10}). Thus the Australian 24 hr guideline appears to be based on a LOAEL without application of a 2-fold safety factor to account for susceptible populations. WHO (2000a&b), UK DOE (2000), and EC (2005) all apply a two-fold safety factor to derive a 24 hr guideline value of 40 or 47 ppb.	**NEPC (1998), Streeton (1997).**
123	47[e]	Ambient air guideline 24 hr avg	Based on epidemiological studies in which the effects of SO_2, particulates, and other associated pollutants are considered. These studies mainly focus on the exacerbation of symptoms in groups of selected sensitive subjects, with symptoms generally developing when SO_2 levels exceeded 87 ppb (250 µg/m³), usually in the presence of particulates (PM_{10}). The LOAEL is divided by 2 to account for susceptible people.	**UK DOE (2000)**
52	20	Ambient air quality guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m³ (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	**NEPC (1998), Streeton (1997).**
52	20	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m³ (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	**WHO (2000b)**
52	20[e]	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m³ (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	**UK DOE (2000)**
52	20[e]	Ambient air guideline Annual avg	Based on prevalence of respiratory symptoms the LOAEL of SO_2 in epidemiological investigations was judged to be 100 µg/m³ (35 ppb) annual average. From this an air quality guideline of 20 ppb was set.	EC (2005)

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APPENDIX B SCREENING FOR SECONDARY EXPOSURE PATHWAYS

B.1.1 Introduction

As depicted in **Figure 4-1** exposure to emissions from the mill can occur through secondary exposure pathways. The process of evaluating health risks from exposure via secondary pathways is difficult and uncertain because empirical relationships for the movement of most substances from air to other media, which humans may be exposed have not been established. Thus the evaluation of secondary exposure pathways inherently requires many postulates which impart unquantifiable uncertainties to the assessment. Consequently in most risk assessments the secondary exposure pathways are either not considered or unrealistic conjectures are made to establish gross worse case scenarios. In lieu of these approaches a pragmatic alternative is to perform screening assessments to evaluate the need for, and hence benefit from a detailed multi-pathway risk assessment. Usually the pre-evaluation is qualitative and based on the assessor's experience. In this risk assessment the pre-screens have been structured to provide a more objective and transparent process for deciding whether secondary exposure pathways are required to be evaluated.

Chemicals of interest for potential evaluation of secondary pathways:

Inspection of the emissions from the mill indicate that the substances likely to be of interest, with regard to secondary exposure pathways are dioxin like substances, metals and polycyclic aromatic hydrocarbons (PAHs). Dioxins are evaluated in Section 9; the evaluation takes into consideration exposures by pathways other than inhalation and tactility accounts for the time it takes to reach environmental equilibrium at the specified dioxin emission rate and bioaccumulation in human food sources.

It is recognised that many of the above chemicals are bound to particulates and may be deposited directly onto soil and vegetation.

In regards to vegetation deposition; the extent to which humans may be exposed by this direct deposition pathway will be influenced by how much particulate is deposited, the extent to which rain washes particulates off vegetation, the extent of washing fruit and vegetables before consumption, and how much of the fruit and vegetables consumed are sourced from the local area. Assuming the vast majority of vegetables and fruit will originate from areas outside of the mill emission dispersion zone and most of the time the food will be washed prior to consumption then exposure via direct deposition will be very small. This premise is supported by comparison of predicted annual ground level air concentrations with background concentrations from remote and rural areas around the world (**Table 11-15**).

Dioxins

For dioxins, the screening procedure is based upon the proportional relationship between intake via inhalation and other exposure pathways under steady state environment-body burden that has been established by many international studies. In the analysis, a conservative estimate of background intake[n] by Australians is factored into the process. The calculated increase over background monthly intake of dioxin like substances from the emissions is very small (less than 0.03%) and the total intake, including background, is much less than the tolerable monthly intake recommended by Australian health authorities. It is therefore concluded that the low level of dioxin emissions from the mill do not present a likely human health risk from direct and/or indirect exposures, and that it is not necessary to conduct a detailed analysis of secondary dioxin exposure pathways.

[n] In the screening risk assessment of dioxins upper bound estimate for background intake by Australian males was used. This is conservative (OCS 2004).

PAHs

Reviews of the scientific literature indicate little uptake and translocation of PAHs by plants from soil. Organisms that metabolise PAH, like fish and higher invertebrates and human food source animals, accumulate little or no PAHs (ATSDR 1995, WHO 1998a). It is concluded (Section A3.2) there is little or no bioaccumulation of PAHs by plants or animals likely to be consumed by humans and therefore evaluation of secondary exposure pathways for the PAHs is not warranted.

Metals

For metals, the screening procedure is pragmatically grounded in a comparison of predicted receptor ground level concentrations with rural background concentrations that are not associated [o] with significant exposures via secondary pathways. This is augmented firstly with a requirement for a significant margin of exposure [p] via inhalation exposure for individual metals such that if exposure was to occur via secondary pathways then there is ample conservatism in the screen to ensure the additional non-inhalation intakes will not result in adverse health effects, this is essentially saying the metal has to be toxic/hazardous to a certain degree before there is concern. Secondly a brief review of the literature which indicated lack of potential for most metals to bio-magnify through the food chain[q]. It is concluded in section B1.5 that since the screening criteria for metals were not satisfied, detailed examination of the secondary exposure pathways is not required.

B.1.2 Polycyclic aromatic hydrocarbons

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and meat. Active or passive inhalation of in tobacco smoke is a major source of exposure for many people. Cooking meat or other food at high temperatures increases the amount of PAHs in the food. There are more than 100 different PAHs which generally occur as part of complex mixtures, thus environmental exposures are not to single PAH compounds. PAHs occur naturally and enter the environment mostly as releases to air from volcanoes, burning off and bush fires, residential wood burning, and exhaust from automobiles and trucks. PAHs breakdown in air by reacting with sunlight and other substances over a period of days to weeks, in soil, water and sediment breakdown generally takes weeks to months and is primarily through the actions of micro-organisms (**ATSDR 1995**).

In a total of 65 studies the most notable health effect in chronic studies with animals is cancer, this has occurred via every exposure route (n=5) and every species tested (n = 7) (**WHO 1998a**). It is therefore a

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[o] This assumption is based on the background concentrations used for comparison being from rural or remote i.e. areas regarded as being unpolluted, where deposition to soil/pasture is not expected or shown to be a problem leading to significant exposure by secondary pathways.

[p] The margin of exposure (MoE) is traditionally established from comparison of estimated exposure concentrations with the No Observed Adverse Effect Level (NOAEL) for the compound. However in this risk assessment the MoE is established by comparison with the guideline value (i.e. the hazard quotient). With this latter comparison the safety factors embedded in the guideline are augmented to provide greater safety.

[q] The exception is cadmium; although the relative biomagnification of cadmium may not be significant sufficient concentrations in certain portions of animals used to prepare food for humans can be high and be a potential health risk.

reasonable assumption that sufficient exposure of humans to PAHs by any exposure route is associated with a risk of cancer induction.

Benzo(a)pyrene is genotoxic in a large number and wide variety of in vitro and in vivo tests that have the capacity for metabolic activation of the substance (**ATSDR 1995, WHO 1998a**).

In occupational epidemiological studies there is a clear dose related association between inhalation exposures to PAH mixtures which include benzo(a)pyrene, and increased risk of lung cancer. Benzo(a)pyrene is frequently used as a marker for PAH mixtures. It is a genotoxic carcinogen in all species tested. Generally the site of tumour formation depends on the route of exposure but is certainly not restricted to application sites. It appears that the extent of absorption from different exposure modes plays an important role in initiation and tissue distribution of PAH – induced tumours. Obviously 'site of contact' tissues (stomach, lung and skin) experience the highest concentrations after ingestion/gavage, inhalation or dermal exposure respectively and logically would be expected to be amongst the most sensitive organs to the tumorigenicity of benzo(a)pyrene. This is in fact what is observed. Thus the health effect of concern for air borne PAH pollutants is lung cancer.

The concentrations of individual PAHs in ambient air around the world varies over several orders of magnitude but are generally in the range of <0.1 – 100 ng/m^3. The average levels of individual PAHs in ambient air of rural areas are generally 0.1 – 1 ng/m^3, and in urban areas 1 – 30 ng/m^3 with some locations being greater than 200 ng/m^3 for specific PAHs (**WHO 1998a**). In a previous HRA undertaken by Toxikos, the highest predicted annual ground level concentrations of PAH from the mill was 0.5 ng/m^3. This concentration is at about the rural background range of 0.1 – 1 ng/m^3 reported by WHO (**1998a**).

The concentration of PAH in vegetation is generally considerably lower than that in soil; bioaccumulation factors ranging from 0.0001-0.33 for benzo (a)pyrene and from 0.001-0.18 for 17 other PAHs have been reported (**WHO1998a**). In UK cropland soils, given repeated applications of PAHs in sewerage sludge over a number of years, the concentrations of PAHs in plants did not correlate with soil concentrations, and PAH on above ground plant parts were concluded as probably being the result of atmospheric deposition. In a separate study there was minimal movement of PAHs from the root peel of carrots to the inner core, suggesting simple adsorption onto the roots was the major process whereby PAHs may be found on plants (**ATSDR 1995**). Thus there is little uptake and translocation of PAHs by plants from soil.

In the aquatic environment species that metabolise PAH to little or no extent, like algae, molluscs, and the primitive invertebrates can accumulate high concentrations of PAH, as would be expected from their log KOW values, but organisms that metabolise PAH, like fish and higher invertebrates accumulate little or no PAHs. Species that can bio-transform PAHs have internal concentrations well below the concentration in the sediment. The average bioaccumulation factors (normalised with respect to lipid content and organic carbon content) for eel, pike, and roach were 0.1 and 0.015 (**WHO 1998a**).

It can be inferred from the available information on the total human body burden that PAHs do not persist in the body and that turnover is rapid. This inference excludes those PAH moieties that become covalently bound to tissue constituents, in particular nucleic acids, and are not removed by repair (**WHO 1998a**).

B.1.2.1 Conclusions for PAHs

From the above information it is concluded that there is little or no bioaccumulation of PAHs by plants or animals likely to be consumed by humans. Similarly PAHs are not likely to biomagnify up the human food chain because they are readily metabolised in higher animals. It is therefore considered that evaluation of secondary exposure pathways for PAHs is not warranted.

B.1.3 Screening assessment – Metals

The majority of metals in ambient air are in association with small particulates, generally less than 5µm. Gravitational settling (i.e. dry deposition) governs the removal of large particles (>~5 µm) from air, whereas smaller particles are removed primarily by wet deposition. The partitioning between dry and wet deposition depends on the frequency, intensity and duration of precipitation, the metal in question, its form in the particulate matter, and particle size. The importance of wet deposition relative to dry deposition generally increases with decreasing particle size. Removal of coarse particles may occur in a matter of hours. Small particles with a size range of <2.5 µm may have an atmospheric half-life as long as 30 days and therefore have the potential to be transported over long distances.

Once deposited on soil or plant surfaces it is not always possible to separate the environmental fate processes relating to transport and partitioning of metals between media from those relating to transformation of metals between redox states and/or various compounds/complexes. Part of the problem is that chemical analyses of metals in air, soil or biological matrixes rarely identify the form of the metal. A change of mobility may result from the transformation of a metal to a more or less soluble form which may have a marked effect on its uptake by plants and bioavailability to higher organisms. Specific local information such as soil pH, redox potential, organic and metallo-complex adsorption content is critical in determining a metal's lability and availability to organisms. Consequently it is very difficult to predict environmental behaviour of the metals on a general geographical basis. In addition for each metal, the resources expended gathering the necessary information for a full multi-pathway environmental fate and health risk assessment are not inconsequential. It is usual therefore to conduct a screening evaluation to justify resource expenditure. The basis for the screening process applied in this risk assessment for deciding the requirement for a multi-pathway evaluation is presented below.

To undertake a screening for secondary pathways for metals, Toxikos has undertaken a two-fold assessment:

1. Comparison of soil metal concentrations with health based guidelines
2. Comparison of background ambient air concentrations for metals with the predicted increment in annual ground level concentrations due to emissions

B.1.3.1 Incremental receptor soil metal concentrations

Toxikos was provided with soil concentrations for 7 metals (As, Pb, Be, Cd, Cu, Cr, Hg, Mn Ni) from 7 monitoring sites around the Mill, sampled in April 2012. The highest concentrations measured were taken as background existing levels (Table 11-11). Also provided were total annual deposition data of these 7 metals at each of the receptors depicted in **Table 4-2.**

Table 11-11: Background soil metal concentration

Parameter	Soil Monitoring Sites (SMS) - 3 April 2012							Highest
	SMS1	SMS2	SMS3	SMS4	SMS5	SMS6	SMS7	
Arsenic (mg/kg)	<5	<5	<5	<5	<5	<5	<5	**5**
Cadmium (mg/kg)	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	**0.2**
Chromium (mg/kg)	**15.5**	4.6	4.2	12.6	6	9.9	10.1	**15.5**
Copper (mg/kg)	4	3.4	3.2	5	3.5	**6.3**	4.9	**6.3**
Lead (mg/kg)	9	5	6	9	10	**19**	8	**19**
Mercury (mg/kg)	**0.19**	0.07	0.09	0.06	<0.05	<0.05	<0.05	**0.19**
Nickel (mg/kg)	2	**14**	2	3	2	3	3	**14**
Zinc (mg/kg)	14.9	14.4	14.4	13.7	12.6	**28.6**	17.3	**28.6**

Background concentration levels were then added to estimated concentrations of metals in soils resulting from deposition of mill emissions and compared to health investigation guideline levels (HILs). To calculate the accumulation of metal in soil the annual deposition rate was simply multiplied by 70 years of exposure based on a human lifetime. This method also assumes no loss of metals from the receiving soil.

The concentration of metals in soil was calculated using Equation 10 (adapted from Equation 5-1 in US EPA 2005). The deposition rate (e.g $Pb_{ann\ dep}$) for each metal was sourced from spread sheets provided by Visy (values for other parameters in Equation 1 are those recommended for untilled (non-agricultural) soil as outlined by US EPA (**2005, pg. 5-21**). The equation assumes the environmental half-life of the metals at each receptor is infinite, and there is no physical loss from the soil due to wind erosion, percolation by rain or water runoff. The US EPA (**2005**) recommend a soil mixing depth of 2 cm for untilled soils (**US EPA 2005**). This mixing depth has been adopted in estimating soil metal concentrations at receptor locations after 70 years operation of the mill.

$$Cs = [Metal_{ann\ dep}\ (g\ Metal/m^2/yr) \times T\ (yr) \times 100] \div [\partial\ (g/cm^3) \times D\ (cm)] \quad \text{.....Equation 10}$$

Where:
Cs = Concentration of metal in soil after a given number of years of mine operation (e.g. mg Pb/kg soil).
$metal_{ann\ dep}$ = Annual deposition of the metal onto residential soil from mill (g metal/m^2/yr).
This value is receptor specific.
T = Lifetime (70years).
100 = Units conversion factor (106 µg/g)/104 cm^2/m^2).
∂ = Soil bulk density, assumed to be 1.5 g/cm3. This value is the recommended default by the US EPA (2005). US EPA guidance states that literature values for soil bulk density range from 0.93-1.84 g/cm3, depending on soil type. It is not stated what soil type coincides with the value of 1.5 g/cm3, and the original references were not available to Toxikos to verify the value selected. Therefore, the default value used by the US EPA (2005) has been adopted on face value in this risk assessment.
D = Soil mixing depth, assumed to be 2 cm (**US EPA 2005**).

The calculated soil concentrations are presented in **Table 11-12** and

Table 11-13 representing 70 years of mill operation.

Table 11-12: Calculated soil metal concentrations (Receptors 1 -9a)

	Receptor									
	1	2	3	4	5	6	7	8	9	9a
As	3.90E-03	5.05E-03	6.24E-03	3.44E-03	**7.47E-03**	2.91E-03	5.04E-03	3.08E-03	6.13E-03	4.35E-03
Cd	3.86E-12	6.61E-12	4.53E-12	6.46E-12	**2.12E-11**	5.40E-12	7.09E-12	4.71E-12	3.33E-12	3.77E-12
CrIV	5.43E-05	9.46E-05	6.53E-05	9.07E-05	**3.03E-04**	7.49E-05	1.02E-04	6.73E-05	4.76E-05	5.38E-05
Cu	2.21E-03	4.24E-03	3.03E-03	3.65E-03	**1.34E-02**	2.80E-03	4.56E-03	2.98E-03	2.12E-03	2.38E-03
Pb	2.65E-03	4.23E-03	2.82E-03	4.47E-03	**1.37E-02**	3.91E-03	4.54E-03	3.05E-03	2.14E-03	2.43E-03
Hg	9.64E-11	1.81E-10	1.28E-10	1.60E-10	**5.74E-10**	1.25E-10	1.94E-10	1.27E-10	9.06E-11	1.02E-10
Ni	1.35E-02	2.27E-02	1.55E-02	2.26E-02	**7.30E-02**	1.91E-02	2.44E-02	1.62E-02	1.14E-02	1.30E-02

Table 11-13: Calculated soil metal concentrations (Receptors 10 - 27)

	Receptor									
	10	11	12	13	14	19	21	22	26	27
As	5.06E-03	4.39E-03	3.07E-03	4.43E-03	2.55E-03	4.86E-03	2.73E-03	2.63E-03	1.03E-03	7.62E-04
Cd	3.53E-12	3.83E-12	2.87E-12	3.14E-12	1.61E-12	3.09E-12	3.80E-12	5.76E-12	1.93E-12	1.17E-12
CrIV	5.08E-05	5.51E-05	4.12E-05	4.50E-05	2.31E-05	4.44E-05	5.38E-05	8.23E-05	2.74E-05	1.68E-05
Cu	2.34E-03	2.52E-03	1.89E-03	2.02E-03	1.04E-03	2.01E-03	2.27E-03	3.65E-03	1.16E-03	7.51E-04
Pb	2.21E-03	2.41E-03	1.80E-03	2.01E-03	1.03E-03	1.96E-03	2.55E-03	3.72E-03	1.29E-03	7.53E-04
Hg	9.89E-11	1.07E-10	8.01E-11	8.60E-11	4.42E-11	8.54E-11	9.82E-11	1.56E-10	5.01E-11	3.20E-11
Ni	1.20E-02	1.31E-02	9.80E-03	1.08E-02	5.53E-03	1.06E-02	1.32E-02	1.98E-02	6.70E-03	4.03E-03

HILs for contaminated sites are an example of guidance values for a particular medium, soil. A 'residential' land use setting is employed for deriving the guidance value and values are based on a default exposure scenario for a 2 year old child because the typical behaviour pattern of this age group gives them the greatest exposures to contaminants in soil. There are relatively higher soil ingestion rates, and dermal and inhalational exposures relative to body weight for a 2 year old compared to older age groups.

Table 11-14 provides a comparison of incremental soil concentrations due to long-term atmospheric deposition with available soil quality guidelines.

Table 11-14: Comparison of incremental soil concentrations due to long-term atmospheric deposition with available soil quality guidelines

Metal	Background (mg/kg)	Calculated Increase (mg/kg)	Cumulative (background + increase) (mg/kg)	HIL[a] (mg/kg)	Hazard Quotient
As	5	7.47E-03	5.01E+00	100	0.05
Cd	0.2	2.12E-11	2.00E-01	20	0.01
CrIV	15.5	3.03E-04	1.55E+01	100	0.16
Cu	6.3	1.34E-02	6.31E+00	1000	0.01
Pb	19	1.37E-02	1.90E+01	300	0.06
Hg	0.19	5.74E-10	1.90E-01	10	0.02
Ni	14	7.30E-02	1.41E+01	600	0.02

[a] enHealth 2001. Health-based Soil Investigation Levels. Commonwealth of Australia.

The cumulative soil metal concentrations resulting from mill emission deposition are orders of magnitude below HILs and therefore further investigation is not warranted.

B.1.3.2 Comparison of background ambient air concentrations for metals - assumptions and screening criteria

a The metal should exhibit significant bioaccumulation by plants, especially common vegetables and grasses, and animals. If biomagnification occurs then concern for human exposure via food increases as does the need for secondary exposure pathway evaluation. Obviously if the metal in question does not accumulate in human food sources then concern regarding potential health effects via secondary exposure pathways is not warranted.

Screening criteria A: The available weight of literature evidence must indicate the metal is able to bioaccumulate, or biomagnify, into human food sources.

b It is assumed the increase in exposure to metals via secondary pathways is proportional, in some manner, to any change in long term concentration of metals in air due to emissions from the pulp mill. For components of industrial emissions to be of concern relative to human exposure through secondary exposure pathways there is a requirement for them to be deposited onto soil or pasture in sufficient quantity that they will increase environmental concentrations at a faster rate than the normal processes of removal. It follows therefore that even for those metals capable of bioaccumulation, that if the predicted chronic air concentrations of emission components are less than those usually found in rural or remote environments then the emission components will not accumulate to an extent where food chain contamination is likely. Hence for there to be an increase in secondary pathway exposures there must be a significant increase, relative to background, in chronic air concentration of the metals at the receptor locations.

Screening criteria B: To trigger detailed evaluation of secondary exposure pathways the predicted incremental increase in annual ground level concentration of the metal must be about the same, or above those measured in rural and remote areas.

c The intake of metals into the body via inhalation is a relatively small proportion of total intake; most metals enter the body via the diet. For bioaccumulative metals a small increase in intake via inhalation could result in a larger overall intake since inhalation is only a small part of total intake. The requirement therefore is to ensure the incremental increase in inhalation intake is sufficiently small to keep total intakes low. This will ensure an adequate safety margin is maintained in the screening process such that total intake in the future is kept below any intake level that may be deleterious to health.

The proposed criteria to trigger evaluation of indirect exposure pathways for metals is for the direct inhalation hazard quotient, for any given metal, to be greater than 0.05. This is saying the incremental increase in exposure to any given metal from the mill should be greater than 5% of its respective health guideline value before secondary exposure pathways need be evaluated.

Within this criteria is an assumption that 5% of the total intake is via inhalation and that the remaining 95%, which is ingested either in diet or by consuming soil, has the same toxicological potency and health effects as inhalation exposure, this is often not the case. Ingested metals are often less toxic because homeostatic mechanisms limit their absorption and the spectrum of effects are different. Therefore the tacit assumption embedded in using a low inhalation hazard quotient as triggering criteria errs on the side of safety.

The rationale underpinning the inhalation exposure 'cut-off' is similar to that used in the dioxin risk assessment. Inhalation exposure of metals generally constitutes only approximately <1 – 20% of background intake (e.g. Langley 1991a,b; Maynard 1991). Arsenic and cadmium are two metals for which concern is often expressed regarding accumulation in the environment. According to the

European Commission (EC 2000) the percentage for arsenic and cadmium uptake via ambient air is less than 1 and 3 % respectively.

Screening criteria C: To trigger detailed evaluation of secondary exposure pathways the hazard quotient for direct inhalational exposure of a metal must be more than 0.05.

Summary of screening trigger criteria for need for assessing secondary exposure pathways:

The criteria for triggering a detailed multimedia/multipathway health risk assessment for metals in the pulp mill emissions are:

1. The metal must be capable of being bioaccumulated by plants and animals relevant for the human food chain, and

2. The predicted annual ground level concentration increment must be above those measured in rural and remote areas, or

3. The direct inhalation hazard quotient for incremental annual average concentration any given metal is greater than 0.05.

B.1.4 Discussion of the trigger screening criteria

Similar criteria have been previously applied by risk assessors of air emissions. Greim (**1990**) evaluated the health risks posed by 69 modern municipal waste incineration plants (49 in operation and 20 in the planning and construction phase) in Germany. The assessment was conducted by comparing conservatively estimated ground level concentrations to background air concentrations in rural areas, threshold levels and air guideline values. The predicted ground level air concentrations of metals were similar or lower than those found in rural areas and because of this it was concluded that overall exposure to metals emitted by municipal waste incinerators did not present a risk to human health.

Boudet et al (**1999**) evaluated a modern municipal waste incinerator in France. The authors only evaluated direct inhalation health effects. While acknowledging secondary pathways are potentially relevant to such assessments, they cited previous studies which had shown the burden of metals in the food chain in the vicinity of modern facilities was not increased. It can therefore be rationalised that inclusion of those pathways would not influence the results from assessment of direct inhalation.

The general approach and conservativeness of the criteria applied herein for assessing the need for evaluating secondary exposure pathways for metals is supported by the results of a multimedia, multipathway risk assessment for waste combustors, conducted for the US EPA (HWC) in support of establishing technical emission standards for these facilities (**RTI 1999a**). This very comprehensive health risk assessment addressed direct and indirect (i.e. secondary food chain) exposures for 79 HWCs, it focussed on the population within a radius of 20 km and evaluated 14 metals for each facility. The assessment included likely maximum exposed individuals who, due to their activities, could be at increased risk (e.g. recreational fishermen, and subsistence farmers and home gardeners).

Lead, arsenic and mercury were considered to be the metals of concern and the following findings were made (**RTI 1999b,c**):

- For lead the highest exposed individual was a child (0 to 5 years old) of a home gardener; nevertheless the incremental increase in blood lead levels due to the point combustion source were predicted to not significantly increase blood concentrations over background exposures.
- Children of dairy farmers were found to have the greatest exposure to arsenic due to their relatively high consumption of milk. High-end lifetime excess cancer risk (for the child of the dairy farmer) was nonetheless below one in a million.
- Recreational fishermen were identified as the individuals with the highest exposure to mercury due to high consumption fish and the type caught containing high concentrations of methylmercury. Children also had high exposures due to high fish consumption relative to body weight compared to adults. However all hazard quotients for all scenarios and exposure percentiles were less than 1.
- For the remaining metals assessed (i.e. antimony, chromium VI, chromium III, barium, nickel, beryllium, selenium, cadmium, silver, and thallium) hazard quotients for indirect pathways were all less than one and generally less than 0.01.

The results from the US EPA waste combustor assessment showed that for the metals evaluated there was no health risks associated with the indirect, secondary exposure pathways of metals.

B.1.4.1 Predicted air concentrations relative to background

Because background air concentrations of metals in the Tumut region are not known at this time, background concentrations for other parts of the world have been sought from authority reviews. These are presented in **Table 11-15** together with the predicted ground level concentration at Receptor 5, as it is the receptor locations with the highest predicted annual ground level concentration for the metals. For all the metals the predicted ground level concentrations are within background concentrations at rural or remote locations elsewhere in the world. Metal exposure via either direct inhalation or secondary pathways has not been flagged as an issue at these background locations.

Table 11-15: Comparison of background ambient air concentrations for metals with the predicted increment in annual ground level concentrations due to emissions

Metal	Predicted annual ground level concentration (ng/m^3)	Ambient background (ng/m^3)	Comment on location	Reference
Arsenic	0.005	1 – 3 1 – 28 4.2 – 9.6 20 – 100 0.09-2.5	US remote locations. US rural areas. Long term mean, Great Lakes. US urban areas. NSW Urban	ATSDR (2000a) NSW (2003)
Cadmium	0.0003	< 5 1-5 5-50 0.03-1	US general ambient. Rural Locations. Urban/ Industrialised Areas NSW Urban	ATSDR (1999b) WHO (1992) NSW (2003)
Chromium	0.038	5-525 <100 <20 4-25	All US Non industrialized areas Australia Urban Launceston, Tasmania	ATSDR (2000) WHO (1988a) EA (2002) EA (2002)
Copper	0.073	5-50 2.4-28 <650 2-6	Rural NSW Urban WA Urban Launceston, Tasmania	WHO (1998d) NSW (2003) EA (2002) EA (2002)
Lead	0.096	0.1-8 200-400 100 1000-3000 5000-1000 2.4-99 140-1570 20 44-173	Remote areas. US urban areas. Australia Rural. Australia Urban Australia- Near heavy traffic NSW Urban WA Urban WA CBD Launceston, Tasmania	WHO (1977) ATSDR (1999c) Maynard (1991) NSW (2003) EA (2002) NEPC (2002b) EA (2002)
Mercury	0.008	10 – 20 1 2-4 10	Industrialised areas. Remote Southern Hemisphere Rural areas. Urban areas	ATSDR (1999d) WHO (1989) WHO (2000)
Nickel	0.48	2.22 0.6-78 1-328 <0.1-1 0.86-20 <10 14-56	General ambient 1996. US rural US urban Marine NSW urban WA Urban Launceston, Tasmania	ATSDR (2003c) WHO (1991) NSW (2003) EA (2002) EA (2002)
Vanadium	0.013	0.001-0.8 0.21-64 50-200 0.16-49 3-4	Remote Rural Urban NSW Urban Launceston, Tasmania	WHO (1988b) WHO (2000) NSW (2003) EA (2002)

B.1.4.2 Concentration in air relative to health guideline

A thorough evaluation of the contribution of inhalation exposure to total intake of metals has not been undertaken. However Langley (**1999a, b**) and Maynard (**1991**) indicate for arsenic, cadmium and lead, inhalation represents approximately 0.2%, 10% and 20% respectively of total intake. According to a European Commission (**EC 2000**) the percentage for arsenic and cadmium uptake via ambient air is less than 1 and 3 % respectively. An inherent assumption in the screening criteria is that oral and inhalation exposure have the same propensity for causing an adverse health effect. However, for many of the metals there is quite different potency for adverse health effects for oral versus inhalation exposure, the latter being of greater concern. Hence since health based guidelines for inhalation exposure is used as the basis for the screening criteria in this section, therefore they are intuitively conservative. Given the above information, for metals in emissions from the mill, it was considered an incremental increase in inhalation exposure of 5% relative to the inhalation health guidelines, i.e. a hazard quotient of 0.05, would leave sufficient conservatism for screening purposes to ensure health effects arising from long term exposure from all exposure pathways was unlikely to result in adverse health effects.

The absolute amount of metals added to the air shed is very small and the highest metal hazard quotient (**Table 11-16**) at any receptor is only 0.0019. Hence it is unlikely a demonstrable increase in either air concentration or deposited metal within the air shed will occur as a result of the mill emissions.

Table 11-16: Margin of Exposure between predicted annual ground level concentrations and chronic guidelines for metals due to emissions

Metal	Predicted annual ground level concentration (ng/m^3)	Chronic Guideline Value b (ng/m^3)	Hazard Quotient c
Arsenic	0.005	30	1.67E-04
Cadmium	0.0003	5	6.00E-05
Chromium III	0.036	60,000	6.00E-07
Chromium VI	0.002	100	2.00E-05
Copper	0.073	1,000	7.30E-05
Lead	0.096	500	1.92E-04
Mercury	0.008	1,000	8.00E-06
Nickel	0.48	50	9.60E-03
Vanadium	0.013	1,000	1.30E-05

It is therefore considered that a multimedia risk assessment for metals is not required.

B.1.4.3 Bioaccumulation of metals

Of the metals considered herein, only cadmium appears to have potential for accumulation and bioconcentration by green plants (**Fergusson 1990**).

It is to be noted that the air guideline used for cadmium in this risk assessment has been established by WHO (**2000**) to prevent further increases of cadmium in agricultural soils that would be likely to increase the dietary intake of future generations. This was established because renal effects were observed in inhabitants of areas contaminated by past emissions of cadmium suggesting the cadmium body burden of the general population in some parts of Europe cannot be increased without endangering renal function.

B.1.5 Notes on the bioaccumulation of metals.

Arsenic

Arsenic is released into the atmosphere primarily as arsenic trioxide or, less frequently, in one of several volatile organic compounds, mainly arsines. Trivalent arsenic and methyl arsines in the atmosphere undergo oxidation to the pentavalent state, and arsenic in the atmosphere is usually a mixture of the trivalent and pentavalent forms.

The predominant dietary source of arsenic is seafood, followed by rice/rice cereal, mushrooms, and poultry. However, most of the arsenic in seafood is of the non-toxic arsenobetaine form.

Arsenic is largely immobile in agricultural soils; therefore, it tends to concentrate and remain in upper soil layers indefinitely. According to ATSDR (2000), studies indicate uptake of arsenic from soil by vegetables is generally not very high. Even when grown on highly polluted soil or soil naturally high in arsenic, the amount of arsenic taken up by the plants is comparatively low.

Because arsenic concentrations in organisms tended to decrease with increasing tropic level, an extensive study of the factors affecting bioaccumulation of arsenic in two streams in western Maryland in 1997–1998 concluded there was no evidence of biomagnification. Arsenic is mainly accumulated in the exoskeleton of invertebrates and in the livers of fish. No differences were found in the arsenic levels in different species of fish which included herbivorous, insectivorous, and carnivorous species. The major bioaccumulation transfer is between water and algae, at the base of the food chain and this has a strong impact on the concentration in fish.

Arsenic is not biomagnified through the food chain (**ATSDR 2000a**).

Cadmium

Atmospheric cadmium is in the form of particulate matter. Cadmium emitted to the atmosphere from combustion processes is usually associated with very small particulates that are in the respirable range (<10 µm) and undergo long-range transport. These cadmium pollutants may be transported from a hundred to a few thousand kilometers and have a typical atmospheric residence time of about 1 -10 days before deposition occurs.

The principal chemical species in air is cadmium oxide, although some cadmium salts, such as cadmium chloride, can enter the air, especially during incineration. These are stable compounds that do not undergo significant chemical transformation. The chief fate of airborne cadmium is to be dispersed by the wind and, subsequently, deposited by wet or dry processes.

Wet and dry deposition of cadmium from the atmosphere may also contribute sizable amounts of cadmium to soil in the areas surrounding sources of atmospheric emissions, such as coal power stations, incinerators and vehicular traffic, which may release cadmium from burned fuel and tire wear.

Contamination of soil by cadmium is of concern because the cadmium is taken up efficiently by some plants and, therefore, enters the food chain for humans and other animals. A low soil pH increases the uptake of cadmium by plants. Cadmium is taken up and retained by aquatic and terrestrial plants and is concentrated in the liver and kidney of animals that eat the plants. Grain and cereal products usually contribute the greatest percentage of dietary cadmium; potatoes, leafy vegetables, and root vegetables also contain relatively high levels. Organ meats (liver and kidney) and shellfish can also contribute to cadmium intake for individuals who consume large amounts of these items.

The data indicates that cadmium bioaccumulates in all levels of the food chain. Cadmium accumulation has been reported in grasses and food crops, and in earthworms, poultry, cattle, horses, and wildlife. The metal burden of a crop depends on uptake by the root system, direct foliar uptake and translocation within the plant, and surface deposition of particulate matter. In general, cadmium accumulates in the leaves of plants and, therefore, is more of a risk in leafy vegetables grown in contaminated soil than in seed or root crops.

Since cadmium accumulates largely in the liver and kidneys of vertebrates and not in the muscle tissue and intestinal absorption of cadmium is low, biomagnification through the food chain may not be significant. Nevertheless, uptake of cadmium from soil by feed crops may result in high levels of cadmium in beef and poultry (especially in the liver and kidneys). This accumulation of cadmium in the food chain has important implications for human exposure to cadmium, whether or not significant biomagnification occurs because these animal parts are often used to manufacture salami and other processed meat products (ATSDR1999b).

Lead

Plants and animals may bioconcentrate lead but biomagnification has not been detected. In general, the highest lead concentrations are found in aquatic and terrestrial organisms that live near lead mining, smelting, and refining facilities; storage battery recycling plants; areas affected by high automobile and truck traffic; sewage sludge and soil disposal areas; sites where dredging has occurred; areas of heavy hunting (lead source from spent shot); and in urban and industrialized areas. Lead may be present on plant surfaces as a result of atmospheric deposition; its presence in internal plant tissues indicates biological uptake from the soil and leaf surfaces. Although the bioavailability of lead in soil to plants is limited because of the strong absorption of lead to soil organic matter, the bioavailability increases as the pH and the organic matter content of the soil are reduced. Lead is not biomagnified in aquatic or terrestrial food chains. It may contaminate terrestrial plants as a result of atmospheric deposition and uptake from soil, and animals as a result of inhalation of contaminated ambient air or ingestion of contaminated plants. Older organisms tend to contain the greatest body burdens of lead. In aquatic organisms, lead concentrations are usually highest in benthic organisms and algae, and lowest in upper trophic level predators (e.g., carnivorous fish) (ATSDR 1999c).

Mercury

Anthropogenic emissions, mainly from combustion of fossil fuels (coal), account for about 25% of mercury emissions to the atmosphere. These mercury emissions eventually may be deposited on the surrounding soil. From power generators and non-utility power and heat generation the percentage mercury emissions were Hg^0 , Hg^+, particulates = 50%, 30 % & 20% respectively. The overall residence time of elemental mercury in the atmosphere has been estimated to be 6 days to 2 years.

Over 95% of the mercury found in the atmosphere is gaseous mercury (Hg^0), the form involved in long-range (global) transport of the element. Residence time in the atmosphere has been estimated to range from 6 days to 2 years. Approximately 5% of atmospheric mercury is associated with particulates, which have a shorter atmospheric residence time, are removed by dry or wet deposition, and may show a regional or local distribution pattern.

Dry deposition may account for approximately 70% of the total atmospheric deposition of mercury during the summer, although on an annual basis, wet and dry deposition may be of equal importance. Wet deposition is the primary method of removal of mercury from the atmosphere (approximately 66%) and may account for virtually all of the mercury content in remote lakes that do not receive inputs from other sources (e.g., industrial effluents). Most inert mercury (Hg^{+2}) in precipitation is bound to aerosol particulates, which are relatively immobile when deposited on soil or water.

Fish appear to accumulate methyl mercury from both food sources and the water column with food being the predominant source. The biological concentration factor (BCF) of methyl mercury in fish can be as high as three million. Bioconcentration of the mercuric forms is less.

The potential for bioaccumulation in terrestrial food chains is demonstrated by the uptake of mercury by edible mushroom, grown on compost containing mercury at concentrations of up to 0.2 mg/kg (ppm). The bioaccumulation factors reported ranged from 65 to 140, indicating that there are potential risks to human health if these mushrooms are eaten in large quantities.

Data from higher plants indicate that virtually no mercury is taken up from the soil into the shoots of plants such as peas, although mercury concentrations in the roots may be significantly elevated and reflect the mercury concentrations of the surrounding soil (ATSDR 1999d).

Nickel

Nickel is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. It is dispersed by wind and removed by gravitational settling (sedimentation), dry deposition (inertial impaction characterized by a deposition velocity), washout by rain (attachment to droplets within clouds), and rainout (scrubbing action below clouds). The removal rate and distance travelled from the source depends on source characteristics (e.g., stack height), particle size and density, and meteorological conditions.

Particulates from power plants tend to be smaller than those from smelters. Submicron particles may have atmospheric half-lives as long as 30 days. Monitoring data confirm that nickel can be transported far from its source. In one coal plant, 53 and 32% of nickel in emissions were associated with particles <3 and <1.5 µm in diameter, respectively. Other studies found that only 17–22% of nickel emissions from coal-fired power plants were associated with particles of >2 µm, and that the mass medium diameter (MMD) of nickel-containing particles from a plant with pollution control devices was 5.4 µm. Surface-adsorbed nickel would be more available than embedded nickel. Wood combustion is potentially an important source of nickel emissions.

The form of nickel emitted to the atmosphere varies according to the type of source. Nickel species associated with combustion, incineration, and metals smelting and refining are often complex nickel oxides, nickel sulphate, and metallic nickel, and in more specialized industries, the species commonly found are nickel silicate, nickel sub-sulphide, and nickel chloride.

The speciation and physicochemical state of nickel is important in considering its behaviour in the environment and availability to biota. Nickel is strongly adsorbed at mineral surfaces such as oxides and hydrous oxides of iron, manganese, and aluminium. Such adsorption plays an important role in controlling the concentration of nickel in natural waters.

Bioconcentration factors for nickel in various aquatic organisms (e.g., algae, arthropods, molluscs, and fish) are about of 100. There is no evidence that nickel biomagnifies in aquatic food webs and, in fact, there is evidence to indicate that the nickel concentrations in organisms decrease with increasing trophic level.

Uptake and accumulation of nickel into various plant species is known to occur. For example alfalfa grown from soils contaminated with a Ni(II) at a loading of 50 mg/kg. Concentration ratios of nickel in plant versus soil ranged between 22 and 26 over a pH range of 4.5–7.1.

Two studies concerning levels in voles and rabbits living on sludge-amended land did not indicate any accumulation of nickel in these herbivores or in the plants they fed upon. The lack of significant bioaccumulation of nickel in aquatic organisms, voles, and rabbits indicates that nickel is not biomagnified in the food chain (ATSDR 2003c).

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APPENDIX C ACUTE HAZARD INDICES AND QUOTIENTS

Table 11-17: Acute hazard quotients and indices at Receptor 1

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	128.3200	47.7490	16.7790	5.9545	10000	0.0128	0.005	0.002	0.001
NO_x (1 hr)	49.3620	17.3462	5.9364	3.0570	226	0.22	0.08	0.0	0.0
SO_2 (1 hr)	14.1910	1.8275	0.1790	0.0974	572	0.02	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.4980	0.9064	0.3634	0.1511	50	0.0300	0.018	0.007	0.00
					Criteria pollutants hazard index	***0.29***	***0.10***	***0.04***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.0000	-				
Mercury (1 hr)	0.0004	0.0002	0.0001	0.0001	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0002	0.0001	0.0000	0.0000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.0003	0.0002	0.0001	0.0001	-				
Cobalt (1 hr)	0.0002	0.0001	0.0000	0.0000	-				
Chromium (1 hr)	0.0017	0.0011	0.0006	0.0004	-				
Copper (1 hr)	0.0037	0.0022	0.0012	0.0009	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.0058	0.0032	0.0016	0.0011	-				
Nickel (1 hr)	0.0234	0.0139	0.0075	0.0051	6	0.0039	0.0023	0.0012	0.0008
Lead (1 hr)	0.0054	0.0030	0.0015	0.0009	-				
Antimony (1 hr)	0.0002	0.0001	0.0001	0.0000	-				
Selenium (1 hr)	0.0004	0.0002	0.0001	0.0001	-				
Tin (1 hr)	0.0035	0.0020	0.0010	0.0007	-				
Vanadium (24 hr)	0.0001	0.0000	0.0000	0.0000	1	0.000065	0.00004	0.00002	0.0000
					Metals hazard index	***0.005***	***0.003***	***0.001***	***0.00***
Chlorine (1 hr)	0.7360	0.4310	0.2435	0.1775	210	0.004	0.002	0.00	0.00
Sulfuric acid (1 hr)	12.5040	6.9272	3.5351	2.4837	120	0.104	0.058	0.03	0.02
Hydrogen chloride (1 hr)	27.8710	18.2440	5.0728	2.2336	2100	0.0133	0.009	0.002	0.001
Hydrogen fluoride (1 hr)	0.0921	0.0513	0.0257	0.0174	240	0.0004	0.0002	0.0001	0.0001
					Acid Hazard index	***0.121***	***0.07***	***0.03***	***0.02***
TRS (as H_2S) (1 hr)	3.9614	1.7537	0.5684	0.2930	-				
TRS (as H_2S) (24 hr)	0.3633	0.1191	0.0228	0.0034	20	0.01817	0.0060	0.0011	0.000
TRS (as H_2S) (30 min)	4.5505	2.0145	0.6529	0.3366	100	0.0455	0.020	0.007	0.003
					Total HI	**0.46**	**0.2**	**0.1**	**0.0**

Table 11-18: Acute hazard quotients and indices at Receptor 2

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	353.2300	102.2200	19.2460	9.2266	10000	0.0353	0.010	0.002	0.001
NOx (1 hr)	16.9206	9.3318	3.4284	2.3480	246	0.07	0.04	0.0	0.0
SO2 (1 hr)	4.8107	1.4451	0.1199	0.0560	572	0.01	0.00	0.00	0.0
TSP (as PM10) (24 hr)	3.9202	1.1074	0.4608	0.1577	50	0.0784	0.022	0.009	0.00
				Criteria pollutants hazard index		**0.19**	**0.07**	**0.03**	**0.01**
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.0000	-				
Mercury (1 hr)	0.0005	0.0003	0.0002	0.0001	1.8	0.0003	0.0002	0.000	0.000
Arsenic (4 hr)	0.0003	0.0001	0.0000	0.0000	0.19	0.002	0.000	0.00	0.00
Beryllium (1 hr)	0.0003	0.0002	0.0001	0.0001	-				
Cobalt (1 hr)	0.0004	0.0001	0.0001	0.0000	-				
Chromium (1 hr)	0.0036	0.0014	0.0008	0.0005	-				
Copper (1 hr)	0.0041	0.0027	0.0017	0.0010	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.0135	0.0052	0.0023	0.0015	-				
Nickel (1 hr)	0.0532	0.0206	0.0098	0.0068	6	0.0089	0.0034	0.0016	0.0011
Lead (1 hr)	0.0129	0.0049	0.0021	0.0013	-				
Antimony (1 hr)	0.0005	0.0002	0.0001	0.0000	-				
Selenium (1 hr)	0.0005	0.0003	0.0002	0.0001	-				
Tin (1 hr)	0.0080	0.0031	0.0014	0.0009	-				
Vanadium (24 hr)	0.0001	0.0000	0.0000	0.0000	1	0.000097	0.00005	0.00002	0.0000
					Metals hazard index	0.011	0.004	0.00	0.00
Chlorine (1 hr)	0.8100	0.5381	0.3346	0.2053	210	0.004	0.003	0.00	0.00
Sulfuric acid (1 hr)	29.2490	11.2250	4.9825	3.2561	120	0.244	0.094	0.04	0.03
Hydrogen chloride (1 hr)	24.0030	12.4970	3.2178	1.3728	2100	0.0114	0.006	0.002	0.001
Hydrogen fluoride (1 hr)	0.2180	0.0833	0.0367	0.0233	240	0.0009	0.0003	0.0002	0.0001
					Acid Hazard index	0.260	0.10	0.04	0.03
TRS (as H_2S) (1 hr)	4.4601	1.2885	0.3943	0.1976	-				
TRS (as H_2S) (24 hr)	0.2245	0.0817	0.0160	0.0029	20	0.01122	0.0041	0.0008	0.000
TRS (as H_2S) (30 min)	5.1233	1.4801	0.4529	0.2270	100	0.0512	0.015	0.005	0.002
					Total HI	0.51	0.2	0.1	0.0

Table 11-19: Acute hazard quotients and indices at Receptor 3

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	93.1400	28.4270	9.2026	4.5810	10000	0.0093	0.003	0.001	0.000
NO_x (1 hr)	12.8038	6.5076	2.9710	1.4895	246	0.05	0.03	0.0	0.0
SO_2 (1 hr)	8.6332	1.0587	0.0767	0.0356	572	0.02	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.8677	0.6250	0.2622	0.0881	50	0.0374	0.013	0.005	0.00
				Criteria pollutants hazard index		**0.11**	**0.044**	**0.02**	**0.01**
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.0000	-				
Mercury (1 hr)	0.0006	0.0003	0.0001	0.0001	1.8	0.0003	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.0000	0.0000	0.0000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.0004	0.0002	0.0001	0.0001	-				
Cobalt (1 hr)	0.0001	0.0001	0.0000	0.0000	-				
Chromium (1 hr)	0.0016	0.0010	0.0005	0.0003	-				
Copper (1 hr)	0.0053	0.0025	0.0012	0.0007	100	0.00005	0.0000	0.0000	0.0000
Manganese (1 hr)	0.0051	0.0028	0.0014	0.0008	-				
Nickel (1 hr)	0.0201	0.0118	0.0063	0.0038	6	0.0033	0.0020	0.0011	0.0006
Lead (1 hr)	0.0049	0.0023	0.0011	0.0007	-				
Antimony (1 hr)	0.0002	0.0001	0.0000	0.0000	-				
Selenium (1 hr)	0.0005	0.0003	0.0001	0.0001	-				
Tin (1 hr)	0.0030	0.0018	0.0009	0.0005	-				
Vanadium (24 hr)	0.0001	0.0000	0.0000	0.0000	1	0.000114	0.00004	0.00002	0.0000
					Metals hazard index	**0.004**	**0.002**	**0.00**	**0.00**
Chlorine (1 hr)	1.0409	0.4882	0.2439	0.1515	210	0.005	0.002	0.00	0.00
Sulfuric acid (1 hr)	11.1220	6.0084	2.9496	1.8001	120	0.093	0.050	0.02	0.02
Hydrogen chloride (1 hr)	15.6450	5.8492	1.6566	0.8224	2100	0.0075	0.003	0.001	0.000
Hydrogen fluoride (1 hr)	0.0832	0.0418	0.0208	0.0125	240	0.0003	0.0002	0.0001	0.0001
					Acid Hazard index	**0.105**	**0.06**	**0.03**	**0.02**
TRS (as H_2S) (1 hr)	6.6130	0.9519	0.2433	0.1456	-				
TRS (as H_2S) (24 hr)	0.6398	0.0471	0.0105	0.0017	20	0.03199	0.0024	0.0005	0.000
TRS (as H_2S) (30 min)	7.5963	1.0935	0.2794	0.1673	100	0.0760	0.011	0.003	0.002
					Total HI	**0.30**	**0.1**	**0.0**	**0.0**

Table 11-20: Acute hazard quotients and indices at Receptor 4

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	126	48	18	10	10000	0.0126	0.005	0.002	0.001
NO_x (1 hr)	23	12	5	3	246	0.09	0.05	0.0	0.0
SO_2 (1 hr)	11	4	0	0	572	0.02	0.01	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.74	0.98	0.4	0.3	50	0.0348	0.020	0.009	0.01
				Criteria pollutants hazard index		***0.16***	***0.08***	***0.03***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0004	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00029	0.00017	0.00011	0.0001	-				
Cobalt (1 hr)	0.0001	0.0001	0.0001	0.0000	-				
Chromium (1 hr)	0.002	0.001	0.001	0.000	-				
Copper (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.005	0.003	0.00	0.00	-				
Nickel (1 hr)	0.0216	0.013	0.008	0.006	6	0.0036	0.0021	0.0014	0.0010
Lead (1 hr)	0.005	0.003	0.00	0.00	-				
Antimony (1 hr)	0.0002	0.0001	0.0001	0.000	-				
Selenium (1 hr)	0.0004	0.0002	0.0001	0.000	-				
Tin (1 hr)	0.003	0.002	0.001	0.00	-				
Vanadium (24 hr)	0.000091	0.00004	0.00002	0.0000	1	0.000091	0.00004	0.00002	0.0000
					Metals hazard index	***0.005***	***0.003***	***0.00***	***0.00***
Chlorine (1 hr)	0.7	0	0	0	210	0.003	0.002	0.00	0.00
Sulfuric acid (1 hr)	11.6	6.9	4	3	120	0.096	0.057	0.04	0.03
Hydrogen chloride (1 hr)	27	16	5	3	2100	0.0129	0.007	0.003	0.001
Hydrogen fluoride (1 hr)	0.09	0.05	0.0	0.0	240	0.0004	0.0002	0.0001	0.0001
					Acid Hazard index	***0.113***	***0.07***	***0.04***	***0.03***
TRS (as H_2S) (1 hr)	15.39	1.96	0.7	0.3	-				
TRS (as H_2S) (24 hr)	1.5416	0.105	0.03	0.01	20	0.07708	0.0052	0.0013	0.000
TRS (as H_2S) (30 min)	17.68	2.3	0.8	0.3	100	0.1768	0.023	0.008	0.003
					Total HI	***0.45***	***0.2***	***0.1***	***0.1***

Table 11-21: Acute hazard quotients and indices at Receptor 5

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	183	102	44	25	10000	0.0183	0.010	0.004	0.003
NO_x (1 hr)	14	5	2	1	246	0.06	0.02	0.0	0.0
SO_2 (1 hr)	111	7	0	0	572	0.19	0.01	0.00	0.0
TSP (as PM_{10}) (24 hr)	2.89	2.06	0.9	0.5	50	0.0577	0.041	0.018	0.01
Criteria pollutants hazard index						***0.33***	***0.08***	***0.03***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0011	0.000	0.000	0.000	1.8	0.0006	0.0003	0.000	0.000
Arsenic (4 hr)	0.0002	0.000	0.000	0.000	0.19	0.001	0.001	0.00	0.00
Beryllium (1 hr)	0.00079	0.00036	0.00021	0.0002	-				
Cobalt (1 hr)	0.0003	0.0002	0.0001	0.0001	-				
Chromium (1 hr)	0.004	0.002	0.001	0.001	-				
Copper (1 hr)	0.010	0.00	0.00	0.00	100	0.00010	0.0000	0.0000	0.0000
Manganese (1 hr)	0.011	0.007	0.00	0.00	-				
Nickel (1 hr)	0.0477	0.027	0.015	0.012	6	0.0079	0.0045	0.0026	0.0020
Lead (1 hr)	0.010	0.006	0.00	0.00	-				
Antimony (1 hr)	0.0004	0.0002	0.0001	0.000	-				
Selenium (1 hr)	0.0010	0.0005	0.0003	0.000	-				
Tin (1 hr)	0.007	0.004	0.002	0.00	-				
Vanadium (24 hr)	0.000131	0.00007	0.00004	0.0000	1	0.000131	0.00007	0.00004	0.0000
Metals hazard index						***0.010***	***0.006***	***0.00***	***0.00***
Chlorine (1 hr)	2.0	1	1	0	210	0.009	0.004	0.00	0.00
Sulfuric acid (1 hr)	14.9	14.6	8	6	120	0.124	0.122	0.07	0.05
Hydrogen chloride (1 hr)	47	28	11	6	2100	0.0224	0.013	0.005	0.003
Hydrogen fluoride (1 hr)	0.18	0.11	0.1	0.0	240	0.0008	0.0004	0.0002	0.0002
Acid Hazard index						**0.156**	**0.14**	**0.07**	**0.05**
TRS (as H_2S) (1 hr)	15.04	6.96	1.4	0.8	-				
TRS (as H_2S) (24 hr)	1.8366	0.272	0.06	0.02	20	0.09183	0.0136	0.0032	0.001
TRS (as H_2S) (30 min)	17.28	8.0	1.7	0.9	100	0.1728	0.080	0.017	0.009
Total HI						**0.67**	**0.3**	**0.1**	**0.1**

Table 11-22: Acute hazard quotients and indices at Receptor 6

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	163	62	23	11	10000	0.0163	0.006	0.002	0.001
NO_x (1 hr)	22	11	7	4	246	0.09	0.05	0.0	0.0
SO_2 (1 hr)	23	9	0	0	572	0.04	0.02	0.00	0.0
TSP (as PM_{10}) (24 hr)	2.23	0.98	0.5	0.3	50	0.0447	0.020	0.011	0.01
Criteria pollutants hazard index						***0.19***	***0.09***	***0.04***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0004	0.000	0.000	0.000	1.8	0.0002	0.0002	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00030	0.00020	0.00012	0.0001	-				
Cobalt (1 hr)	0.0001	0.0001	0.0001	0.0000	-				
Chromium (1 hr)	0.002	0.001	0.001	0.001	-				
Copper (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.005	0.004	0.00	0.00	-				
Nickel (1 hr)	0.0222	0.015	0.009	0.007	6	0.0037	0.0025	0.0015	0.0011
Lead (1 hr)	0.005	0.003	0.00	0.00	-				
Antimony (1 hr)	0.0002	0.0001	0.0001	0.000	-				
Selenium (1 hr)	0.0004	0.0003	0.0002	0.000	-				
Tin (1 hr)	0.003	0.002	0.001	0.00	-				
Vanadium (24 hr)	0.000098	0.00004	0.00002	0.0000	1	0.000098	0.00004	0.00002	0.0000
Metals hazard index						0.005	0.003	0.00	0.00
Chlorine (1 hr)	0.7	0	0	0	210	0.004	0.002	0.00	0.00
Sulfuric acid (1 hr)	11.7	7.7	5	3	120	0.097	0.064	0.04	0.03
Hydrogen chloride (1 hr)	31	17	7	4	2100	0.0149	0.008	0.003	0.002
Hydrogen fluoride (1 hr)	0.09	0.06	0.0	0.0	240	0.0004	0.0002	0.0001	0.0001
Acid Hazard index						**0.116**	**0.07**	**0.04**	**0.03**
TRS (as H_2S) (1 hr)	10.53	1.53	0.6	0.3	-				
TRS (as H_2S) (24 hr)	0.6517	0.116	0.03	0.01	20	0.03258	0.0058	0.0014	0.000
TRS (as H_2S) (30 min)	12.09	1.8	0.7	0.4	100	0.1209	0.018	0.007	0.004
Total HI						**0.43**	**0.2**	**0.1**	**0.1**

Table 11-23: Acute hazard quotients and indices at Receptor 7

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	113	58	17	10	10000	0.0113	0.006	0.002	0.001
NO_x (1 hr)	22	10	4	2	246	0.09	0.04	0.0	0.0
SO_2 (1 hr)	68	2	0	0	572	0.12	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.68	0.89	0.4	0.2	50	0.0337	0.018	0.007	0.00
				Criteria pollutants hazard index		***0.25***	***0.07***	***0.02***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0013	0.000	0.000	0.000	1.8	0.0007	0.0002	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00098	0.00023	0.00013	0.0001	-				
Cobalt (1 hr)	0.0003	0.0001	0.0000	0.0000	-				
Chromium (1 hr)	0.004	0.001	0.001	0.000	-				
Copper (1 hr)	0.013	0.00	0.00	0.00	100	0.00013	0.0000	0.0000	0.0000
Manganese (1 hr)	0.011	0.003	0.00	0.00	-				
Nickel (1 hr)	0.0489	0.014	0.008	0.005	6	0.0082	0.0024	0.0013	0.0008
Lead (1 hr)	0.009	0.003	0.00	0.00	-				
Antimony (1 hr)	0.0003	0.0001	0.0000	0.000	-				
Selenium (1 hr)	0.0013	0.0003	0.0002	0.000	-				
Tin (1 hr)	0.007	0.002	0.001	0.00	-				
Vanadium (24 hr)	0.000109	0.00005	0.00002	0.0000	1	0.000109	0.00005	0.00002	0.0000
					Metals hazard index	***0.010***	***0.003***	***0.00***	***0.00***
Chlorine (1 hr)	2.4	1	0	0	210	0.012	0.003	0.00	0.00
Sulfuric acid (1 hr)	22.9	7.0	3	2	120	0.191	0.058	0.03	0.02
Hydrogen chloride (1 hr)	35	9	3	2	2100	0.0167	0.004	0.001	0.001
Hydrogen fluoride (1 hr)	0.16	0.05	0.0	0.0	240	0.0007	0.0002	0.0001	0.0001
					Acid Hazard index	**0.220**	**0.07**	**0.03**	**0.02**
TRS (as H_2S) (1 hr)	2.69	0.99	0.3	0.2	-				
TRS (as H_2S) (24 hr)	0.2897	0.055	0.01	0.00	20	0.01449	0.0027	0.0007	0.000
TRS (as H_2S) (30 min)	3.09	1.1	0.4	0.2	100	0.0309	0.011	0.004	0.002
					Total HI	**0.52**	**0.1**	**0.1**	**0.0**

Table 11-24: Acute hazard quotients and indices at Receptor 8

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	98	49	15	7	10000	0.0098	0.005	0.002	0.001
NO_x (1 hr)	23	9	3	2	246	0.09	0.04	0.0	0.0
SO_2 (1 hr)	131	2	0	0	572	0.23	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.76	0.79	0.3	0.2	50	0.0353	0.016	0.007	0.00
				Criteria pollutants hazard index		***0.37***	***0.06***	***0.02***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0017	0.000	0.000	0.000	1.8	0.0009	0.0002	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00128	0.00031	0.00012	0.0001	-				
Cobalt (1 hr)	0.0003	0.0001	0.0000	0.0000	-				
Chromium (1 hr)	0.005	0.001	0.001	0.000	-				
Copper (1 hr)	0.016	0.00	0.00	0.00	100	0.00016	0.0000	0.0000	0.0000
Manganese (1 hr)	0.011	0.003	0.00	0.00	-				
Nickel (1 hr)	0.0502	0.015	0.007	0.005	6	0.0084	0.0025	0.0012	0.0008
Lead (1 hr)	0.009	0.003	0.00	0.00	-				
Antimony (1 hr)	0.0003	0.0001	0.0000	0.000	-				
Selenium (1 hr)	0.0016	0.0004	0.0002	0.000	-				
Tin (1 hr)	0.007	0.002	0.001	0.00	-				
Vanadium (24 hr)	0.000151	0.00004	0.00002	0.0000	1	0.000151	0.00004	0.00002	0.0000
					Metals hazard index	***0.010***	***0.003***	***0.00***	***0.00***
Chlorine (1 hr)	3.2	1	0	0	210	0.015	0.004	0.00	0.00
Sulfuric acid (1 hr)	23.6	6.9	3	2	120	0.197	0.057	0.03	0.02
Hydrogen chloride (1 hr)	41	7	2	1	2100	0.0195	0.003	0.001	0.001
Hydrogen fluoride (1 hr)	0.16	0.05	0.0	0.0	240	0.0007	0.0002	0.0001	0.0001
					Acid Hazard index	***0.232***	***0.06***	***0.03***	***0.02***
TRS (as H_2S) (1 hr)	2.86	0.98	0.3	0.1	-				
TRS (as H_2S) (24 hr)	0.2869	0.042	0.01	0.00	20	0.01434	0.0021	0.0007	0.000
TRS (as H_2S) (30 min)	3.29	1.1	0.3	0.2	100	0.0329	0.011	0.003	0.002
					Total HI	***0.64***	***0.1***	***0.1***	***0.0***

Table 11-25: Acute hazard quotients and indices at Receptor 9

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	53	23	9	5	10000	0.0053	0.002	0.001	0.000
NO_x (1 hr)	8	3	2	1	246	0.03	0.01	0.0	0.0
SO_2 (1 hr)	12	2	0	0	572	0.02	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.13	0.43	0.2	0.1	50	0.0225	0.009	0.004	0.00
Criteria pollutants hazard index						***0.08***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0003	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00023	0.00012	0.00007	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.001	0.000	0.000	-				
Copper (1 hr)	0.003	0.00	0.00	0.00	100	0.00003	0.0000	0.0000	0.0000
Manganese (1 hr)	0.003	0.001	0.00	0.00	-				
Nickel (1 hr)	0.0131	0.005	0.004	0.003	6	0.0022	0.0009	0.0006	0.0005
Lead (1 hr)	0.003	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0003	0.0002	0.0001	0.000	-				
Tin (1 hr)	0.002	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000068	0.00003	0.00001	0.0000	1	0.000068	0.00003	0.00001	0.0000
Metals hazard index						***0.003***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.6	0	0	0	210	0.003	0.001	0.00	0.00
Sulfuric acid (1 hr)	6.2	2.8	2	1	120	0.052	0.023	0.01	0.01
Hydrogen chloride (1 hr)	14	6	2	1	2100	0.0068	0.003	0.001	0.000
Hydrogen fluoride (1 hr)	0.04	0.02	0.0	0.0	240	0.0002	0.0001	0.0001	0.0000
Acid Hazard index						***0.061***	***0.03***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	1.96	0.51	0.2	0.1	-				
TRS (as H_2S) (24 hr)	0.1358	0.033	0.01	0.00	20	0.00679	0.0016	0.0004	0.000
TRS (as H_2S) (30 min)	2.25	0.6	0.2	0.1	100	0.0225	0.006	0.002	0.001
Total HI						***0.17***	***0.1***	***0.0***	***0.0***

Table 11-26: Acute hazard quotients and indices at Receptor 9A

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	102	29	10	5	10000	0.0102	0.003	0.001	0.001
NO_x (1 hr)	14	5	2	1	246	0.06	0.02	0.0	0.0
SO_2 (1 hr)	15	2	0	0	572	0.03	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.15	0.68	0.2	0.1	50	0.0230	0.014	0.004	0.00
				Criteria pollutants hazard index		***0.12***	***0.04***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0006	0.000	0.000	0.000	1.8	0.0003	0.0001	0.000	0.000
Arsenic (4 hr)	0.0002	0.000	0.000	0.000	-				
Beryllium (1 hr)	0.0001	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Cobalt (1 hr)	0.00045	0.00018	0.00008	0.0001	-				
Chromium (1 hr)	0.0001	0.0001	0.0000	0.0000	-				
Copper (1 hr)	0.002	0.001	0.000	0.000	-				
Manganese (1 hr)	0.006	0.00	0.00	0.00	100	0.00006	0.0000	0.0000	0.0000
Nickel (1 hr)	0.005	0.002	0.00	0.00	-				
Lead (1 hr)	0.0252	0.010	0.005	0.003	6	0.0042	0.0016	0.0008	0.0006
Antimony (1 hr)	0.004	0.002	0.00	0.00	-				
Selenium (1 hr)	0.0001	0.0001	0.0000	0.000	-				
Tin (1 hr)	5.85E-04	2.35E-04	1.03E-04	7.08E-05	-				
Vanadium (24 hr)	0.003	0.001	0.001	0.00	-				
					Metals hazard index	***0.005***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	1.1	0	0	0	210	0.005	0.002	0.00	0.00
Sulfuric acid (1 hr)	11.7	4.6	2	2	120	0.098	0.038	0.02	0.01
Hydrogen chloride (1 hr)	18	6	2	1	2100	0.0086	0.003	0.001	0.001
Hydrogen fluoride (1 hr)	0.08	0.03	0.0	0.0	240	0.0003	0.0001	0.0001	0.0000
					Acid Hazard index	***0.112***	***0.04***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	0.90	0.54	0.2	0.1	-				
TRS (as H_2S) (24 hr)	0.0749	0.037	0.01	0.00	20	0.00375	0.0018	0.0005	0.000
TRS (as H_2S) (30 min)	1.04	0.6	0.2	0.1	100	0.0104	0.006	0.002	0.001
					Total HI	***0.24***	***0.1***	***0.0***	***0.0***

Table 11-27: Acute hazard quotients and indices at Receptor 10

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	104	22	9	4	10000	0.0104	0.002	0.001	0.000
NO_x (1 hr)	9	3	2	1	246	0.04	0.01	0.0	0.0
SO_2 (1 hr)	10	1	0	0	572	0.02	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.07	0.47	0.2	0.1	50	0.0213	0.009	0.003	0.00
				Criteria pollutants hazard index		***0.09***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0005	0.000	0.000	0.000	1.8	0.0003	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00037	0.00012	0.00008	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.002	0.001	0.000	0.000	-				
Copper (1 hr)	0.005	0.00	0.00	0.00	100	0.00005	0.0000	0.0000	0.0000
Manganese (1 hr)	0.004	0.001	0.00	0.00	-				
Nickel (1 hr)	0.0188	0.007	0.004	0.003	6	0.0031	0.0011	0.0007	0.0005
Lead (1 hr)	0.003	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0005	0.0001	0.0001	0.000	-				
Tin (1 hr)	0.003	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000056	0.00003	0.00001	0.0000	1	0.000056	0.00003	0.00001	0.0000
					Metals hazard index	***0.004***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.9	0	0	0	210	0.004	0.001	0.00	0.00
Sulfuric acid (1 hr)	8.5	3.2	2	1	120	0.071	0.027	0.02	0.01
Hydrogen chloride (1 hr)	13	3	1	1	2100	0.0064	0.002	0.001	0.000
Hydrogen fluoride (1 hr)	0.06	0.02	0.0	0.0	240	0.0002	0.0001	0.0001	0.0000
					Acid Hazard index	***0.082***	***0.03***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	0.58	0.30	0.1	0.1	-				
TRS (as H_2S) (24 hr)	0.0707	0.021	0.01	0.00	20	0.00353	0.0011	0.0004	0.000
TRS (as H_2S) (30 min)	0.67	0.3	0.1	0.1	100	0.0067	0.003	0.001	0.001
					Total HI	***0.18***	***0.1***	***0.0***	***0.0***

Table 11-28: Acute hazard quotients and indices at Receptor 11

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	505	124	47	37	-				
NO_x (1 hr)	85	27	10	5	10000	0.0085	0.003	0.001	0.000
SO_2 (1 hr)	10	4	2	1	246	0.04	0.02	0.0	0.0
TSP (as PM_{10}) (24 hr)	11	1	0	0	572	0.02	0.00	0.00	0.0
				Criteria pollutants hazard index		***0.11***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0004	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00029	0.00014	0.00008	0.0001	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.001	0.000	0.000	-				
Copper (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.004	0.002	0.00	0.00	-				
Nickel (1 hr)	0.0170	0.008	0.004	0.003	6	0.0028	0.0014	0.0007	0.0006
Lead (1 hr)	0.003	0.002	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0001	0.0000	0.000	-				
Selenium (1 hr)	0.0004	0.0002	0.0001	0.000	-				
Tin (1 hr)	0.002	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000104	0.00003	0.00001	0.0000	1	0.000104	0.00003	0.00001	0.0000
					Metals hazard index	***0.004***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	0.7	0	0	0	210	0.003	0.002	0.00	0.00
Sulfuric acid (1 hr)	8.1	3.9	2	2	120	0.067	0.033	0.02	0.01
Hydrogen chloride (1 hr)	8	5	2	1	2100	0.0040	0.002	0.001	0.000
Hydrogen fluoride (1 hr)	0.06	0.03	0.0	0.0	240	0.0002	0.0001	0.0001	0.0000
					Acid Hazard index	***0.075***	***0.04***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	0.99	0.43	0.2	0.1	-				
TRS (as H_2S) (24 hr)	0.1519	0.031	0.01	0.00	20	0.00759	0.0016	0.0004	0.000
TRS (as H_2S) (30 min)	1.14	0.5	0.2	0.1	100	0.0114	0.005	0.002	0.001
					Total HI	***0.20***	***0.1***	***0.0***	***0.0***

Table 11-29: Acute hazard quotients and indices at Receptor 1

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	72	24	8	4	10000	0.0072	0.002	0.001	0.000
NO_x (1 hr)	9	4	2	1	246	0.04	0.01	0.0	0.0
SO_2 (1 hr)	4	0	0	0	572	0.01	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.83	0.41	0.1	0.1	50	0.0367	0.008	0.003	0.00
Criteria pollutants hazard index						***0.09***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0004	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00029	0.00013	0.00007	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.001	0.000	0.000	-				
Copper (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.003	0.002	0.00	0.00	-				
Nickel (1 hr)	0.0116	0.009	0.004	0.003	6	0.0019	0.0014	0.0006	0.0004
Lead (1 hr)	0.002	0.002	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0004	0.0002	0.0001	0.000	-				
Tin (1 hr)	0.002	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000096	0.00003	0.00001	0.0000	1	0.000096	0.00003	0.00001	0.0000
Metals hazard index						***0.003***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	0.7	0	0	0	210	0.003	0.002	0.00	0.00
Sulfuric acid (1 hr)	5.5	4.1	2	1	120	0.046	0.034	0.02	0.01
Hydrogen chloride (1 hr)	9	4	2	1	2100	0.0045	0.002	0.001	0.000
Hydrogen fluoride (1 hr)	0.04	0.03	0.0	0.0	240	0.0002	0.0001	0.0001	0.0000
Acid Hazard index						***0.054***	***0.04***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	0.74	0.31	0.1	0.1	-				
TRS (as H_2S) (24 hr)	0.1174	0.021	0.01	0.00	20	0.00587	0.0011	0.0003	0.000
TRS (as H_2S) (30 min)	0.85	0.4	0.1	0.1	100	0.0085	0.004	0.001	0.001
Total HI						***0.15***	***0.1***	***0.0***	***0.0***

Table 11-30: Acute hazard quotients and indices at Receptor 13

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	186	26	7	4	10000	0.0186	0.003	0.001	0.000
NO_x (1 hr)	8	4	2	1	246	0.03	0.02	0.0	0.0
SO_2 (1 hr)	8	1	0	0	572	0.01	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.82	0.57	0.2	0.1	50	0.0364	0.011	0.003	0.00
				Criteria pollutants hazard index		***0.10***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0005	0.000	0.000	0.000	1.8	0.0003	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00034	0.00013	0.00007	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.002	0.001	0.000	0.000	-				
Copper (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Manganese (1 hr)	0.004	0.002	0.00	0.00	-				
Nickel (1 hr)	0.0198	0.008	0.004	0.003	6	0.0033	0.0013	0.0007	0.0005
Lead (1 hr)	0.004	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0004	0.0002	0.0001	0.000	-				
Tin (1 hr)	0.003	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000117	0.00004	0.00001	0.0000	1	0.000117	0.00004	0.00001	0.0000
					Metals hazard index	***0.004***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	0.8	0	0	0	210	0.004	0.002	0.00	0.00
Sulfuric acid (1 hr)	9.7	4.0	2	1	120	0.081	0.033	0.02	0.01
Hydrogen chloride (1 hr)	11	5	2	1	2100	0.0052	0.002	0.001	0.000
Hydrogen fluoride (1 hr)	0.07	0.03	0.0	0.0	240	0.0003	0.0001	0.0001	0.0000
					Acid Hazard index	***0.090***	***0.04***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	2.79	0.41	0.1	0.1	-				
TRS (as H_2S) (24 hr)	0.2192	0.026	0.01	0.00	20	0.01096	0.0013	0.0003	0.000
TRS (as H_2S) (30 min)	3.20	0.5	0.1	0.1	100	0.0320	0.005	0.001	0.001
					Total HI	***0.23***	***0.1***	***0.0***	***0.0***

Table 11-31: Acute hazard quotients and indices at Receptor 14

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	414	85	28	20	-				
NO_x (1 hr)	75	18	5	3	10000	0.0075	0.002	0.001	0.000
SO_2 (1 hr)	5	2	1	1	246	0.02	0.01	0.0	0.0
TSP (as PM_{10}) (24 hr)	8	1	0	0	572	0.01	0.00	0.00	0.0
				Criteria pollutants hazard index		***0.05***	***0.02***	***0.01***	***0.00***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0003	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0000	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00023	0.00010	0.00005	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.000	0.000	0.000	-				
Copper (1 hr)	0.003	0.00	0.00	0.00	100	0.00003	0.0000	0.0000	0.0000
Manganese (1 hr)	0.002	0.001	0.00	0.00	-				
Nickel (1 hr)	0.0107	0.005	0.003	0.002	6	0.0018	0.0008	0.0005	0.0003
Lead (1 hr)	0.002	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0003	0.0001	0.0001	0.000	-				
Tin (1 hr)	0.002	0.001	0.000	0.00	-				
Vanadium (24 hr)	0.000042	0.00002	0.00001	0.0000	1	0.000042	0.00002	0.00001	0.0000
					Metals hazard index	***0.002***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.6	0	0	0	210	0.003	0.001	0.00	0.00
Sulfuric acid (1 hr)	5.2	2.3	1	1	120	0.043	0.019	0.01	0.01
Hydrogen chloride (1 hr)	7	3	1	1	2100	0.0032	0.001	0.000	0.000
Hydrogen fluoride (1 hr)	0.04	0.02	0.0	0.0	240	0.0002	0.0001	0.0000	0.0000
					Acid Hazard index	***0.049***	***0.02***	***0.01***	***0.01***
TRS (as H_2S) (1 hr)	0.38	0.18	0.1	0.0	-				
TRS (as H_2S) (24 hr)	0.0362	0.015	0.00	0.00	20	0.00181	0.0007	0.0002	0.000
TRS (as H_2S) (30 min)	0.44	0.2	0.1	0.1	100	0.0044	0.002	0.001	0.001
					Total HI	***0.11***	***0.0***	***0.0***	***0.0***

Table 11-32: Acute hazard quotients and indices at Receptor 19

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	107	23	8	4	10000	0.0107	0.002	0.001	0.000
NO_x (1 hr)	6	3	2	1	246	0.03	0.01	0.0	0.0
SO_2 (1 hr)	14	1	0	0	572	0.02	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.03	0.43	0.2	0.1	50	0.0206	0.009	0.004	0.00
Criteria pollutants hazard index						***0.08***	***0.03***	***0.01***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0003	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0000	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00023	0.00011	0.00007	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.001	0.000	0.000	-				
Copper (1 hr)	0.003	0.00	0.00	0.00	100	0.00003	0.0000	0.0000	0.0000
Manganese (1 hr)	0.002	0.002	0.00	0.00	-				
Nickel (1 hr)	0.0104	0.007	0.004	0.003	6	0.0017	0.0011	0.0006	0.0004
Lead (1 hr)	0.002	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0003	0.0001	0.0001	0.000	-				
Tin (1 hr)	0.001	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000062	0.00003	0.00001	0.0000	1	0.000062	0.00003	0.00001	0.0000
Metals hazard index						***0.002***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.6	0	0	0	210	0.003	0.001	0.00	0.00
Sulfuric acid (1 hr)	4.8	3.4	2	1	120	0.040	0.028	0.02	0.01
Hydrogen chloride (1 hr)	10	4	1	1	2100	0.0047	0.002	0.001	0.000
Hydrogen fluoride (1 hr)	0.03	0.02	0.0	0.0	240	0.0001	0.0001	0.0001	0.0000
Acid Hazard index						***0.048***	***0.03***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	0.49	0.30	0.1	0.1	-				
TRS (as H_2S) (24 hr)	0.0527	0.020	0.01	0.00	20	0.00263	0.0010	0.0003	0.000
TRS (as H_2S) (30 min)	0.56	0.4	0.1	0.1	100	0.0056	0.004	0.001	0.001
Total HI						***0.14***	***0.1***	***0.0***	***0.0***

Table 11-33: Acute hazard quotients and indices at Receptor 21

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	122	55	22	11	10000	0.0122	0.005	0.002	0.001
NO_x (1 hr)	23	10	5	3	246	0.09	0.04	0.0	0.0
SO_2 (1 hr)	40	8	0	0	572	0.07	0.01	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.57	0.89	0.4	0.2	50	0.0313	0.018	0.007	0.00
Criteria pollutants hazard index						***0.21***	***0.08***	***0.03***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0005	0.000	0.000	0.000	1.8	0.0003	0.0001	0.000	0.000
Arsenic (4 hr)	0.0002	0.000	0.000	0.000	-				
Beryllium (1 hr)	0.0001	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Cobalt (1 hr)	0.00035	0.00017	0.00010	0.0001	-				
Chromium (1 hr)	0.0001	0.0001	0.0000	0.0000	-				
Copper (1 hr)	0.002	0.001	0.001	0.000	-				
Manganese (1 hr)	0.004	0.00	0.00	0.00	100	0.00004	0.0000	0.0000	0.0000
Nickel (1 hr)	0.004	0.003	0.00	0.00	-				
Lead (1 hr)	0.0187	0.012	0.007	0.005	6	0.0031	0.0020	0.0011	0.0008
Antimony (1 hr)	0.004	0.003	0.00	0.00	-				
Selenium (1 hr)	0.0001	0.0001	0.0000	0.000	-				
Tin (1 hr)	0.0005	0.0002	0.0001	0.000	-				
Vanadium (24 hr)	0.003	0.002	0.001	0.00	-				
Metals hazard index						***0.004***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	0.9	0	0	0	210	0.004	0.002	0.00	0.00
Sulfuric acid (1 hr)	8.6	6.3	3	3	120	0.072	0.053	0.03	0.02
Hydrogen chloride (1 hr)	26	13	6	3	2100	0.0126	0.006	0.003	0.001
Hydrogen fluoride (1 hr)	0.06	0.05	0.0	0.0	240	0.0003	0.0002	0.0001	0.0001
Acid Hazard index						***0.089***	***0.06***	***0.03***	***0.02***
TRS (as H_2S) (1 hr)	7.13	1.81	0.5	0.2	-				
TRS (as H_2S) (24 hr)	0.8803	0.091	0.02	0.01	20	0.04401	0.0046	0.0012	0.000
TRS (as H_2S) (30 min)	8.19	2.1	0.6	0.3	100	0.0819	0.021	0.006	0.003
Total HI						***0.38***	***0.2***	***0.1***	***0.0***

Table 11-34: Acute hazard quotients and indices at Receptor 22

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	232	77	19	10	10000	0.0232	0.008	0.002	0.001
NO_x (1 hr)	37	8	4	3	246	0.15	0.03	0.0	0.0
SO_2 (1 hr)	70	5	0	0	572	0.12	0.01	0.00	0.0
TSP (as PM_{10}) (24 hr)	1.28	0.79	0.4	0.2	50	0.0257	0.016	0.008	0.00
				Criteria pollutants hazard index		***0.32***	***0.07***	***0.03***	***0.02***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0008	0.000	0.000	0.000	1.8	0.0004	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	0.19	0.001	0.000	0.00	0.00
Beryllium (1 hr)	0.00060	0.00019	0.00012	0.0001	-				
Cobalt (1 hr)	0.0002	0.0001	0.0000	0.0000	-				
Chromium (1 hr)	0.003	0.001	0.001	0.000	-				
Copper (1 hr)	0.008	0.00	0.00	0.00	100	0.00008	0.0000	0.0000	0.0000
Manganese (1 hr)	0.007	0.003	0.00	0.00	-				
Nickel (1 hr)	0.0306	0.011	0.007	0.005	6	0.0051	0.0018	0.0011	0.0009
Lead (1 hr)	0.007	0.002	0.00	0.00	-				
Antimony (1 hr)	0.0002	0.0001	0.0000	0.000	-				
Selenium (1 hr)	0.0008	0.0002	0.0002	0.000	-				
Tin (1 hr)	0.004	0.002	0.001	0.00	-				
Vanadium (24 hr)	0.000068	0.00005	0.00002	0.0000	1	0.000068	0.00005	0.00002	0.0000
				Metals hazard index		***0.006***	***0.002***	***0.00***	***0.00***
Chlorine (1 hr)	1.5	0	0	0	210	0.007	0.002	0.00	0.00
Sulfuric acid (1 hr)	16.1	5.7	3	2	120	0.134	0.048	0.03	0.02
Hydrogen chloride (1 hr)	20	9	4	2	2100	0.0097	0.004	0.002	0.001
Hydrogen fluoride (1 hr)	0.12	0.04	0.0	0.0	240	0.0005	0.0002	0.0001	0.0001
				Acid Hazard index		***0.151***	***0.05***	***0.03***	***0.02***
TRS (as H_2S) (1 hr)	5.09	0.85	0.3	0.2	-				
TRS (as H_2S) (24 hr)	0.1519	0.067	0.02	0.00	20	0.00759	0.0034	0.0008	0.000
TRS (as H_2S) (30 min)	5.85	1.0	0.3	0.2	100	0.0585	0.010	0.003	0.002
					Total HI	***0.54***	***0.1***	***0.1***	***0.0***

Table 11-35: Acute hazard quotients and indices at Receptor 26

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	60	27	11	5	10000	0.0060	0.003	0.001	0.000
NO_x (1 hr)	9	4	2	2	246	0.03	0.02	0.0	0.0
SO_2 (1 hr)	13	5	0	0	572	0.02	0.01	0.00	0.0
TSP (as PM_{10}) (24 hr)	0.64	0.47	0.2	0.1	50	0.0127	0.009	0.004	0.00
				Criteria pollutants hazard index		***0.08***	***0.04***	***0.02***	***0.01***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0003	0.000	0.000	0.000	1.8	0.0002	0.0001	0.000	0.000
Arsenic (4 hr)	0.0000	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Beryllium (1 hr)	0.00022	0.00011	0.00007	0.0000	-				
Cobalt (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Chromium (1 hr)	0.001	0.001	0.000	0.000	-				
Copper (1 hr)	0.003	0.00	0.00	0.00	100	0.00003	0.0000	0.0000	0.0000
Manganese (1 hr)	0.003	0.002	0.00	0.00	-				
Nickel (1 hr)	0.0126	0.007	0.004	0.003	6	0.0021	0.0011	0.0007	0.0005
Lead (1 hr)	0.002	0.001	0.00	0.00	-				
Antimony (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Selenium (1 hr)	0.0003	0.0001	0.0001	0.000	-				
Tin (1 hr)	0.002	0.001	0.001	0.00	-				
Vanadium (24 hr)	0.000034	0.00002	0.00001	0.0000	1	0.000034	0.00002	0.00001	0.0000
					Metals hazard index	***0.003***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.5	0	0	0	210	0.003	0.001	0.00	0.00
Sulfuric acid (1 hr)	5.9	3.4	2	1	120	0.049	0.028	0.02	0.01
Hydrogen chloride (1 hr)	14	6	2	1	2100	0.0066	0.003	0.001	0.001
Hydrogen fluoride (1 hr)	0.04	0.02	0.0	0.0	240	0.0002	0.0001	0.0001	0.0000
					Acid Hazard index	***0.059***	***0.03***	***0.02***	***0.01***
TRS (as H_2S) (1 hr)	2.58	0.75	0.2	0.1	-				
TRS (as H_2S) (24 hr)	0.4252	0.030	0.01	0.00	20	0.02126	0.0015	0.0004	0.000
TRS (as H_2S) (30 min)	2.97	0.9	0.2	0.1	100	0.0297	0.009	0.002	0.001
					Total HI	***0.17***	***0.1***	***0.0***	***0.0***

Table 11-36: Acute hazard quotients and indices at Receptor 27

	Percentiles (ug/m^3)				AGV	Hazard quotients			
Pollutant	**100**	**99.9**	**99.5**	**99**		**100**	**99.9**	**99.5**	**99**
CO (8 hr)	34	15	5	2	10000	0.0034	0.001	0.000	0.000
NO_x (1 hr)	6	3	1	1	246	0.02	0.01	0.0	0.0
SO_2 (1 hr)	4	1	0	0	572	0.01	0.00	0.00	0.0
TSP (as PM_{10}) (24 hr)	0.64	0.30	0.1	0.0	50	0.0129	0.006	0.002	0.00
				Criteria pollutants hazard index		***0.05***	***0.02***	***0.01***	***0.00***
Cadmium (1 hr)	0.0000	0.0000	0.0000	0.000	-				
Mercury (1 hr)	0.0002	0.000	0.000	0.000	1.8	0.0001	0.0001	0.000	0.000
Arsenic (4 hr)	0.0001	0.000	0.000	0.000	-				
Beryllium (1 hr)	0.0000	0.000	0.000	0.000	0.19	0.000	0.000	0.00	0.00
Cobalt (1 hr)	0.00014	0.00009	0.00004	0.0000	-				
Chromium (1 hr)	0.0001	0.0000	0.0000	0.0000	-				
Copper (1 hr)	0.001	0.000	0.000	0.000	-				
Manganese (1 hr)	0.002	0.00	0.00	0.00	100	0.00002	0.0000	0.0000	0.0000
Nickel (1 hr)	0.002	0.001	0.00	0.00	-				
Lead (1 hr)	0.0101	0.005	0.002	0.002	6	0.0017	0.0008	0.0004	0.0003
Antimony (1 hr)	0.002	0.001	0.00	0.00	-				
Selenium (1 hr)	0.0001	0.0000	0.0000	0.000	-				
Tin (1 hr)	0.0002	0.0001	0.0001	0.000	-				
Vanadium (24 hr)	0.001	0.001	0.000	0.00	-				
					Metals hazard index	***0.002***	***0.001***	***0.00***	***0.00***
Chlorine (1 hr)	0.4	0	0	0	210	0.002	0.001	0.00	0.00
Sulfuric acid (1 hr)	5.0	2.4	1	1	120	0.042	0.020	0.01	0.01
Hydrogen chloride (1 hr)	11	3	1	1	2100	0.0054	0.001	0.000	0.000
Hydrogen fluoride (1 hr)	0.04	0.02	0.0	0.0	240	0.0001	0.0001	0.0000	0.0000
					Acid Hazard index	***0.049***	***0.02***	***0.01***	***0.01***
TRS (as H_2S) (1 hr)	2.78	0.40	0.1	0.1	-				
TRS (as H_2S) (24 hr)	0.3274	0.024	0.00	0.00	20	0.01637	0.0012	0.0002	0.000
TRS (as H_2S) (30 min)	3.19	0.5	0.1	0.1	100	0.0319	0.005	0.001	0.001
					Total HI	***0.13***	***0.0***	***0.0***	***0.0***

APPENDIX D CHRONIC HAZARD INDICES AND QUOTIENTS

Table 11-37: Chronic hazard quotients and indices at Receptor 1

Pollutant	100	AGV	HQ
CO	2.2199	-	
NO_2	0.2	62	0.003
SO_2	0.0088328	57	0.000
TSP	0.11944	20	0.0060
		Criteria pollutants hazard index	**0.009**
Cadmium	1.2209E-07	0.005	0.0000
Mercury	3.5009E-06	1	0.000004
Arsenic	1.9724E-06	0.03	0.0001
Beryllium	0.000002563	0.02	0.0001282
Cobalt	0.000001083	0.1	0.00001
Chromium	0.000014791	-	
Cr III (95%)	0.000014	60	0.0000002
Cr VI (5%)	0.0000007	0.1	0.000007
Copper	0.000030705	1	0.00003
Manganese	0.000041559	0.15	0.0003
Nickel	0.00018243	0.05	0.0036
Lead	0.000035501	0.5	0.00007
Antimony	1.1715E-06	0.2	0.000006
Selenium	0.000003355	20	0.0000002
Tin	0.000025796	48	0.0000005
Vanadium	5.6025E-06	1	0.000006
		Metals hazard index	**0.004**
Chlorine	0.0062115	0.2	0.031
Sulfuric acid	0.089981	1	0.090
Hydrogen chloride	0.10137	9	0.011
Hydrogen fluoride	0.00063923	-	
		Acid Hazard index	0.13
TRS (as H_2S)	0.011789	2	0.0059
		Total HI	**0.15**

Table 11-38: Chronic hazard quotients and indices at Receptor 2

Pollutant	100	AGV	HQ
CO	3.39E+00	-	
NO_2	4.00E-01	62	0.006
SO_2	5.34E-03	57	0.000
TSP	1.68E-01	20	0.0084
		Criteria pollutants hazard index	**0.015**
Cadmium	1.68E-07	0.005	0.0000
Mercury	4.60E-06	1	0.000005
Arsenic	2.76E-06	0.03	0.0001
Beryllium	3.36E-06	0.02	0.0001679
Cobalt	1.50E-06	0.1	0.00002
Chromium	2.00E-05	-	
Cr III (95%)	0.000019	60	0.0000003
Cr VI (5%)	0.0000010	0.1	0.000010
Copper	3.99E-05	1	0.00004
Manganese	0.00006	0.15	0.0004
Nickel	2.50E-04	0.05	0.0050
Lead	4.96E-05	0.5	0.00010
Antimony	1.65E-06	0.2	0.000008
Selenium	4.41E-06	20	0.0000002
Tin	3.56E-05	48	0.0000007
Vanadium	7.28E-06	1	0.000007
		Metals hazard index	**0.006**
Chlorine	8.11E-03	0.2	0.041
Sulfuric acid	1.25E-01	1	0.125
Hydrogen chloride	7.31E-02	9	0.008
Hydrogen fluoride	8.89E-04	-	
		Acid Hazard index	**0.17**
TRS (as H_2S)	8.15E-03	2	0.0041
		Total HI	**0.20**

Table 11-39: Chronic hazard quotients and indices at Receptor 3

Pollutant	100	AGV	HQ
CO	6.15E+00	-	
NO_2	2.80E-01	62	0.005
SO_2	5.43E-02	57	0.001
TSP	3.31E-01	20	0.0166
		Criteria pollutants hazard index	**0.022**
Cadmium	3.19E-07	0.005	0.0001
Mercury	8.46E-06	1	0.000008
Arsenic	5.33E-06	0.03	0.0002
Beryllium	6.17E-06	0.02	0.0003083
Cobalt	2.88E-06	0.1	0.00003
Chromium	3.76E-05	-	
Cr III (95%)	0.000036	60	0.0000006
Cr VI (5%)	0.0000019	0.1	0.000019
Copper	7.29E-05	1	0.00007
Manganese	1.10E-04	0.15	0.0007
Nickel	4.75E-04	0.05	0.0095
Lead	9.55E-05	0.5	0.00019
Antimony	3.19E-06	0.2	0.000016
Selenium	8.10E-06	20	0.0000004
Tin	6.78E-05	48	0.0000014
Vanadium	1.33E-05	1	0.000013
		Metals hazard index	0.011
Chlorine	1.49E-02	0.2	0.074
Sulfuric acid	2.38E-01	1	0.238
Hydrogen chloride	2.26E-01	9	0.025
Hydrogen fluoride	1.70E-03	-	
		Acid Hazard index	0.34
TRS (as H_2S)	3.65E-02	2	0.0182
		Total HI	0.39

Table 11-40: Chronic hazard quotients and indices at Receptor 4

Pollutant	100	AGV	HQ
CO	2.92E+00	-	
NO_2	1.22E-01	62	0.002
SO_2	1.24E-02	57	0.000
TSP	1.67E-01	20	0.0083
		Criteria pollutants hazard index	0.011
Cadmium	1.67E-07	0.005	0.0000
Mercury	4.70E-06	1	0.000005
Arsenic	2.72E-06	0.03	0.0001
Beryllium	3.44E-06	0.02	0.0001720
Cobalt	1.49E-06	0.1	0.00001
Chromium	2.01E-05	-	
Cr III (95%)	0.000019	60	0.0000003
Cr VI (5%)	0.0000010	0.1	0.000010
Copper	4.11E-05	1	0.00004
Manganese	5.70E-05	0.15	0.0004
Nickel	2.49E-04	0.05	0.0050
Lead	4.89E-05	0.5	0.00010
Antimony	1.62E-06	0.2	0.000008
Selenium	4.51E-06	20	0.0000002
Tin	3.53E-05	48	0.0000007
Vanadium	7.49E-06	1	0.000007
		Metals hazard index	0.006
Chlorine	8.33E-03	0.2	0.042
Sulfuric acid	1.23E-01	1	0.123
Hydrogen chloride	1.07E-01	9	0.012
Hydrogen fluoride	8.78E-04	-	
		Acid Hazard index	0.18
TRS (as H_2S)	1.93E-02	2	0.0096
		Total HI	0.20

Table 11-41: Chronic hazard quotients and indices at Receptor 5

Pollutant	100	AGV	HQ
CO	6.15E+00	-	
NO_2	2.80E-01	62	0.005
SO_2	5.43E-02	57	0.001
TSP	3.31E-01	20	0.0166
		Criteria pollutants hazard index	0.022
Cadmium	3.19E-07	0.005	0.0001
Mercury	8.46E-06	1	0.000008
Arsenic	5.33E-06	0.03	0.0002
Beryllium	6.17E-06	0.02	0.0003083
Cobalt	2.88E-06	0.1	0.00003
Chromium	3.76E-05	-	
Cr III (95%)	0.000036	60	0.0000006
Cr VI (5%)	0.0000019	0.1	0.000019
Copper	7.29E-05	1	0.00007
Manganese	1.10E-04	0.15	0.0007
Nickel	4.75E-04	0.05	0.0095
Lead	9.55E-05	0.5	0.00019
Antimony	3.19E-06	0.2	0.000016
Selenium	8.10E-06	20	0.0000004
Tin	6.78E-05	48	0.0000014
Vanadium	1.33E-05	1	0.000013
		Metals hazard index	0.011
Chlorine	1.49E-02	0.2	0.074
Sulfuric acid	2.38E-01	1	0.238
Hydrogen chloride	2.26E-01	9	0.025
Hydrogen fluoride	1.70E-03	-	
		Acid Hazard index	0.34
TRS (as H_2S)	3.65E-02	2	0.0182
		Total HI	0.39

Table 11-42: Chronic hazard quotients and indices at Receptor 6

Pollutant	100	AGV	HQ
CO	3.36E+00	-	
NO_2	1.48E-01	62	0.002
SO_2	2.23E-02	57	0.000
TSP	1.89E-01	20	0.0094
		Criteria pollutants hazard index	0.012
Cadmium	1.86E-07	0.005	0.0000
Mercury	5.14E-06	1	0.000005
Arsenic	3.05E-06	0.03	0.0001
Beryllium	3.75E-06	0.02	0.0001877
Cobalt	1.66E-06	0.1	0.00002
Chromium	2.22E-05	-	
Cr III (95%)	0.000021	60	0.0000004
Cr VI (5%)	0.0000011	0.1	0.000011
Copper	4.47E-05	1	0.00004
Manganese	6.36E-05	0.15	0.0004
Nickel	2.77E-04	0.05	0.0055
Lead	5.48E-05	0.5	0.00011
Antimony	1.82E-06	0.2	0.000009
Selenium	4.92E-06	20	0.0000002
Tin	3.94E-05	48	0.0000008
Vanadium	8.15E-06	1	0.000008
		Metals hazard index	0.006
Chlorine	9.08E-03	0.2	0.045
Sulfuric acid	1.38E-01	1	0.138
Hydrogen chloride	1.33E-01	9	0.015
Hydrogen fluoride	9.81E-04	-	
		Acid Hazard index	0.20
TRS (as H_2S)	1.45E-02	2	0.0073
		Total HI	0.22

Table 11-43: Chronic hazard quotients and indices at Receptor 7

Pollutant	100	AGV	HQ
CO	2.826700	-	
NO_2	0.10	62	0.002
SO_2	0.0	57	0.000
TSP	1.41E-01	20	0.0070
		Criteria pollutants hazard index	0.009
Cadmium	1.39E-07	0.005	0.0000
Mercury	4.69E-06	1	0.000005
Arsenic	0.00000	0.03	0.0001
Beryllium	0.000003465	0.02	0.0001733
Cobalt	1.1891E-06	0.1	0.0000119
Chromium	0.000018	-	
Cr III (95%)	0.000017	60	0.0000003
Cr VI (5%)	0.0000009	0.1	0.000009
Copper	0.00004	1	0.00004
Manganese	4.61E-05	0.15	0.0003
Nickel	2.10E-04	0.05	0.0042
Lead	3.78E-05	0.5	0.00008
Antimony	1.21E-06	0.2	0.000006
Selenium	0.000005	20	0.0000002
Tin	0.00003	48	0.0000006
Vanadium	0.000008	1	0.000008
		Metals hazard index	0.005
Chlorine	8.48E-03	0.2	0.042
Sulfuric acid	9.99E-02	1	0.100
Hydrogen chloride	7.14E-02	9	0.008
Hydrogen fluoride	6.97E-04	-	
		Acid Hazard index	0.15
TRS (as H_2S)	0.0069	2	0.0035
		Total HI	0.17

Table 11-44: Chronic hazard quotients and indices at Receptor 8

Pollutant	100	AGV	HQ
CO	2.37E+00	-	
NO_2	0.08	62	0.001
SO_2	2.64E-02	57	0.000
TSP	1.30E-01	20	0.0065
		Criteria pollutants hazard index	0.008
Cadmium	1.30E-07	0.005	0.0000
Mercury	4.42E-06	1	0.000004
Arsenic	1.93E-06	0.03	0.0001
Beryllium	3.27E-06	0.02	0.0001633
Cobalt	1.11E-06	0.1	0.00001
Chromium	1.68E-05	-	
Cr III (95%)	0.000016	60	0.0000003
Cr VI (5%)	0.0000008	0.1	0.000008
Copper	4.01E-05	1	0.00004
Manganese	4.30E-05	0.15	0.0003
Nickel	1.96E-04	0.05	0.0039
Lead	3.51E-05	0.5	0.00007
Antimony	1.12E-06	0.2	0.000006
Selenium	4.25E-06	20	0.0000002
Tin	2.71E-05	48	0.0000006
Vanadium	7.35E-06	1	0.000007
		Metals hazard index	0.005
Chlorine	7.99E-03	0.2	0.040
Sulfuric acid	9.32E-02	1	0.093
Hydrogen chloride	6.12E-02	9	0.007
Hydrogen fluoride	6.49E-04	-	
		Acid Hazard index	0.14
TRS (as H_2S)	6.13E-03	2	0.0031
		Total HI	0.16

Table 11-45: Chronic hazard quotients and indices at Receptor 9

Pollutant	100	AGV	HQ
CO	1.3	-	
NO_2	0.04	62	0.001
SO_2	0.0	57	0.000
TSP	0.07	20	0.0035
		Criteria pollutants hazard index	0.004
Cadmium	0.000000	0.005	0.0000
Mercury	0.000002	1	0.000002
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001785	0.02	0.0000893
Cobalt	0.000001	0.1	0.00001
Chromium	0.000009	-	
Cr III (95%)	0.000009	60	0.0000001
Cr VI (5%)	0.0000005	0.1	0.000005
Copper	0.00002	1	0.00002
Manganese	0.00002	0.15	0.0002
Nickel	0.000107	0.05	0.0021
Lead	0.00002	0.5	0.00004
Antimony	0.000001	0.2	0.000003
Selenium	0.000002	20	0.0000001
Tin	0.00001	48	0.0000003
Vanadium	0.000004	1	0.000004
		Metals hazard index	0.003
Chlorine	0.004	0.2	0.022
Sulfuric acid	0.051	1	0.051
Hydrogen chloride	0.04	9	0.005
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.08
TRS (as H_2S)	0.0038	2	0.0019
		Total HI	0.09

Table 11-46: Chronic hazard quotients and indices at Receptor 9A

Pollutant	100	AGV	HQ
CO	1.6	-	
NO_2	0.06	62	0.001
SO_2	0.0	57	0.000
TSP	0.09	20	0.0046
		Criteria pollutants hazard index	0.006
Cadmium	0.000000	0.005	0.0000
Mercury	0.000003	1	0.000003
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000002245	0.02	0.0001123
Cobalt	0.000001	0.1	0.00001
Chromium	0.000012	-	
Cr III (95%)	0.000011	60	0.0000002
Cr VI (5%)	0.0000006	0.1	0.000006
Copper	0.00003	1	0.00003
Manganese	0.00003	0.15	0.0002
Nickel	0.000140	0.05	0.0028
Lead	0.00003	0.5	0.00005
Antimony	0.000001	0.2	0.000004
Selenium	0.000003	20	0.0000001
Tin	0.00002	48	0.0000004
Vanadium	0.000005	1	0.000005
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.027
Sulfuric acid	0.067	1	0.067
Hydrogen chloride	0.05	9	0.005
Hydrogen fluoride	0.0005	-	
		Acid Hazard index	0.10
TRS (as H_2S)	0.0042	2	0.0021
		Total HI	0.11

Table 11-47: Chronic hazard quotients and indices at Receptor 10

Pollutant	100	AGV	HQ
CO	1.3	-	
NO_2	0.05	62	0.001
SO_2	0.0	57	0.000
TSP	0.07	20	0.0037
		Criteria pollutants hazard index	0.004
Cadmium	0.000000	0.005	0.0000
Mercury	0.000003	1	0.000003
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001892	0.02	0.0000946
Cobalt	0.000001	0.1	0.00001
Chromium	0.000010	-	
Cr III (95%)	0.000009	60	0.0000002
Cr VI (5%)	0.0000005	0.1	0.000005
Copper	0.00002	1	0.00002
Manganese	0.00002	0.15	0.0002
Nickel	0.000112	0.05	0.0022
Lead	0.00002	0.5	0.00004
Antimony	0.000001	0.2	0.000003
Selenium	0.000002	20	0.0000001
Tin	0.00002	48	0.0000003
Vanadium	0.000004	1	0.000004
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.023
Sulfuric acid	0.053	1	0.053
Hydrogen chloride	0.03	9	0.004
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.08
TRS (as H_2S)	0.0028	2	0.0014
		Total HI	0.09

Table 11-48: Chronic hazard quotients and indices at Receptor 11

Pollutant	100	AGV	HQ
CO	1.4	-	
NO_2	0.05	62	0.001
SO_2	0.0	57	0.000
TSP	0.09	20	0.0043
		Criteria pollutants hazard index	0.005
Cadmium	0.000000	0.005	0.0000
Mercury	0.000003	1	0.000003
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000002191	0.02	0.0001096
Cobalt	0.000001	0.1	0.00001
Chromium	0.000011	-	
Cr III (95%)	0.000011	60	0.0000002
Cr VI (5%)	0.0000006	0.1	0.000006
Copper	0.00003	1	0.00003
Manganese	0.00003	0.15	0.0002
Nickel	0.000131	0.05	0.0026
Lead	0.00002	0.5	0.00005
Antimony	0.000001	0.2	0.000004
Selenium	0.000003	20	0.0000001
Tin	0.00002	48	0.0000004
Vanadium	0.000005	1	0.000005
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.027
Sulfuric acid	0.062	1	0.062
Hydrogen chloride	0.04	9	0.005
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.09
TRS (as H_2S)	0.0035	2	0.0018
		Total HI	0.10

Table 11-49: Chronic hazard quotients and indices at Receptor 12

Pollutant	100	AGV	HQ
CO	1.2	-	
NO_2	0.04	62	0.001
SO_2	0.003	57	0.000
TSP	0.07	20	0.0035
		Criteria pollutants hazard index	0.004
Cadmium	0.000000	0.005	0.0000
Mercury	0.000002	1	0.000002
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001841	0.02	0.0000921
Cobalt	0.000001	0.1	0.00001
Chromium	0.000009	-	
Cr III (95%)	0.000009	60	0.0000001
Cr VI (5%)	0.0000005	0.1	0.000005
Copper	0.00002	1	0.00002
Manganese	0.00002	0.15	0.0002
Nickel	0.000109	0.05	0.0022
Lead	0.00002	0.5	0.00004
Antimony	0.000001	0.2	0.000003
Selenium	0.000002	20	0.0000001
Tin	0.00002	48	0.0000003
Vanadium	0.000004	1	0.000004
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.023
Sulfuric acid	0.052	1	0.052
Hydrogen chloride	0.04	9	0.004
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.08
TRS (as H_2S)	0.0029	2	0.0014
		Total HI	0.09

Table 11-50: Chronic hazard quotients and indices at Receptor 13

Pollutant	100	AGV	HQ
CO	1.3	-	
NO_2	0.05	62	0.001
SO_2	0.0	57	0.000
TSP	0.08	20	0.0039
		Criteria pollutants hazard index	0.005
Cadmium	0.000000	0.005	0.0000
Mercury	0.000003	1	0.000003
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000002012	0.02	0.0001006
Cobalt	0.000001	0.1	0.00001
Chromium	0.000010	-	
Cr III (95%)	0.000010	60	0.0000002
Cr VI (5%)	0.0000005	0.1	0.000005
Copper	0.00002	1	0.00002
Manganese	0.00003	0.15	0.0002
Nickel	0.000123	0.05	0.0025
Lead	0.00002	0.5	0.00004
Antimony	0.000001	0.2	0.000004
Selenium	0.000003	20	0.0000001
Tin	0.00002	48	0.0000004
Vanadium	0.000005	1	0.000005
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.025
Sulfuric acid	0.058	1	0.058
Hydrogen chloride	0.04	9	0.004
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.09
TRS (as H_2S)	0.0035	2	0.0017
		Total HI	0.10

Table 11-51: Chronic hazard quotients and indices at Receptor 14

Pollutant	100	AGV	HQ
CO	0.8	-	
NO_2	0.03	62	0.000
SO_2	0.0	57	0.000
TSP	0.04	20	0.0022
		Criteria pollutants hazard index	0.003
Cadmium	0.000000	0.005	0.0000
Mercury	0.000002	1	0.000002
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001203	0.02	0.0000602
Cobalt	0.000000	0.1	0.00000
Chromium	0.000006	-	
Cr III (95%)	0.000006	60	0.0000001
Cr VI (5%)	0.0000003	0.1	0.000003
Copper	0.00001	1	0.00001
Manganese	0.00002	0.15	0.0001
Nickel	0.000071	0.05	0.0014
Lead	0.00001	0.5	0.00003
Antimony	0.000000	0.2	0.000002
Selenium	0.000002	20	0.0000001
Tin	0.00001	48	0.0000002
Vanadium	0.000003	1	0.000003
		Metals hazard index	0.002
Chlorine	0.003	0.2	0.015
Sulfuric acid	0.033	1	0.033
Hydrogen chloride	0.02	9	0.002
Hydrogen fluoride	0.0002	-	
		Acid Hazard index	0.05
TRS (as H_2S)	0.0018	2	0.0009
		Total HI	0.06

Table 11-52: Chronic hazard quotients and indices at Receptor 19

Pollutant	100	AGV	HQ
CO	1.2	-	
NO_2	0.04	62	0.001
SO_2	0.0	57	0.000
TSP	0.07	20	0.0034
		Criteria pollutants hazard index	0.004
Cadmium	0.000000	0.005	0.0000
Mercury	0.000002	1	0.000002
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001734	0.02	0.0000867
Cobalt	0.000001	0.1	0.00001
Chromium	0.000009	-	
Cr III (95%)	0.000008	60	0.0000001
Cr VI (5%)	0.0000004	0.1	0.000004
Copper	0.00002	1	0.00002
Manganese	0.00002	0.15	0.0002
Nickel	0.000103	0.05	0.0021
Lead	0.00002	0.5	0.00004
Antimony	0.000001	0.2	0.000003
Selenium	0.000002	20	0.0000001
Tin	0.00001	48	0.0000003
Vanadium	0.000004	1	0.000004
		Metals hazard index	0.002
Chlorine	0.004	0.2	0.021
Sulfuric acid	0.049	1	0.049
Hydrogen chloride	0.03	9	0.003
Hydrogen fluoride	0.0003	-	
		Acid Hazard index	0.07
TRS (as H_2S)	0.0026	2	0.0013
		Total HI	0.08

Table 11-53: Chronic hazard quotients and indices at Receptor 21

Pollutant	100	AGV	HQ
CO	3.2	-	
NO_2	0.13	62	0.002
SO_2	0.0	57	0.001
TSP	0.15	20	0.0076
		Criteria pollutants hazard index	0.010
Cadmium	0.000000	0.005	0.0000
Mercury	0.000005	1	0.000005
Arsenic	0.00000	0.03	0.0001
Beryllium	0.000003342	0.02	0.0001671
Cobalt	0.000001	0.1	0.00001
Chromium	0.000019	-	
Cr III (95%)	0.000018	60	0.0000003
Cr VI (5%)	0.0000009	0.1	0.000009
Copper	0.00004	1	0.00004
Manganese	0.00005	0.15	0.0003
Nickel	0.000224	0.05	0.0045
Lead	0.00004	0.5	0.00008
Antimony	0.000001	0.2	0.000007
Selenium	0.000004	20	0.0000002
Tin	0.00003	48	0.0000007
Vanadium	0.000007	1	0.000007
		Metals hazard index	0.005
Chlorine	0.008	0.2	0.041
Sulfuric acid	0.109	1	0.109
Hydrogen chloride	0.12	9	0.013
Hydrogen fluoride	0.0008	-	
		Acid Hazard index	0.16
TRS (as H_2S)	0.0132	2	0.0066
		Total HI	0.19

Table 11-54: Chronic hazard quotients and indices at Receptor 22

Pollutant	100	AGV	HQ
CO	3.3	-	
NO_2	0.10	62	0.002
SO_2	0.0	57	0.000
TSP	0.15	20	0.0074
		Criteria pollutants hazard index	0.009
Cadmium	0.000000	0.005	0.0000
Mercury	0.000005	1	0.000005
Arsenic	0.00000	0.03	0.0001
Beryllium	0.000003539	0.02	0.0001769
Cobalt	0.000001	0.1	0.00001
Chromium	0.000019	-	
Cr III (95%)	0.000018	60	0.0000003
Cr VI (5%)	0.0000009	0.1	0.000009
Copper	0.00004	1	0.00004
Manganese	0.00005	0.15	0.0003
Nickel	0.000224	0.05	0.0045
Lead	0.00004	0.5	0.00008
Antimony	0.000001	0.2	0.000007
Selenium	0.000005	20	0.0000002
Tin	0.00003	48	0.0000006
Vanadium	0.000008	1	0.000008
		Metals hazard index	0.005
Chlorine	0.009	0.2	0.043
Sulfuric acid	0.108	1	0.108
Hydrogen chloride	0.08	9	0.009
Hydrogen fluoride	0.0008	-	
		Acid Hazard index	0.16
TRS (as H_2S)	0.0079	2	0.0039
		Total HI	0.18

Table 11-55: Chronic hazard quotients and indices at Receptor 26

Pollutant	100	AGV	HQ
CO	1.4	-	
NO_2	0.06	62	0.001
SO_2	0.0	57	0.000
TSP	0.08	20	0.0041
		Criteria pollutants hazard index	0.005
Cadmium	0.000000	0.005	0.0000
Mercury	0.000003	1	0.000003
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000002033	0.02	0.0001017
Cobalt	0.000001	0.1	0.00001
Chromium	0.000011	-	
Cr III (95%)	0.000010	60	0.0000002
Cr VI (5%)	0.0000005	0.1	0.000005
Copper	0.00002	1	0.00002
Manganese	0.00003	0.15	0.0002
Nickel	0.000129	0.05	0.0026
Lead	0.00002	0.5	0.00005
Antimony	0.000001	0.2	0.000004
Selenium	0.000003	20	0.0000001
Tin	0.00002	48	0.0000004
Vanadium	0.000005	1	0.000005
		Metals hazard index	0.003
Chlorine	0.005	0.2	0.025
Sulfuric acid	0.062	1	0.062
Hydrogen chloride	0.05	9	0.006
Hydrogen fluoride	0.0004	-	
		Acid Hazard index	0.09
TRS (as H_2S)	0.0051	2	0.0025
		Total HI	0.10

Table 11-56: Chronic hazard quotients and indices at Receptor 27

Pollutant	100	AGV	HQ
CO	0.8	-	
NO_2	0.03	62	0.000
SO_2	0.0	57	0.000
TSP	0.04	20	0.0021
		Criteria pollutants hazard index	0.003
Cadmium	0.000000	0.005	0.0000
Mercury	0.000002	1	0.000002
Arsenic	0.00000	0.03	0.0000
Beryllium	0.000001147	0.02	0.0000574
Cobalt	0.000000	0.1	0.00000
Chromium	0.000006	-	
Cr III (95%)	0.000006	60	0.0000001
Cr VI (5%)	0.0000003	0.1	0.000003
Copper	0.00001	1	0.00001
Manganese	0.00002	0.15	0.0001
Nickel	0.000069	0.05	0.0014
Lead	0.00001	0.5	0.00002
Antimony	0.000000	0.2	0.000002
Selenium	0.000001	20	0.0000001
Tin	0.00001	48	0.0000002
Vanadium	0.000003	1	0.000003
		Metals hazard index	0.002
Chlorine	0.003	0.2	0.014
Sulfuric acid	0.033	1	0.033
Hydrogen chloride	0.02	9	0.002
Hydrogen fluoride	0.0002	-	
		Acid Hazard index	0.05
TRS (as H_2S)	0.0030	2	0.0015
		Total HI	0.05

APPENDIX E CALCULATIONS FOR CANCER RISKS

Table 11-57: Cancer calculations Receptor 1

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.22E-07	0.0018	2.2E-10
Arsenic	1.97E-06	0.0015	3.0E-09
Beryllium	2.56E-06	0.00243	6.2E-09
Chromium (VI)	7.40E-07	0.04	3.0E-08
Nickel	1.82E-04	0.00038	6.9E-08
Total Carcinogenic Risk			1.1E-07

Table 11-58: Cancer calculations Receptor 2

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.68E-07	0.0018	3.0E-10
Arsenic	2.76E-06	0.0015	4.1E-09
Beryllium	3.36E-06	0.00243	8.2E-09
Chromium (VI)	1.00E-06	0.04	4.0E-08
Nickel	2.50E-04	0.00038	9.5E-08
Total Carcinogenic Risk			1.5E-07

Table 11-59: Cancer calculations Receptor 3

Compound	GLC	URF	Carcinogenic Risk
Cadmium	9.41E-08	0.0018	1.7E-10
Arsenic	1.40E-06	0.0015	2.1E-09
Beryllium	2.33E-06	0.00243	5.7E-09
Chromium (VI)	6.04E-07	0.04	2.4E-08
Nickel	1.42E-04	0.00038	5.4E-08
Total Carcinogenic Risk			8.6E-08

Table 11-60: Cancer calculations Receptor 4

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.67E-07	0.0018	3.0E-10
Arsenic	2.72E-06	0.0015	4.1E-09
Beryllium	3.44E-06	0.00243	8.4E-09
Chromium (VI)	1.01E-06	0.04	4.0E-08
Nickel	2.49E-04	0.00038	9.5E-08
Total Carcinogenic Risk			1.5E-07

Table 11-61: Cancer calculations Receptor 5

Compound	GLC	URF	Carcinogenic Risk
Cadmium	3.19E-07	0.0018	5.7E-10
Arsenic	5.33E-06	0.0015	8.0E-09
Beryllium	6.17E-06	0.00243	1.5E-08
Chromium (VI)	1.88E-06	0.04	7.5E-08
Nickel	4.75E-04	0.00038	1.8E-07
Total Carcinogenic Risk			2.8E-07

Table 11-62: Cancer calculations Receptor 6

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.86E-07	0.0018	3.3E-10
Arsenic	3.05E-06	0.0015	4.6E-09
Beryllium	3.75E-06	0.00243	9.1E-09
Chromium (VI)	1.11E-06	0.04	4.4E-08
Nickel	2.77E-04	0.00038	1.1E-07
Total Carcinogenic Risk			1.6E-07

Table 11-63: Cancer calculations Receptor 7

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.39E-07	0.0018	2.5E-10
Arsenic	2.08E-06	0.0015	3.1E-09
Beryllium	3.47E-06	0.00243	8.4E-09
Chromium (VI)	8.96E-07	0.04	3.6E-08
Nickel	2.10E-04	0.00038	8.0E-08
Total Carcinogenic Risk			1.3E-07

Table 11-64: Cancer calculations Receptor 8

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.30E-07	0.0018	2.3E-10
Arsenic	1.93E-06	0.0015	2.9E-09
Beryllium	3.27E-06	0.00243	7.9E-09
Chromium (VI)	8.40E-07	0.04	3.4E-08
Nickel	1.96E-04	0.00038	7.5E-08
Total Carcinogenic Risk			1.2E-07

Table 11-65: Cancer calculations Receptor 9

Compound	GLC	URF	Carcinogenic Risk
Cadmium	7.13E-08	0.0018	1.3E-10
Arsenic	1.06E-06	0.0015	1.6E-09
Beryllium	1.79E-06	0.00243	4.3E-09
Chromium (VI)	4.59E-07	0.04	1.8E-08
Nickel	1.07E-04	0.00038	4.1E-08
Total Carcinogenic Risk			6.5E-08

Table 11-66: Cancer calculations Receptor 9A

Compound	GLC	URF	Carcinogenic Risk
Cadmium	9.31E-08	0.0018	1.7E-10
Arsenic	1.41E-06	0.0015	2.1E-09
Beryllium	2.25E-06	0.00243	5.5E-09
Chromium (VI)	5.92E-07	0.04	2.4E-08
Nickel	1.40E-04	0.00038	5.3E-08
Total Carcinogenic Risk			8.5E-08

Table 11-67: Cancer calculations Receptor 10

Compound	GLC	URF	Carcinogenic Risk
Cadmium	7.43E-08	0.0018	1.3E-10
Arsenic	1.09E-06	0.0015	1.6E-09
Beryllium	1.89E-06	0.00243	4.6E-09
Chromium (VI)	4.82E-07	0.04	1.9E-08
Nickel	1.12E-04	0.00038	4.3E-08
Total Carcinogenic Risk			6.8E-08

Table 11-68: Cancer calculations Receptor 11

Compound	GLC	URF	Carcinogenic Risk
Cadmium	8.65E-08	0.0018	1.6E-10
Arsenic	1.28E-06	0.0015	1.9E-09
Beryllium	2.19E-06	0.00243	5.3E-09
Chromium (VI)	5.60E-07	0.04	2.2E-08
Nickel	1.31E-04	0.00038	5.0E-08
Total Carcinogenic Risk			7.9E-08

Table 11-69: Cancer calculations Receptor 12

Compound	GLC	URF	Carcinogenic Risk
Cadmium	7.24E-08	0.0018	1.3E-10
Arsenic	1.07E-06	0.0015	1.6E-09
Beryllium	1.84E-06	0.00243	4.5E-09
Chromium (VI)	4.69E-07	0.04	1.9E-08
Nickel	1.09E-04	0.00038	4.2E-08
Total Carcinogenic Risk			6.7E-08

Table 11-70: Cancer calculations Receptor 13

Compound	GLC	URF	Carcinogenic Risk
Cadmium	8.13E-08	0.0018	1.5E-10
Arsenic	1.22E-06	0.0015	1.8E-09
Beryllium	2.01E-06	0.00243	4.9E-09
Chromium (VI)	5.22E-07	0.04	2.1E-08
Nickel	1.23E-04	0.00038	4.7E-08
Total Carcinogenic Risk			7.4E-08

Table 11-71: Cancer calculations Receptor 14

Compound	GLC	URF	Carcinogenic Risk
Cadmium	4.69E-08	0.0018	8.4E-11
Arsenic	6.86E-07	0.0015	1.0E-09
Beryllium	1.20E-06	0.00243	2.9E-09
Chromium (VI)	3.05E-07	0.04	1.2E-08
Nickel	7.08E-05	0.00038	2.7E-08
Total Carcinogenic Risk			4.3E-08

Table 11-72: Cancer calculations Receptor 19

Compound	GLC	URF	Carcinogenic Risk
Cadmium	6.86E-08	0.0018	1.2E-10
Arsenic	1.01E-06	0.0015	1.5E-09
Beryllium	1.73E-06	0.00243	4.2E-09
Chromium (VI)	4.44E-07	0.04	1.8E-08
Nickel	1.03E-04	0.00038	3.9E-08
Total Carcinogenic Risk			6.3E-08

Table 11-73: Cancer calculations Receptor 21

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.50E-07	0.0018	2.7E-10
Arsenic	2.35E-06	0.0015	3.5E-09
Beryllium	3.34E-06	0.00243	8.1E-09
Chromium (VI)	9.25E-07	0.04	3.7E-08
Nickel	2.24E-04	0.00038	8.5E-08
Total Carcinogenic Risk			1.3E-07

Table 11-74: Cancer calculations Receptor 22

Compound	GLC	URF	Carcinogenic Risk
Cadmium	1.49E-07	0.0018	2.7E-10
Arsenic	2.27E-06	0.0015	3.4E-09
Beryllium	3.54E-06	0.00243	8.6E-09
Chromium (VI)	9.42E-07	0.04	3.8E-08
Nickel	2.24E-04	0.00038	8.5E-08
Total Carcinogenic Risk			1.4E-07

Table 11-75: Cancer calculations Receptor 26

Compound	GLC	URF	Carcinogenic Risk
Cadmium	8.57E-08	0.0018	1.5E-10
Arsenic	1.31E-06	0.0015	2.0E-09
Beryllium	2.03E-06	0.00243	4.9E-09
Chromium (VI)	5.41E-07	0.04	2.2E-08
Nickel	1.29E-04	0.00038	4.9E-08
Total Carcinogenic Risk			7.8E-08

Table 11-76: Cancer calculations Receptor 27

Compound	GLC	URF	Carcinogenic Risk
Cadmium	4.56E-08	0.0018	8.2E-11
Arsenic	6.76E-07	0.0015	1.0E-09
Beryllium	1.15E-06	0.00243	2.8E-09
Chromium (VI)	2.94E-07	0.04	1.2E-08
Nickel	6.88E-05	0.00038	2.6E-08
Total Carcinogenic Risk			4.2E-08